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BEGUN BY THE LATE

CHARLES HILTON FAGGE, M.D., F.R.C.P.
SOMETIME PHYSICIAN TO GUY'S HOSPITAL

COMPLETED AFTER HIS DEATH AND SINCE REVISED OR RE-WRITTEN BY

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Fourth Edition

IN TWO VOLUMES

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PREFACE.

IN the present, as in the three preceding editions of this book, many additions have been made, much obsolete matter has been expunged, and some of the chapters have been rearranged as well as expanded. At present there is scarcely a page of the late Dr Fagge's work as he left it at the time of his premature death, in November, 1883. Perhaps the passages least altered are those describing the anatomy of Enteric Fever, the clinical account of Variola and Scarlatina, the course of malarial fever, the description of Neuralgia and Migraine, of Apoplexy and Hemiplegia, of Paraplegia from compression and of Epilepsy, the account of Bronchitis, Emphysema and spasmodic Asthma, and the description of chronic Bright's disease.

For the chapters on disease of the Heart and for those on affections of the Skin I am entirely responsible, as also for the sections on the Œsophagus, on Actinomycosis, Acromegaly, Beri-beri, Sweating Sickness, Rubella, Peripheral Neuritis, Friedreich's Ataxia, Thomsen's disease, Syringomyelia, Insular Sclerosis, certain disorders resembling Chorea, and diseases of the Pancreas and Spleen.

The introductory sections on general pathology and those on Diabetes, Rheumatism, Osteo-arthritis, and Gonorrhœal Synovitis, on Influenza, Enteric Fever, Diphtheria, and Pneumonia, on Typhlitis, Myxœdema, Dysentery, and Colitis, the chapters on Hepatic disorders, on Anæmia and Phthisis, on Intestinal obstruction, on spastic and atrophic Paralysis and Tabes, and on the methods of Percussion and Auscultation of the chest, have all been more or less re-written for the present edition.

I have endeavoured to make the contents of each chapter, the head-lines of pages and of paragraphs, and the arrangement and sequence of subjects more uniform, and therefore more convenient for students. A short historical sketch begins the more important chapters, and is followed by the clinical symptoms and course, the diagnosis, the anatomy, the natural history and ætiology, ending with the prognosis and the treatment, both preventive and curative. I do not apologise for the historical and etymological notes and quotations, and wish that

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more space could have been given to them, for I believe that to know a subject thoroughly we must learn how the knowledge we now possess has been obtained. I have added much and omitted something from the sections on treatment, but have designedly not given prescriptions "good for" this or that disorder; nor have I attempted to help diagnosis by tabular opposition of symptoms, which appears to me to be always imperfect and often misleading. As much as possible I have tried to depend on my own experience for the statements made, and have noticed the instances in which I have been obliged to take the account of rare or exotic diseases on the authority of other writers.

Throughout both volumes "the writer" stands in lieu of the first personal pronoun; and the initials C. H. F. denote that the case or foot-note immediately preceding them belongs to the late Dr Fagge.

In conclusion, I must express my obligations to many old pupils and other medical friends who have drawn my attention to misprints or obscurities or misstatements in the last edition, and to hope that I may be favoured by such valuable criticism on the present. It is difficult in so long a work—which can, moreover, only be written in intervals liable to interruption—to avoid these errors entirely. But I was so fortunate as to obtain the acute and accurate supervision in reading for the press of my friend John Cavafy, late senior physician to St. George's Hospital; and so unfortunate as to lose that assistance, and his long and highly valued friendship, by his sudden and lamented death. This happened when the chapters on Cardiac Disease were passing through the press, and from that point I have been indebted to the generous help of a friend as old and as highly valued, Dr Payne, consulting physician to St. Thomas's Hospital, who has most kindly added to his self-imposed task that of frequent and judicious advice on many subjects, particularly in the latter portions of the book.

P. H. P.-S.

BROOK STREET, LONDON;
August, 1901.

⁂ The publishers, Messrs Churchill, have thought it desirable to issue the present volume at once. The second volume is already in the press, and will, unless unforeseen accident prevent, be ready for publication by the end of the year.

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ERRATA.

Vol. I, page 102, for "myxœdema" read "mycœdema."
„ „ 880 (Contents), for "Weir's" read "Weir Mitchell's."

TEXT-BOOK OF MEDICINE

VOL. I

INTRODUCTORY CHAPTER

> ’Ιατρεύει (ὁ ἰατρὸς) καθ’ ἕκαστα.
>
> ARISTOTLE.

Definition of medicine and of disease—Nosology: organic and functional diseases—pathological anatomy and clinical characters as bases of nosology—Diagnosis—symptoms and diseases—symptoms and physical signs—General Ætiology: exciting and predisposing causes of disease—natural history of Disease—General Prognosis—General Prophylaxis and Therapeutics—Classification—Nomenclature—Plan and arrangement of the present work.

MEDICINE, *Ars medendi*, *Ars valetudinis*, is the art of detecting, of relieving, and of preventing diseases.

Preventive Medicine, as applied to preserving the health of the community, forms a separate branch of the Art best called HYGIENICS, and in its relation to government is known as State Medicine. As applied to preserving individuals from diseases it will find its place in several parts of the present work under the name of PROPHYLAXIS. It depends upon our knowledge of the causes and origin of each malady.

Curative Medicine, or THERAPEUTICS, includes not only the application of means which entirely remove the disease, and without which it would continue, but also the far more frequent cases in which, without directly overcoming the malady, its course is guided in the most favourable manner toward recovery, and those moreover in which, although the disorder cannot be arrested, its progress is retarded and its pain relieved. *La médecine guérit quelquefois, elle soulage souvent, elle console toujours.*

This art of mitigating the sufferings and of more or less directly saving the lives of the sick, is, like all arts, empirical and individual,—that is to say, it goes upon experience, and is concerned more with the patient than with the disease. The physician deals with cases severally. Nevertheless it rests on a scientific basis: first on knowledge of the several disorders of the human body and of the natural functions from which they are deviations—Pathology; and secondly, on knowledge of the various means, mechanical, physical, chemical, and vital, by which the morbid structures and functions may be influenced—Pharmacology, in the most extended sense of the word. Inasmuch as our knowledge on both sides is very imperfect, we depend on the results of experience to correct our conclusions and to regulate our future methods.

The practice of medicine, then, is an art, controlled by experience, guided by individual skill and insight, but resting upon the science of pathology, or the natural history of diseases, as its basis; and on an exact acquaintance with their origin, immediate and remote, their physiological course, and their natural termination.

The art of medicine has been compared to the art of watch-mending, to the art of war, and to the art of education; but perhaps the best illustration of its scope and methods is the art of navigation, which depends on scientific principles, but consists in the application of science to individual and varying conditions, guided by natural acumen, and controlled by long experience.

The present work attempts to deal with both branches of Medicine, its principles and its practice. By an arbitrary but useful convention external injuries and diseases which are chiefly curable by operations are separately dealt with as Surgical Pathology and Therapeutics; and the diseases peculiar to women, as well as those incident to the eye and ear, are also treated of in modern times by special treatises.

DISEASE.—The only definition of *disease* that can stand criticism is—the opposite of *health*, a disorder of the body in the whole or in its parts, which causes pain or shortens life.

In truth, both Disease and Health are incapable of precise definition, because they are not scientific terms. They are descriptive, popular, and subjective. "Dis-ease" is discomfort. Whatever causes bodily uneasiness, or whatever by experience will sooner or later cause it, whatever interferes with our bodily functions, whatever tends to death, is disease. Health is the opposite condition of comfort, ease, and ability to eat, sleep, move, and perform the other functions of life.

Diseases have only this in common, that they all interfere with comfort or shorten life. There is no common cause for the pains of inflammation, of childbirth, and of mechanical injury.

Regarded apart from our feelings no line can be drawn between health and disease. Pathology is only physiology under various disturbing causes. Decay and death are as much physiological events as birth and life.

All diseases imply two things—an exciting cause, *quidquid irritans*, mechanical, thermal, chemical, parasitic, infective, or of unknown nature; and a reacting living organism, *quidquid irritabile*. Stone in the bladder is not a disease till it causes symptoms by exciting reaction. The severest injuries, the most violent poisons, produce no disease in a corpse.

There is a tendency after disturbance to return to the previous condition, if the equilibrium has not been too far upset. This tendency has been called *Vis medicatrix Naturæ*; but there is no evidence of the existence of such a force, which is contrary both to Philosophy and to Theology. Indeed, the so-called "efforts of Nature" often aggravate instead of cure the mischief. Our mortal bodies are not made to last for ever.

It is clear that if disease is not a single state, nor the result of a single cause, it cannot be removed by any single method, or on any universal principle. Hence all "Systems" of Medicine, like all "Universal remedies," are of necessity false. Iatro-mechanical and iatro-chemical schools, Brunonian and Antiphlogistic theories, Allopathy and Homœopathy, are all equally unreasonable; not wrong solutions of a scientific problem, but ignorant answers to an absurd question.

The art of medicine, therefore, is not concerned with Disease in the abstract, but with separate diseases, with pains and discomforts, the cause, seat, and origin of which the physician seeks to detect, and, if possible, to cure in each case—*ἰατρεύει γὰρ καθ' ἕκαστα.*

Diseases and nosology.—Since diseases are of diverse nature, so that they are not mutually comparable, it is plain that a scientific classification of them is impossible. Hence there is little left of the importance formerly attached to Nosology, *i. e.* the right nomenclature, definition, and classification of diseases.

It may, however, still be asked—and the inquiry is not without a practical bearing—whether in naming and classifying diseases we should do so according to their *causes*, their character as physiological *processes*, the *structural changes* they produce, or the *symptoms* by which we recognise them during life.

The first is the most satisfactory basis, for it is fundamental. When we define scabies as the effects of the presence of an acarus in the skin, or "dropt wrist" as paralysis from lead, we know the most important points at once, the leading indications for prevention and for cure.

But our knowledge is far too imperfect to render an ætiological classification possible.

A pathological arrangement is almost as hopeless, for in many cases we are ignorant of the physiological derangements which are taking place, and the long list of "inflammations" includes very diverse diseases.

What has in modern times been called a pathological basis of diseases is not one of processes, but of results. It is really based, not on morbid physiology, but on morbid anatomy. During the present century the utmost zeal has been devoted to the search for organic lesions in the dead body, and to tracing the relations between such changes after death and states of ill-health during life. Many anatomical lesions have been thus discovered, which were unknown to the physicians of former ages; and many of these can be recognised without difficulty by skilled observers, even though the patient is unaware of their existence.

On the other hand, there is a not inconsiderable number of disorders which force themselves into notice by the pain or discomfort they occasion, and which may in some cases destroy life; and yet after death all the organs and tissues seem to be unaltered, even when they are examined with the highest powers of the microscope and by every chemical test that can be devised.

After taking due account of toxic disorders—from mineral poisons and from products of animal and vegetable secretion or decomposition—there still remain defects of secretion, of movement, and of innervation, which we must regard as purely functional.

But although we may still distinguish between *organic* or *structural* diseases attended with recognisable morbid changes, and *functional* diseases in which no such lesions can be found, yet the progress of science is continually transferring maladies from the latter to the former class. So that the distinction between functional and organic disorders is, though convenient, not fundamental.

In both cases the "disease" is the *condition* of the living patient, the pathological *process*, the *derangement* of the functions, and thus includes the "symptoms" (which are generally the most obvious phenomena in the living patient, and which used themselves to be called diseases) and the anatomical lesion, if known, which underlies them.

Hence, as knowledge improves, what was a "disease" becomes a "symptom," and what was an anatomical lesion becomes the evidence of a pathological process. Dropsy, jaundice, apoplexy, paralysis, are terms which now denote only the first, not the final stage of diagnosis. Phthisis, which at first meant "wasting," afterwards meant a special anatomical lesion of the lungs, and now means the whole physiological process, including structural, functional, and symptomatic disorders which are the result of infection by a microscopic plant.

It is not necessary that every disease should be defined alike; in *scabies* the fundamental character is the presence of a special cause, in *aneurysm* it is an anatomical lesion, in *chorea* it is a series of disordered functions. Whenever a group of clinical cases can be traced to a single structural change, this should be regarded as the most essential element in the definition of the disease, and it should, if possible, furnish the name. But if the effects of several lesions are identical, so that they are clinically undistinguishable from one another, it is better to retain a common designation. Such an arrangement may be regarded as provisional, but it is likely to be needed for many years to come.

DIAGNOSIS.—Until we have traced a disease to a definite *organic lesion* we can seldom be sure that cases which seem to be identical are really so; and upon their real identity depends all certainty of knowledge as to their cure. This is anatomical diagnosis, the determination of the sort or nature of the lesion, and of its seat.

Again, until we know the *origin* of the structural lesion we cannot tell how to guard against its production, *i. e.* how to prevent the whole process which we call the disease. This is ætiological diagnosis, the determination of the causes of the lesion.

The process by which we give a name to a case is one of analysis; and in some cases we can carry this process further than in others. A complete diagnosis, such as "variola," "syphilitic iritis," "glioma of the left lobe of the cerebellum," includes recognition of the characteristic symptoms, of the anatomical lesion in its seat and structure, and of the origin of the whole morbid process. Nevertheless, to say that a patient has "hepatic ascites," though an incomplete diagnosis, is sometimes more accurate than to say that he has "cirrhosis of the liver," for we cannot always exclude perihepatitis, or cancer, or gummata. To define a cerebral case as one of "hemiplegia" is sometimes more accurate than to call it "cerebral embolism." Yet when we can be practically sure that cirrhosis of the liver or that softening of the brain is present, it is obviously better to name such cases according to their anatomical lesions.

The phenomena which are spoken of as symptoms are parts of the disease to which they belong, no less than the lesion, or the specific cause, or whatever else is taken as its main characteristic. It is true that, when they enable us to infer the existence of a hidden morbid change, we are working back from effects to their cause; but this only shows that the term symptom is a relative one. So long as we are unable, either during life or after death, to discover any cause for a set of clinical phenomena, they certainly are not symptoms, for they indicate nothing. There can be no symptom until the disease of which it is a symptom is discovered. As the process of analysing a case or a group of cases advances, the name which we apply is rightly changed at every step; and each "disease" becomes in its turn a "symptom" of the disease which underlies it. Itching was once hardly distinguished from the itch; afterwards, when the characters of the eruption of scabies were made out, itching was looked on as a symptom, and the eruption was the disease; now that the *sarcoptes* has been discovered, the eruption is itself reduced to a symptom.

If, however, the analysis is always arrested at a certain point, it is absurd to say that the ultimate fact in such cases is "only a symptom." So far from hesitating to classify clinical symptoms as diseases when we are unable to trace them to their causes, it is to be wished that we had a more complete supply of suitable names denoting functional disturbance, without deciding on its origin. Such "clinical nomenclature" is most important in returning "causes of death" in certificates, for we can be sure of acute paraplegia or ascites, but cannot without an autopsy be sure of myelitis or gumma of the liver. "Apoplexy" is often truer than "cerebral hæmorrhage," cardiac disease than fatty degeneration of the heart, and abdominal cancer than carcinoma of the head of the pancreas.

Even when we understand the whole pathology of a case, and when the clinical phenomena which characterise it are rightly called symptoms, the distinction between them and the anatomical lesion is often arbitrary. Thus, in acute tuberculosis, if the ophthalmoscope discovers a tubercle in the choroid membrane of the patient's eye, it is called a symptom; but the disease itself is anatomically only a multitude of precisely similar tubercles. So, again, the enlarged liver of mitral disease, the nodes of syphilis, the swollen joints of rheumatism, belong in strictness to the pathological anatomy of these several complaints. Nor, if we would be logical, can we stop even here; the same thing must be said of all cutaneous eruptions, down to the rose-rash of enteric fever.

On the other hand, in the case of a functional disorder, to speak of the clinical phenomena as symptoms is really to reduce the "disease" itself to a mere name.

Symptoms and signs.—Symptoms may be divided into two classes. Some of them are "subjective;" that is, they are appreciable by no one but the patient, so that our knowledge of them rests solely on his statements. Others are "objective;" that is, they can be directly observed by other persons, even when the patient himself is unconscious.

Subjective symptoms are open to fallacy. They can often be feigned by one who desires to deceive. Moreover their severity depends upon the individual in whom they occur: one man will take no notice of a pain which would drive another to his bed. In the former case one must be careful not to overlook, in the latter not to assume, the presence of disease. Yet constant attention must be paid to subjective symptoms, for they often yield information which can be obtained in no other way.

The physician elicits physical signs by examining the patient's body with hands, eyes, and ears. Until the time of Auenbrugger, Corvisart, and Laennec, he could do little more than feel the pulse and look at the tongue. Then came the discoveries of percussion and of auscultation, which gave to the diagnosis of diseases a precision undreamt of before. It was natural that those who introduced the new methods should endeavour to mark their value by giving a special name to the indications afforded by them. And thus it became usual to speak of "physical signs" as opposed to "symptoms." In reality, however, the distinction is untenable. A raised temperature, an internal squint, albumen in the urine, are all "physical signs," no less than a cardiac murmur or a dull percussion-note; and small crepitation is as much a symptom as rusty sputa.

This may, however, be said, that physical signs, usually so-called, which are obtained by percussion and auscultation, are signs only of physical, not of pathological, conditions, whereas paralysis, pyrexia, jaundice are signs of physiological (*i. e.* pathological) processes.

It is misleading to call a particular symptom "pathognomic," as if it bore so close a relation to the disease that, whereas all the rest of the symptoms afford only presumptive indications of its presence, this one proves it absolutely. No symptom and no sign leads straight to a diagnosis and renders investigation unnecessary.

In order to make a successful diagnosis we must never make a hasty one. In many cases a first glance shows us that a patient is the subject of phthisis, of syphilis, or of cirrhosis; but even then other concomitant conditions may be overlooked unless we methodically examine the several organs. We must give primary importance to direct objective symptoms, "physical signs" included; but we must not neglect the general aspect of the patient, nor the probabilities afforded by knowledge of his occupation, previous life, and family history. First thoughts and third are usually best. We must often be content with a preliminary diagnosis of "organic disease of the brain," "hepatic dropsy," or "acute paraplegia," and make it more precise as the case develops itself. Ultimately we should aim at a complete diagnosis in every case, *i. e.* one which includes the organ and part of the organ affected, the nature of the disease, and its origin.

ÆTIOLOGY.—The real *causes* of diseases are, for the most part, unknown. True, there is often little difficulty in perceiving that an illness has been preceded by some definite change in the outward circumstances of the patient; he may have been exposed to cold, he may have eaten unwholesome food, he may have been over-excited or over-fatigued; and he naturally calls this the *cause* of the disease. But, on further consideration, it appears that people often go through weather no less inclement, indulge their appetites as foolishly, and have their mental and bodily powers strained as much, without being attacked by the same, or indeed by any disorder. We must, then, assume that some other cause has also been in operation. This may act in two ways: it may combine with the exciting cause to produce the disease in those who fall ill, or it may counteract the operation of the exciting cause in those who remain well. But we generally assume that all such causes have a positive rather than a negative action, and group them together as "*predisposing causes.*"

There are, however, some apparently predisposing causes of diseases which do not operate in either of the ways just mentioned. A complaint may affect persons at a particular age, merely because at that age they are more exposed to the action of the exciting cause. Thus scalds and burns are particularly frequent among young children; while scalds of the throat from drinking hot water from a kettle scarcely occur in grown-up persons. On the other hand, young men are much more liable to break their limbs than are women or old men. Such differences arise, not from a predisposing influence of one period of life, nor from a counteracting influence of another period, but from infants being ignorant of the effects of heat, and from men at their prime being obliged to work under exposure to accidents. So again, if general paralysis and locomotor ataxy and many other diseases of the nervous centres are more frequent in men than in women, the reason may be that more men than women come under the operation of the exciting causes of these complaints, such as mental excitement, sexual excesses, drink, and above all syphilis.

The most obscure of predisposing causes are those which are commonly included under the term "idiosyncrasy." For example, in certain persons nettle-rash is produced by strawberries, and in others the odour of cats excites asthma. In such cases the exciting cause is obvious, and we must regard as predisposing causes the conditions, whatever they may be, which lead to its effect upon one person and not on another. But of these conditions we are altogether ignorant.

In some cases predisposition seems to be unnecessary. The presence of the acarus in the skin is the sole and efficient cause of scabies, and needs nothing more than a normal human skin to produce the lesions and the symptoms which make up the disease scabies. Yet even here it appears that certain abnormal conditions, as typhus, and perhaps other febrile states, counteract the ordinary operation of the irritant. Again, contact with another is the constant precedent of every case of variola, yet here again a previous attack renders the organism "refractory" to the normal operation of the contagion. That grown people are little susceptible to measles is probably explained by most of them having already had it.

Hereditary predisposition to certain diseases is undoubted, but the limits of its action are difficult to define. Gout, phthisis, and hæmophilia are certainly hereditary, but not always. Syphilis is not hereditary in the same sense, but is caught by the fœtus from the parent. It does not therefore furnish an instance of inheritance of an acquired character which biologists have been driven to seek in Pathology; and in the case of both gout and tubercle it is rather the innate proclivity to the disease or lack of power of resistance to its attacks which is transmitted from parent to offspring, than the acquired disease itself.

If we attempt to classify diseases by their causes, we may put first those which are due to the presence of animal or vegetable *parasites*, both such as act mechanically by their presence as irritants, and those minute parasitic plants which multiply excessively and fill the whole organism that they invade with swarms of microphytes. In many cases diseases are *infectious* where no such parasites have been detected, but these are no less strictly dependent on a previous case, which furnishes the necessary contagion.

Next may be placed, from an ætiological point of view, those disorders which are the direct result of *chemical poisons*, the paralysis caused by lead, the delirium of belladonna poisoning, the tremors of workers in mercury, the fatty degeneration caused by phosphorus. In these cases the effects are very uniform in character, and depend in degree upon the dose of the poison, with little reference to the recipient.

Next we may put *dietetic* diseases, as cirrhosis of the liver and delirium tremens, which are caused by spirit drinking. Here, although the cause is true and constant, its effect depends upon something in the individual, for some drunkards die rapidly from the effect of intemperance on the brain, others more slowly from its effect on the liver, and others escape both.

The action of *cold* is still less constant. Exposure to weather produces in one man rheumatic fever, in another bronchitis, in a third pleurisy or pneumonia, so that we cannot predict what disease will be produced by the cause, or whether any at all.

The remaining "causes" of disease are so vague and uncertain in their action that we must suppose that some peculiar combination of conditions is needful to produce the morbid effect, and that of one or more of these conditions we are ignorant.

When we cannot assign any probable cause for a disease we often call it "idiopathic," "primary," or "spontaneous;" meaning that so it appears to be until its true ætiology is ascertained.

A small ætiological group of diseases consists of those which are the result of *defect of formation*, as cyanosis from congenital disease of the heart. Possibly some forms of paralysis are due to imperfect development of the cord.

A larger group includes those which depend upon various *senile degenerations* of the tissues and organs. But many of these changes which occur as the result of ordinary wear and tear in most persons above a certain age may also be found in those much younger, in whom excessive strain has prematurely produced the same effects.

One mode of origin of diseases is in slight perversions of physiological functions, at first occasional, then frequent, and at last constant, until structural changes at length ensue. Disease of the heart and aorta from muscular strain, emphysema from chronic bronchitis originating in an occasional cough, and gutta rosea from frequent flushing of the face, are examples of this process (see the writer's Lumleian Lectures on Ætiology, pp. 22—28).

On the whole it may be said that the cause of diseases is more confidently assigned by the patient than by the physician.*

When degenerative, developmental, contagious, parasitic, toxic, and traumatic diseases are excluded, there remain by far the greater number of the real origin of which we know nothing.

Even in these cases, however, their natural history—the conditions of age, sex, occupation, geographical distribution under which they most commonly occur—is of much interest. Such knowledge is often a useful help in diagnosis, and may one day lead to more complete conclusions as to ætiology. So that under this head such details are appropriately placed.

It is in reference to the prevention of diseases that a knowledge of their ætiology is of the greatest practical use, but indirectly it often has an important bearing upon their diagnosis and their cure. The process of analysing the clinical symptoms, which constitutes the diagnosis of a case, often stops short of tracing it to a definite anatomical lesion; and we are unable by that path to reach any basis for a sound plan of treatment. In such cases, every evidence of possibly exciting or predisposing causes of the patient's illness may be of great value in helping us to treat it successfully.

PROGNOSIS.—The proof of the reality of diagnosis is the power of prediction; and before we attempt to modify the progress of a disease by treatment we must know what is its natural course when undisturbed by our art. We are then able to give what is termed a *prognosis* or forecast of the case. This part of our duty may be of the highest importance to the patient and his friends, and our own reputation is more concerned in prognosis than even in treatment.

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* As Sir William Gull well put it, "in disease as well as in astronomy, savages explain, science investigates."

In some cases, indeed, the prognosis is of such a character as almost to make any further services to our patient unnecessary. We may be able to declare that the disease will quickly and safely pass off, without tendency to relapse, and without risk of its leaving any ill effects behind it. A case in point is an attack of shingles in a child. Or we may have to say that the malady is one which must inevitably prove rapidly fatal in spite of every effort that can be made to arrest it. Such a prognosis must be given in many cases of apoplexy, of aneurysm, and of tetanus, and in almost all of cancer.

But, as a rule, our prognosis applies not so much to the natural and undisturbed course of the disorder, as to that which we believe it will take under the most judicious treatment we can devise.

Prognosis is not only concerned with the favourable or unfavourable event of the disease, but also with its probable duration, the chances of its return, and the possible or probable complications against which it is most important for the physician to be forewarned.*

When we see a patient we instinctively frame a forecast of the issue of his illness, and natural shrewdness of observation aided by experience may thus reach "something like prophetic strain." But such prognosis is limited to the immediate future, and even then is often at fault. The futility of judgments of this kind expressed by those conversant with the sick, but ignorant of pathology, only escapes general recognition by the occasional hits being marked and the more frequent misses forgotten. Prognosis without diagnosis is even more baseless than the hopes or the forebodings of the patient himself. True prognosis depends first on recognition of the nature and seat of the illness in question, of its natural history, its course and probable event if left to itself; and, secondly, on our estimation of the patient's power of resisting the disease. based on his age, his habits, the condition of his organs, and experience of previous attacks of illness.

Advanced age is a serious symptom in most diseases, and alone determines the gravity of enteric and most other specific fevers, of pneumonia, of bronchitis, and of zona. But certain other diseases, notably diabetes, phthisis, and rheumatism, are far more dangerous at twenty-five than at sixty. In early childhood all diseases are serious, and many which are manageable in adults are often fatal; above all diphtheria, but also bronchitis and diarrhœa. Surgical operations, also, are dangerous in young children. And yet a sickly infant often shows wonderful tenacity of life. and will recover from tuberculous disease, marasmus, and general eczema which seemed to be hopeless.

Two phrases—the *constitution* of the patient and his *temperament*—are still often used in making a prognosis; but, like most abstract terms. they are ambiguous and misleading. Knowledge of the constitution of a patient can only mean knowledge of his height and weight and chest-girth. and of the characters of his pulse and urine. Such knowledge, combined with information as to his habits, temperance, occupation, residence in foreign countries, and previous disorders, is valuable in forming a prognosis in cases of life assurance; but the several facts are best stated as such, and are only obscured by so meaningless a term as "constitution."

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* 'Guy's Hospital Reports' (1887), vol. xliv, p. 59.

The older physicians spoke of "laxity of the fibres" or "deficiency of the animal spirits" as part of their patient's constitution, but these obsolete phrases are not more absurd than to discuss whether a patient has "stamina," or whether his nerves are "highly strung."

Temperament meant originally the mixture of the four humours of Greek physiology—the Blood formed in the liver, the Bile in the gall-bladder, the Phlegm (*pituita*, mucus) in the pituitary gland, and the Black bile in the spleen. If any one of the humours were in excess, the result was a Sanguine, a Choleric (bilious), a Phlegmatic (lymphatic), or a Melancholic (atrabilious) temper. These words have now only an antiquarian interest, and should be dismissed from scientific medicine with the whole fabric of humoral pathology, which was long ago exploded.

Family history is an important element in forming a prognosis both of a healthy and a diseased body. Absence of longevity in a large family is a serious fact. Some conditions are marked by heredity, as insanity, drunkenness, phthisis, and gout; others are occasionally so, as cancer and diabetes. But great care is necessary not to be misled by words. In insurance papers phthisis is often concealed under such phrases as "the result of childbirth," or "bronchitis," or even "accident." Again, we must not speak of family tendency to nervous disease because one relative died from hemiplegia due to arterial degeneration, another from paraplegia due to caries of the spine, a third from tumour of the brain, a fourth from tabes, a fifth from puerperal convulsions, and a sixth from suicide during "temporary insanity." As well might we assume liability to "skin-disease" in a family, some of whose members had suffered from scabies, psoriasis, zona, rupia, or lupus; or an arthritic diathesis from the accidental conjunction of such divers affections as rheumatic fever, gout, traumatic synovitis, osteo-arthritis, and tuberculous disease of a joint.

TREATMENT.—This, the end and object of Pathology, consists of two parts—curative and preventive. The latter is proverbially the more important. It rests mainly on a knowledge of ætiology, and is therefore of very modern growth. It deals rather with communities than with individuals, but will not be unnoticed in the following chapters, particularly in the case of epidemic diseases.

Therapeutics, or the art of curing diseases, depends much more upon the physician's knowledge and skill, and much less upon specific drugs, than is supposed by the laity. In few cases can a malady be certainly and directly "cured" or arrested by a given method as soon as the diagnosis has been made; but in still fewer is medicine powerless for the comfort and welfare of the patient.

In some diseases no treatment is needful. All that is important is that they should be recognised, and the patient assured of his safety.

In others the only reasonable treatment is expectant; that is, to refrain from meddling without knowledge, and to watch carefully for opportunities of helping favourable and relieving unfavourable symptoms.

In others, again, treatment is strictly rational, *i. e.* it consists in employing mechanical, chemical, or physiological means for directly meeting an injurious condition. Such is the treatment of laryngeal dyspnœa by opening the trachea, of poisons taken into the stomach by chemical antidotes and by emetics, of high temperature by cold affusion. In the same way we treat iritis by belladonna, dropsy by diuretics, typhlitis by opium, and uterine hæmorrhage by ergot.

There remain a considerable number of cases in which, without our being able to see the physiological action of the means that are used, we are able to modify or arrest the progress of a disease by treatment which depends entirely upon experience of its value. As examples may be mentioned the relief given by external warm applications in most inflammatory states, the benefit of counter-irritation, the use of purging in certain cerebral affections, of colchicum in gout, of tarry applications in psoriasis, of iron in chlorosis, and of mercury in syphilis. These last so-called "specific" medicines have doubtless a physiological action which, if known, would explain their practical value; and in some cases we can perceive an obscure connection betwen their known properties and their therapeutical effects. But at present their action is "special," peculiar, and inexplicable.

Therapeutical systems.—Attempts to cure diseases naturally preceded knowledge of their real nature or origin, and thus vast systems of traditional therapeutics, useless or mischievous as a rule, were part of Indian, Egyptian, Greek, Arabian, and European civilisation.

Most of them were based on theoretic principles, now meaningless or disproved, and aimed at some universal principle of treatment. One dogma was treatment by similars, as of scarlet fever by red cloths; another was treatment by opposites, as of fever by fish diet, fishes being of a cold and moist habit. Or "*signatures*" guided the choice of remedies —roses for hæmorrhage, saffron for jaundice, willow-bark for agues. Or, all diseases, being due to "debility," must be cured by corroborants, as the Brunonians asserted, and as many in this century have practised. Or some were "sthenic," and must be jugulated by bleeding and purging. Or all diseases were to be cured by diet, or by water, or by infinitesimal doses of drugs.

All these, and any other universal system of treatment, are demonstrably false. Others will, no doubt, arise, but they will never again be upheld by any but the ignorant.

We may say without boasting that rational treatment was never so bold, and yet so safe, so self-critical and even sceptical, yet so thorough and so successful.

Classification.—The distribution of diseases into classes, orders, genera, and species is a fruitless and useless task, from which the student of medicine at the present day is happily released. The group of specific fevers is the only one which can be called "natural." They agree in ætiology, and in the nature of the disturbance set up by the exciting cause, *i. e.* in their pathology; they agree in being infectious, in their general symptoms and course, in their relations to time, and in their protective power. Lastly, they are usefully studied together from the practical points of view of diagnosis, prognosis, and treatment.

If we classify diseases by their *origin* we can form certain large classes which would express true relations, but would be impracticable for adoption in a systematic treatise. For instance, we should collect together *contagious* disorders; *malarial* diseases; those directly the result of mechanical *injury*, of chemical irritants, or of heat—"traumatic," as they are sometimes called; those produced by exposure to *chills*, the "catarrhal," or, as some German writers unfortunately call them, "rheumatic" diseases; those produced by chemical *poisons*, as lead colic, lead palsy, and saturnine gout, the "zinc-ague" of brassfounders, mercurial tremors and stomatitis; those produced by *drugs*, as urticaria from copaiba, or by animal poisons, as sapræmia, by vegetable parasites, as ringworm, leprosy, and relapsing fever, and by animal parasites, as trichinosis; those produced by unwholesome *food*, as alcoholic delirium and cirrhosis, gout, and the long list of dietetic disorders.

Then would come the diseases of imperfect *development*, in intra-uterine life or in infancy, at dentition or at puberty, and the large and important series of morbid changes which are the result of premature or normally supervening *senile* changes—atheroma, fatty and fibrous degeneration, involution of the uterus or the ovaries or the breast; atrophy of certain organs and hypertrophy of others. These, when delayed to the full term of life, are the "natural" diseases which lead to a kindly death.

If we attempted a *pathological* classification we should place next to the specific fevers those maladies in which the same process of pyrexia is excited by local inflammation, and make a class of inflammatory fevers, as was done by Cullen and by Hildenbrand.

A nosology based on *morbid anatomy* would follow the lines traced by Rokitansky and his successors, but we have only to turn to the best systematic works on the subject to see how unsuitable such a plan would be for a treatise of medicine. Of many diseases we know neither the seat nor the cause; some whose cause we know have no anatomy, and much morbid anatomy has no clinical history.

A *histological* classification would be impracticable as the basis of a complete system; although it is instructive to remember that serous membranes, lymphatic organs, mucous surfaces, bones, and secreting glands have points in common under morbid as well as under healthy conditions. We shall, indeed, find it convenient to group together in this work primary diseases of the blood, diseases of the skin, and diseases of the joints.

The only remaining course open to one who plans a systematic treatise like the present is, after setting aside general specific diseases, to put those together which affect the several organs—the brain and spinal cord, the larynx and lungs, the heart, the digestive apparatus, and so on; and this plan is followed here. Functional diseases are classed with those of the structures which are the seat of the disordered functions—neuralgia with neuritis, epilepsy with cerebral diseases, asthma with bronchitis.

Nomenclature.—Closely allied to classification is terminology. In the case of specific diseases with a constant origin and a definite course, the best names are those which, first, are meaningless; secondly, consist of a single word, and that short, distinctive, and euphonious; and thirdly, can form adjectives. Syphilis and Typhus (not "typhus fever") are two of the best names we have. When, however, the disease is less definite and certain, names otherwise good become ambiguous, as Rheumatism and Pneumonia.

In choosing names we must in the end be guided by usage—

> Quem penes arbitrium est et jus et norma loquendi;

but we should follow the best usage; and in this country it is in every way desirable to keep, as nearly as possible, to the official nomenclature of the Royal College of Physicians, the Navy and Army, and the Registrar-General.

Some names are vernacular, like Measles, Mumps, Smallpox, Shingles, Dengue, Glanders, Ague, Gout; and these are excellent.

Others are Greek or Latin names, classical or barbarous, as Morbilli, Variola, Zona, Psoriasis, Lupus, Scabies; and these are as good or better. Similar names may be more or less successfully coined, as Diphtheria, Pertussis, Enterica, Equinia, Purpura, Leuchæmia.

Personal names are undesirable. They are seldom generally accepted; they are cumbersome, they are often ambiguous, and they are seldom historically accurate. Such are Pott's disease, Morbus maculosus Werlhofii, Bell's palsy, Maladie de Raynaud, Graves' or Basedow's disease, Cruveilhier's paralysis, Parkinson's disease, Hodgkin's disease.

When the anatomical lesion which causes the symptoms is known, it furnishes the best designation of the whole train of structural and functional disturbance, as abscess of the liver, annular carcinoma of the descending colon, lithic-acid calculus of the kidney, cirrhosis of the liver, stenosis of the mitral valve, glioma of the middle lobe of the cerebellum, and so on. These, however, are rather diagnoses of morbid anatomy than names of diseased processes.

Better, perhaps, are the names which express the pathological processes that lead to the structural results, *e. g.* suppurative hepatitis, anterior poliomyelitis, tuberculous peritonitis, acute yellow atrophy of the liver.

Often, when neither the structural change nor the morbid process is certainly known, we must be content with clinical names, denoting a recurrent group of physiological events—a "complex of symptoms" the German writers call it—as Epilepsy, Chorea, Asthma, Chlorosis, Scurvy.

Arrangement of the present work.—As above explained, no scientific classification of diseases is possible, inasmuch as they are incommensurable objects. The most practically convenient arrangement is the best. For the reasons already given, the anatomical lesion is, when ascertained, the most definite and satisfactory basis of nomenclature and classification; when this fails, we generally name and group diseases by their most important clinical features, or in accordance with the organ most obviously affected; and these associations are often more important than affinities of pathology and causation. Thus scabies is more usefully studied along with other forms of dermatitis than with Bilharzia or hydatids, and phthisis goes more naturally with bronchitis and pneumonia than with lupus.

In accordance with general practice, from which it is inconvenient to deviate except for some sufficient reason, we shall take first among diseases those which have been long known as specific fevers affecting the whole body, and running a more or less constant and limited course. To these will succeed other general diseases, of the nature of which we are still ignorant, but which are more conveniently placed here than among local organic disorders.

The order in which we take the various local diseases is of little consequence. After considering the specific infective diseases, and others of uncertain pathology but certainly not purely local, we shall follow the traditional order of taking the most noble cavity, the Head, first. After the diseases of the brain and nervous system will follow chapters on the diseases of the Chest; then will come diseases of the Abdomen and Digestive Organs generally, of the Liver, the Kidneys, and the Spleen, with its lymphatic allies.

The next group will consist of scurvy and other disorders, accompanied by anæmia and hæmorrhage, which may provisionally be regarded as primary *hæmatoses;* rheumatism, gout, and other "idiopathic" and multiple affections of the joints (*arthroses*). The last section will deal with the numerous and complicated disorders which affect the skin and its appendages.

In each section we shall more or less closely follow the same order, dealing first with the name, symptoms, and history of the disease; then with its clinical characters, course and complications, and its diagnosis from other diseases; next with its anatomy and pathology, its ætiology and natural history. Lastly will come its prognosis and prophylactic, palliative, or remedial treatment.

Before, however, we begin the study of the several specific fevers, it will be well to devote a few preliminary chapters to certain pathological processes which are common to them and to many of the local diseases to be afterwards described.

These are contagion, fever, inflammation, pyæmia, and the formation of new growths. Other general pathological processes, as anæmia, dropsy, embolism, and the various forms of degeneration, will be better considered afterwards with the diseases of which they are the most important symptoms.

INFECTION

Οὐρῆας μὲν πρῶτον ἐπῴχετο καὶ κύνας ἀργούς
Αὐτὰρ ἔπειτ' αὐτοῖσι βέλος ἐχεπευκὲς ἐφιεὶς
Βάλλ', αἰεὶ δὲ πυραὶ νεκύων καίοντο θαμειαί.
HOMER.

Transmission of diseases—Contagion and miasm—Theory of infection—Contagium virum—Microbes—Their origin and life—Their exclusive effect in producing specific diseases—Their mode of action—Immunity from contagion—Protective inoculation—Modes of transference of miasmata and contagia—Theory and practice of disinfection.

FROM an early period it has been observed that certain diseases are contagious, or "catching," either by direct contact with the sick person, or by means of clothing or buildings, or by conveyance through the air of a presumed *materies morbi*. When actual contact was necessary to reproduce it the disease was called *contagious;* when it was transmissible by the air it was given the wider name of *infectious;* when the vehicles of transmission were recognised they were called *fomites*.* The distinction is not fundamental. For whether the pus from a case of smallpox is touched by the finger, or is transmitted by a handkerchief and then touched, or is dried up and carried by the air to a neighbour's house, the process is really the same—a speck of contagion from the sick person's body is transferred to that of one in health. And this is the only way in which diseases are "caught." The two adjectives contagious and infectious are therefore often used as synonymous. The word "*infective*" has been used of late to denote processes which spread, not always from one person to another, but from one tissue or organ of the body to another.

Infectious diseases frequently spread so rapidly that they become *epidemic*, and thus have devastated whole kingdoms and changed the course of history.

They have been, and still are, called *specific* diseases—partly from the special and peculiar course which they run, unlike that of diseases produced by injury or cold or poison or local inflammation—partly because each one was believed to be produced by a single definite efficient cause, the supposed *virus* or *contagium* or *materies morbi*, the presence and nature of which is now in many cases demonstrated.

There are, however, other "specific" toxic disorders which must be distinguished from the contagious diseases. These are—

(1) The effects of chemical poisons. Mercury, arsenic, lead, phosphorus, morphia, strychnia, when absorbed into the lymph and blood so

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* *Fomites, fomes*, touchwood, tinder, by which fire is conveyed:

"Ac primum silici scintillam excudit Achates,
Suscepitque ignem foliis, abque arida circum
Nutrimenta dedit, rapuitque in fomite flammam."
Æn., i, 174–6.

as to reach the tissues, produce definite and constant effects, special in their locality and symptoms, and pointing unmistakably to their "specific" origin. But these effects are strictly limited by the dose of the poison, and they are not transmissible from one person to another.

(2) The effects of certain organic poisons the chemical nature of which is imperfectly known. Such is the venom of snakes, a nitrogenous and proteid compound, probably an albumose; the albuminoid principle of jequirity seeds, called *Abrin;* and the remarkable ferment discovered by the late Dr Wooldridge, the injection of which produces instant coagulation of the blood in the portal vein.

(3) The effect of certain products of decomposition, causing septic poisoning: these are nitrogenous but alkaline, and probably compound ammonias. They have been called *ptomaïnes* or cadaveric alkaloids, *leucomaines*, and *toxines.*

In all these cases the poison is a chemical compound, and acts in proportion to its strength; moreover the morbid conditions produced are not transmissible.

(4) We must also separate certain diseases which are truly contagious, and depend on the presence of living organisms, but are only local in their effects, and do not produce any general physiological disorder. Examples are scabies and ringworm.*

(5) It is well to consider apart from other infectious diseases those which depend on animal parasites in the blood or tissue, such as malarial fever and dysentery.

(6) There remain the specific contagious febrile diseases, in which the contagion consists of a swarm of living organisms, *microbes,* which multiply in the host, set up a disturbance of the whole body, and render it for the time a focus of fresh infection.

Infectious diseases are also divided as follows:

(*a*) *Contagious diseases proper.*—Each of these maladies owes its origin to a *virus* or *contagium* derived from a person already suffering from it, or from one of the lower animals. Among these are the Exanthemata, Typhus, the Plague, Mumps, Whooping-cough, and Glanders.

(*b*) *Miasmatic diseases.*—These are caused by a contagium which is derived from the soil, or from the water, or from the air of a place, independently of the occurrence of similar illness in another person. Ague is an example of this group.

(*c*) *Miasmatic-contagious diseases.*—This name was proposed by Henle in 1840 to denote the fact that smallpox, scarlet fever, typhus, and other maladies often seem at first to break out epidemically, as though they might have been caused by a miasma, whereas afterwards the individual cases afford clear evidence of contagion. But Liebermeister and other writers in Germany assigned a fresh meaning to the term "miasmatic-contagious," and limited its application to a special class of maladies, of which Enteric Fever and Cholera are examples. These diseases undoubtedly spread by human intercourse, and are traceable to previous cases; but they are very seldom directly communicated from one person to another. Liebermeister's hypothesis was that patients give off a contagion which is inoperative until it has passed through further stages of development outside the human body, and that these changes may be taken as converting it into a kind of *miasm.*

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* A different use of the terms infectious and contagious is to confine the latter to diseases which are practically to be shunned as "catching," contagious in the popular sense, as scabies, ringworm, syphilis, gonorrhœa, typhus and variola, diphtheria and scarlatina. Infectious would then remain as the technical term to qualify all diseases transmitted by bacterial agency, including enteric fever, cholera, and relapsing fever, which are not, *as a rule,* catching or directly contagious from one person to another; but excluding (1) contagious diseases in which the presence of a specific microbe has not been demonstrated, as typhus and variola; (2) those in which the "contagion" is a poison, not an organism, as plumbism, sapræmia, and "intoxication," to use the French term, by ptomaines or toxines or other products of specific bacteria, by snake poison or by alcohol. (3) Those in which the contagion is a microzoon, not a microphyte, as malaria. This distinction appears to be arbitrary and inexpedient, but the decision in this matter does not depend on argument, but on usage.

The distinction is already obsolete, though it served a purpose in directing attention to the fact of some infectious diseases not being transmitted by direct contact with the sick. Thus tuberculosis, though occasionally acquired directly from a patient, is more often the result of microbes conveyed in dust to the lungs. Anthrax, usually directly conveyed by fomites, is sometimes acquired by cattle from the soil on which they feed In fact, the methods of transference are not constant for each infectious disease, but vary with the natural history of the living organism on which they depend. Some of these can live outside the human body, others cannot. Some can live in the lower animals, and others habitually live in inanimate objects, and only occasionally become parasitic in habit.

(*d*) An important distinction among contagious, infective, specific diseases is that in some of them (as relapsing fever) the poison is conveyed everywhere throughout the body by the living organisms which secrete it; while in others (as diphtheria) the microbes themselves do not spread far from the seat of invasion, but manufacture *toxines* there, which are conveyed over the body by the lymphatics and blood-vessels. In the former diseases the characteristic microbes will be found in the blood, lymph-spaces, or tissues; in the latter only in certain foci.

Theory of infection.—Contagia often appear as fluids; this is the case, for example, with the vaccine lymph, with the contents of smallpox vesicles or pustules, with the nasal discharge of glanders, and probably that of measles, and with the faucial secretion of diphtheria. But Chauveau in France, and Burdòn Sanderson in England, have demonstrated the fact that the activity of these fluids belongs not to chemical compounds dissolved, but to minute particles suspended in them. They have shown, for example, that when vaccine lymph is allowed to diffuse into distilled water through a porcelain diaphragm, the diffused liquid is incapable of conveying cow-pox by inoculation.

That contagia are "particulate" is therefore clear. The next point is as to the nature of the particles. For a long time the dominant view was that they were inorganic, or at least devoid of life, that their properties were essentially chemical, and that they acted "catalytically," or after the manner of *ferments.* On the ground of this analogy the epithet "zymotic" was coined, and applied to the whole class of infective diseases, and the contagia were called *microzymes.*

But ferments are either chemical like pepsin, and snake's venom, or particulate like yeast; and there is now little doubt that all particulate contagia which secrete a ferment are living organisms of exceedingly minute size, the microbes above mentioned. This conclusion is probable from the rapid multiplication of the virus its dry and cold, and its destruction by heat.* The actual proof, however, remains incomplete in the case of some of the most important and common of infectious diseases, as Measles, Smallpox, and Chicken-pox. We have in their case, however, the strong argument from analogy offered by the exact demonstration of the specific microbes in many similar diseases, of which the number is constantly augmenting.†

It is, however, important not to go before the strict demonstration of facts, and we may divide infectious diseases into three groups, the members of which are continually liable to shift, as fresh inquiries are made, from the third to the second, and from the second to the first group:—(1) Those in which the living contagion has been identified with a certain microbe, demonstrated to be its sole and efficient cause. (2) Those in which one or more microbes have been discovered which are probably specific. (3) Those in which no constant microbe has yet been discovered.

Of the first class the earliest members were two infective diseases, Relapsing Fever and Anthrax. To these may now be added Tuberculosis, Lupus, Leprosy, Diphtheria, Tetanus, Glanders, Erysipelas, and several forms of Septicæmia in the lower animals. To the second class belong Enterica, Cholera, Influenza, Pneumonia, Plague. In Syphilis, Typhus, Hydrophobia, Variola, Scarlatina, Measles, and Pertussis no characteristic microbe has yet been discovered.

The conditions which justify our regarding a given organism as the material cause, the *contagium vivum*, of a given malady, have been stated by Professor Robert Koch, the discoverer of the microbes of Tubercle and of Cholera, in the following three rules, to which we may add (4) and (5):

(1) The organism thus identified must occur in the blood or tissues—not merely on the surface, cutaneous or intestinal, but below the epithelium, in the lymph-spaces or blood-vessels—in every case of the disease in question.

(2) It must not occur in the human body except in cases of the particular disease in question. It may, however, conceivably exist in other animals without giving rise to the same pathological symptoms; or in air or water—as it exists in the test-tubes and plates of "pure cultivations," and in whatever media convey the contagion from one person to another.

(3) When a pure cultivation of the organism is introduced into the blood and tissues of an animal, the phenomena, clinical and anatomical, of the disease in question must be reproduced.

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* Lister, in a paper read before the Pathological Society in 1877, established that the lactic acid fermentation, or souring of milk, is essentially dependent upon what he termed the *Bacterium lactis*; and Pasteur had previously shown that the alcoholic and the butyric fermentation have a similar origin.

† The organisms concerned in these various processes differ remarkably in size, and therefore in the readiness with which the microscope reveals them. The *Torula cerevisiæ*, or yeast plant, which brings about the alcoholic fermentation, is made up of rounded cells which develop by budding, and have a diameter of $\frac{3}{10000}$ or $\frac{4}{10000}$ of an inch. The *Bacterium lactis*, which causes the souring of milk, consists of oval bodies, arranged in pairs or sometimes in chains, multiplying by fission, and measuring at the most $\frac{1}{20000}$ of an inch,—that is, being no larger than the granules which are contained in the cells of the torula. Consequently, as Lister argues, there is nothing improbable in the supposition that yet other organisms may exist which may be as much smaller than the *Bacterium lactis* as it is smaller than the torula. Heydenreich aptly remarks that, were it not for the length of the spiral threads which form the microbe of relapsing fever, it would in all probability have escaped detection; rounded or oval bodies not exceeding in diameter the breadth of the *spirillum* would be almost invisible with the highest powers of the microscope. —C. H. F.

(4) The malady must have such distinct and constant features, whether clinical or anatomical, as shall enable it to be identified.

(5) The microphyte must be itself distinguishable from others by its size and shape, its staining properties, but above all by its mode of propagation in "a pure cultivation" (*i. e.* artificially separated from other organisms) and by the form and colour of the colonies it produces.

These conditions in all their rigour have been satisfied for the diseases in the first class above mentioned.

The following are the most important parasitic microphytes. They all agree in not containing chlorophyll:

I. Moulds (*Mucorini*, *Hyphomycetes*, *Schimmelpilze* of Nägeli). These are long-branched filaments, which form numerous spores. To the pathologist they have limited interest, but one species is sometimes found lining the interior of dry vomicæ in the lungs (*Aspergillus fumigatus*), and others are found in the external auditory meatus. Similar fungi cause certain cutaneous affections, as Ringworm and Favus.

II. Budding fungi (*Saccharomycetes*, *Blastomycetes*, *Sprosspilze* of Nägeli). These consist of rounded or oval cells, which give off buds, and may form beaded threads. To this group belong Saccharomyces, Torula, and Mycoderma. Another fungus (*Saccharomyces capillitii*) has been recognised on the hairy scalp and elsewhere on the skin. Two remarkable pathogenic fungi are found in cases of madura foot in India (*Chionyphe Carteri*) and of Actinomycosis, but their identity and relation to each other and their botanical position are still subjects of controversy.

III. Fission-fungi (*Schizomycetes*, *Spaltpilze* of Nägeli, often called Bacteria generally). These are minute unicellular plants, which multiply by spontaneous division, with or without production of endogenous spores.

1. Spherical bacteria (*spherobacteria* of Cohn, *coccaceæ* of Zopf, *micrococci* or *cocci*) are exceedingly minute. After fission they sometimes cohere in pairs (*diplococci*), sometimes in chains or garlands (*streptococci*), and sometimes in heaps or clusters (*staphylococci*), and sometimes they are aggregated into masses held together by a jelly-like material (*zooglœa*). *Sarcinæ* are micrococci arranged in square or cubical packets, the results of fission in different planes.[*]

2. *Bacilli* (*desmobacteria* of Cohn; *bacteria* in the restricted but almost obsolete sense, *microbacteria* of Cohn) are cylindrical rod-shaped bodies, which sometimes remain united after they have undergone fission, so as to form threads of considerable length. They are often slightly constricted in the centre, or dumb-bell shaped. *Closteridia* are fusiform bacteria. Many bacilli are flagellate and motile, others appear to have no power of movement.

3. *Spirilla* and *spirochætæ* are spiral filaments (*vibriones*, *spirobacteria* of Cohn), having a well-marked corkscrew motion.

These names are all descriptive of shape and size, and not indicative of botanical or pathological relations. The same microbe may be described as a short bacillus or an oval or lancet-shaped micrococcus. A coccus may appear in certain conditions alone, in others as a diplo- or streptococcus.

A more important distinction is between microphytes which form endo-

[*] By Billroth rod-shaped and spherical bacteria were associated together under the name of *coccobacteria*. By earlier authors the bacteria and bacilli were separated, but the present tendency is to use bacteria as a general term for fission-fungi and bacillus for

genous spores, like *Bacillus anthracis*, and those which multiply by fission only. Another is the reaction of the microbes to stains, and the most important of all is their behaviour when cultivated in broth, gelatine, or other suitable media or on solid surfaces. Lastly, there are bacteria which are indistinguishable in form, size, staining, or cultivation, which nevertheless are proved to be different by their pathological effects on the lower animals.

Some bacteria live in air (*aërobic*), others only excluded from air (*anaërobic*), and many in both conditions (" facultative aërobism ").

An important distinction is that made by Flügge between *septic* (*saprogenic*) or putrefactive microphytes, *zymogenic* or fermentative, *chromogenic* or pigment-forming, and *pathogenic* or productive of specific diseases.

Zopf carried Nägeli's and Billroth's doctrine of the " pleomorphism " of the schizomycetes to its utmost limit. But more critical biologists admit variation in form of one and the same organism only within certain narrow limits. This was first proved by Lankester in the case of *Bacterium rubescens*, a chromogenic microphyte, since named *Clathrocystis roseo-persicina* by Cohn (' Quart. Journ. Micr. Soc.,' 1876).

There is no reason to believe that chromogenic microphytes can be cultivated into zymogenic, or septic into pathogenic. The assertion by Buchner that the common *Bacillus subtilis* of infusion of hay was interchangeable with the bacillus of anthrax, and *vice versâ*, was refuted by Klein; and it seems probable that the *Bacillus coli communis* is specifically distinct from the typhoid and other pathogenic bacteria.

Pathogenic bacteria, however, may undoubtedly suffer diminution of their malignancy (or may acquire it in intenser degree) by being passed through a series of hosts. This is the explanation of Jenner's vaccination, for there is little question that the cow-pox is variola passed through successive generations of cattle. On the same principle Pasteur succeeded in "attenuating" the virus of anthrax, of so-called "chicken cholera," and of hydrophobia, and thus in protecting against the unmodified disease by inoculation (or, as he called it, " vaccination ") with the attenuated contagium.

It is often convenient to be able to refer to bacteria under a common name without specifying their exact characters; and for that purpose the word *microzyme* was suggested by Béchamp and adopted by Sanderson. *Microphyte* is perhaps a better term. *Microbe* was the one used by Pasteur. But bacteria is now commonly used as a general term of all pathogenic fission-fungi.*

The long recognised contagious properties of pus depend on the presence of certain micrococci first described by Ogston in 1881, and cultivated by Rosenbach, Garré, and other pathologists, *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, *S. pyogenes albus*, *Bacillus pyocyaneus*, &c.

Specific diseases never arise *de novo*, independently of contagion from previous cases. No doubt all such diseases must have had a commencement at some period of the world's history, just as animals and plants must have had a beginning; but as physicians we are only concerned with the question whether specific diseases ever start afresh in the world as it is ; and the same answer must be given as to the question of spontaneous generation of animals and plants. When the outbreak of a contagious malady has been investigated with sufficient care, it can always be traced to a previous case, from which it has been derived in some indirect and unexpected way.

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* The "genera" bacterium, vibrio, spirillum, and spirochæte, date from Ehrenberg's great work on infusoria (1838); the other terms have been since introduced. *Bacterium* (*βακτήριον*) means a little rod or staff: *bacillus*, a little stick or rod; *closter* (κλωστήρ), a spindle; *coccus* (κόκκος), a berry; *mucor*, mould; *myces*, pl. *mycetes* (μύκης), a fungus; *schizomycetes*, splitting fungi; *sarcina*, a package; *zooglœa*, living glue.

We may therefore absolutely reject the doctrine that any of the specific diseases can be produced by dirt, overcrowding, or starvation alone; or even by the inhalation of sewer gas which contains no specific virus.

It is, however, probable that the microphytes of an infective malady, after escaping from patients, may sometimes go on multiplying for long periods of time, and spread to distant localities, before they again enter the human body. In the miasmatic-contagious diseases such a process seems to be the rule; and in some also of the strictly contagious diseases there is reason to believe that it is a stage in their development.

Some infectious diseases may be caught from the lower animals. Ringworm and anthrax are instances. So is, in all probability, diphtheria (especially from cats and birds) and influenza (from horses), while glanders and hydrophobia are always derived by infection from a horse or a dog respectively. Another important fact of this kind is the transmission of tubercle from the udder of a cow by means of the milk.

Course of infection.—In most cases, when pathogenic microbes enter the body, an interval follows during which the health of the patient remains apparently undisturbed ; this is called the period of *incubation*, and is often of remarkably definite duration. We are thus enabled to say, when a person has been exposed to infection, that after a lapse of a certain number of days or weeks, he is safe, and may mix with other people without risk. On this foundation rests the practice of *Quarantine*. The incubation-period of each specific disease can be positively determined when there has been only a single exposure to contagion ; but others, in which the exposure (though repeated) began only a few days before the patient's illness showed itself, are valid as proofs of short incubation ; and others again, in which the exposure ceased many days before he fell sick, are valid as proofs of a long incubation. The interval of incubation is no doubt occupied by the multiplication of the virus within the body. According to Nägeli, schizomycetes commonly double their numbers in from twenty to twenty-five minutes at the temperature of the blood. Lister found that the *Bacterium lactis* took about an hour in completing the process of growth and subdivision. There is evidence that in the rare instances of the syphilitic poison being communicated by vaccination, the resulting chancre has yielded an infective material ten days afterwards, much more rapidly than in ordinary cases.

An unfortunate case once occurred at the Charité Hospital of Berlin, in which variola was conveyed to a patient by the operation of skin-grafting, the graft having been taken from a person who happened to be in the incubation stage of the disease. This seems to show that from a very early period the whole of the skin contained the virus, which can have reached it only through the blood; and if so, the same thing must occur likewise in the exceptional instances in which smallpox is inoculated into the skin at a particular spot.

In many specific diseases there are certain characteristic local lesions or [illegible] [illegible] induration of a syphilitic sore, the pustules of variola, the tonsillitis of diphtheria, the intestinal ulcer of enteric fever, the parotid of mumps, the buboes of Oriental plague. These are at once the result of the infection from without, and a fresh form of infection from within the body. The former is their constant, the latter their occasional significance.

In typhus and in relapsing fever there are no characteristic local lesions; and, in the exanthemata, pyrexia and other general symptoms precede the cutaneous eruptions by a considerable interval of time.

Here, however, we must distinguish between such cases as Diphtheria, Syphilis, and Tetanus, where the local focus is the seat of multiplication of the contagion, and cases like Tuberculosis, where the contagion rapidly spreads over the whole body. Hydrophobia is probably an extreme example of the former group of diseases and the exanthems of the latter.

Physiological action of the microphytes; their poisonous products.—With regard to the mode of production of the pyrexia and of the other symptoms of specific diseases by their contagia we know at present scarcely anything. The microbes, in multiplying so enormously as they do, must of course abstract nutrient materials from the blood and from the tissues; and like other plants without chlorophyll they must absorb much oxygen from the blood and lymph. It is, however, rare for them to increase to such numbers as to act as emboli, or to starve or to stifle the tissues they invade.

The most general, important, and in some cases the sole physiological action of infectious microbes is by means of the chemical poisons they secrete. These are at present imperfectly known. The first described were alkaloids of the amine type called *ptomaines* (from *πτῶμα*, a corpse), leucomaines, &c. Others, also nitrogenous, are proteid in structure, and resemble the intermediate products of gastric or pancreatic digestion, between albumen and peptone. They are therefore called *albumoses*. Other products are fatty and aromatic acids, indol, &c. A convenient term for all such chemical products of infective microphytes is *toxine*. Dr Sidney Martin has isolated the toxine of Diphtheria, and has shown that it is possible to identify this infectious disease not only by the specific microbes which produce it, but also by the specific toxines which they produce. This has also been accomplished in the case of Anthrax, Diphtheria, and Tetanus.

In some diseases which are due to infection with pathogenic microbes the organism itself does not spread beyond a limited area, and all the effects of the disease on the nervous system, heart, and tissues generally, are due to poisoning with the chemical products of the bacteria. In fact, it is not too much to say that most of the symptoms of specific fevers are due to "intoxication" or poisoning with chemical products: in some the pathogenic microbe penetrates to every corner of the body, and there distils its venom; in others it multiplies in one or more limited spots, and there secretes the poison which is carried by the lymph and blood all over the body.

The idea of a "struggle for existence" between these organisms and the tissues of the host was carried out in detail by Nägeli. He argued that when a person is exposed to contagion, his taking the disease or resisting it depends probably in part upon the number of microbes which enter his body, in part upon the condition of his blood and of his tissues at the time. It is found in cultivating bacteria that when more kinds of organism than one are present, a slight change in the composition of the fluid in which they are placed may completely alter the result, enabling a microbe which previously was weaker to outgrow that which had been stronger. And so Nägeli supposed that slight alterations in the state of the blood may greatly favour or oppose a contagion in the competition on which it enters. Moreover the remarkable observations of Metchnikoff, first made on a minute crustacean, but afterwards repeated on frogs, seem to show that bacteria introduced into the blood either kill the leucocytes, or are themselves absorbed and digested by these guardians of the invaded organisms (' Virchow's Archiv,' Bd. xcvii).*

This action of the leucocytes on the invading bacteria is termed chemiotaxis. But the power of resistance of the invaded organism is not limited to the action of the living leucocytes, for blood-serum itself may be antagonistic to the life of bacteria.

The analogy of the various fermentations suggests (as Lister has remarked) that, besides appropriating the materials for their own growth, microzymes may also cause further disturbance by catalytically decomposing other substances of which they make no use. They also secrete digestive products, which act like pepsin in softening the tissues.

While living, these minute organisms are constantly excreting waste products, and these, when accumulated by an enormous number of microbes, become a large dose of poisonous materials for the host. Moreover, the mere physical results of immense swarms of these microbes may possibly lead to blocking of lymph-channels, or starvation of ganglion-cells, or suffocation of living protoplasm.

Immunity.—It is a matter of common experience that most specific infectious diseases protect from future invasion the organism which has once survived their attack. There are exceptions to the rule, and the degree and duration of the protection varies for each malady; but most of the specific contagious diseases occur only once in the life of the same person, and few of them are entirely devoid of a protecting influence.

Those which protect best are Smallpox, Typhus, and Whooping-cough, of which a second attack is very rare indeed. Scarcely less infrequent is a second attack of Enteric Fever, Varicella, Mumps, or Syphilis. Scarlatina protects rather better than Measles or than Rubeola. Cholera and Plague probably protect, but they are comparatively rare and their mortality is high. Yellow fever is believed to protect efficiently from a second attack if the patient survives the first. Relapsing Fever scarcely protects at all. Glanders, Tetanus, and Hydrophobia are too often fatal to furnish instances of protection. Lastly, Erysipelas, Influenza, and Pneumonia not only do not prevent a second attack but probably predispose to it.

It appears as if in the course of each disease the blood or the tissues undergo such a "sterilisation" that they no longer afford the conditions requisite for the development of its peculiar microphytes; and it has been found experimentally that by cultivation of a certain bacillus in a neutral fluid the latter becomes so "exhausted," or at least so altered, that it will no longer support cultivation.

There are, however, obvious objections to the notion that human beings in general are born provided with a number of different materials which serve no known purpose but that of affording nutriment to the various contagia, in case of their invasion. Nor is it easy to suppose that the leucocytes, when once they have got the better of the invading swarms of bacteria, are so habituated to the attack that they gain an easy victory over any subsequent irruption of the same species, while yet they are as unable as at first to deal with any other species; and that they transmit to their descendants the same power of destruction, with the same limitation, down to a generation of phagocytes many years distant.

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* See Metchnikoff's 'Lectures on the Comparative Pathology of Inflammation,' translated by Dr Starling (1893).

The immunity from Scarlet Fever of a person who has once had that disease is an *acquired* immunity. But there is also a natural immunity from Scarlet Fever which protects some who are exposed to the infection. This may depend in some cases on accidental circumstances which hinder the entrance of the contagium; but when it has gained an entrance, when contagious matter is inoculated, some persons and some animals resist and are naturally immune to that infection, though they may suffer from another. This is probably due to the phagocytic power of leucocytes, and partly to the destructive power of serum; but even if one or both theories are universally true, they only carry the explanation further back a step. How a single cell resists one microbe is as hard a problem as how a colony of cells, an entire organism, resists a swarm of microbes.

Natural immunity appears to be hereditary—acquired immunity probably is not. Some species of animals resist one specific disease absolutely, and others comparatively. Thus rodents are prone to tuberculosis, while dogs and horses are "refractory" but not immune.

That immunity is not a mere question of phagocytosis seems clear, not only from its being true of diseases, like diphtheria, which are immediately the result not of bacteria, but of the toxines they secrete, but also from acquired immunity applying to purely chemical intoxication like belladonna, arsenic, and alcohol.

Artificial immunity follows the same rule of "habit, use, and wont." An animal is first inoculated with a small dose of virus and recovers, it can then bear a larger one, and at last becomes immune to what at first would have been a lethal dose. Natural, acquired, and artificial immunity are all more or less temporary as a protection. Vaccination must be repeated. After many years an attack of Measles no longer protects, and the artificial immunity from Diphtheria only lasts a few weeks. Thus protection varies as much in duration as in degree.

It is an interesting question whether acquired immunity (as from Typhus or Measles) may not, in some cases, be transmitted from parent to child. It is well known that exanthemata often rage with extraordinary virulence when they are introduced into communities that have before been free from them. Other explanations of this fact are possible; but the reason may be that the victims come of a stock which has not for many generations been exposed to the contagion. It would be interesting to investigate to what extent differences in the severity of a specific disease, attacking various individuals under the same conditions, in countries where it is always more or less prevalent, may depend upon whether one or both of the parents of the several patients had that disease in childhood.

Prevention of infectious diseases.—We cannot "stamp out" scarlatina and cholera as we can anthrax or pleuro-pneumonia in cattle—by destroying each patient as soon as he is attacked; and if we could, sooner or later each disease would be sure to find an entrance, and it would probably commit unheard-of ravages among a population long free from it. It is even doubtful whether we ought to teach the public to look upon the maladies in question as in a special sense "preventable by segregation of patients." They are certainly not so much so as those which result from over-indulgence in food and drink, or from syphilis.

A more promising method of combating infection is that of which we have an illustration in vaccination. Pasteur demonstrated the fact that the virus of anthrax can be so diminished in intensity that it is no longer fatal to cattle, and that after inoculation with this modified or attenuated virus they are no longer susceptible of the disease. The same success attended him in dealing with a contagious disease of poultry (called by a misnomer *cholèra des poules*); and he afterwards applied a similar method to the prevention, and practically to the treatment, of hydrophobia. We may hope for future advance in the same method, which has made small-pox a preventable though unhappily not always a prevented disease. Similar success now follows the elaboration of an antitoxin of diphtheria in the serum of horses rendered immune to this disease. Injection of this serum under the skin not only confers a temporary immunity from infection, but in the case of those already attacked by diphtheria greatly reduces the violence of the disease and increases the number of successful cases. Pneumonia, tetanus, and suppuration have been attacked by a similar method of treatment, and with some promising results, and still more recently Plague and Enteric Fever have been treated with encouraging success by the same method.

There is, lastly, another method by which plague and typhus and ague have been almost banished from England—improvement of the circumstances of life, by which either the organism is better able to resist the invasion of the virus, or the conditions have been rendered unfavourable for the multiplication and even the survival of the living parasite itself.

Method of transference of contagia.—We have next to consider how the microphytes of contagion gain access to the human body.

When suspended in the air they probably penetrate into the capillaries of the lungs through the stomata of the pulmonary alveoli; they make their way through the mucous membrane of the mouth or the fauces by the mucous crypts, particularly those of the tonsils; or they enter the blood or the lymph through wounds or abrasions.* After being swallowed the bacteria pass through the mucous membrane of the alimentary canal, and are distributed by the blood-current.

In describing the invasion of the several specific diseases, we shall find evidence that their contagia differ much in the readiness with which they pass the natural barriers of the skin and mucous membrane.

Contagia have traditionally been divided into those which are "fixed" and those which are "volatile." As we have seen, it is certain that none of them are gases or vapours, and that they all consist of solid particles of exceedingly small size. But is it possible that the particles in question are capable of escaping with the water which evaporates from liquids or from moist solid surfaces, and of diffusing themselves in the air? Nägeli demonstrated that this is impossible. He performed a series of experiments with V-shaped tubes connected together. In one bend he placed a liquid suitable for the growth of microzymes; another bend he filled with sand saturated with a putrefying liquid. Even when air was drawn through the apparatus he found that no microzymes passed from one tube to the other. And on theoretical grounds he shows that no other result could have been anticipated. Later observations on the transference of the contagion of Enteric fever, tubercle, and septicæmia have confirmed these early conclusions. It is, however, quite possible for bacteria to be conveyed in splashes or in mucus ejected from the nose or the trachea.

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* It has been supposed that examples of such a mode of infection are sometimes afforded by the so-called "surgical scarlet fever," which so often breaks out in children after operations; but, since Dr Goodhart showed in the 'Guy's Hospital Reports' for 1879 that its occurrence is not prevented by the strictest "antiseptic" treatment, there appears to be more probability of its arising in the ordinary way

It was formerly supposed that the breath of persons suffering from an infective disease is highly charged with the virus, and that the offensive odours exhaled from his moist skin, or from his excreta, are highly dangerous. But this belief is erroneous; the presence of fœtor is generally a sign that the substances which give rise to it are still moist, and are therefore incapable of setting free any microzymes which they may contain. Increasing experience has shown that the fear formerly entertained of microbes floating in the air was greatly exaggerated. The contagion in cases of wounds and in surgical operations is conveyed in the vast majority of cases by the hands of the patient or others, by the instruments used, or by accidental contamination. So also contagia from the lungs and throat are conveyed by the sputum or saliva ejected in coughing, but not by the moist breath; and it is only when dried up that the dust becomes again a source of infection through the air. Pathogenic microbes generally seem to require a conveyance—water, mucus, fæces, epithelium, &c. (moist or dried), and do not exist to a dangerous extent free in the air.

Some infectious diseases, as tetanus, hydrophobia, and syphilis, appear to be always communicated by *inoculation,* the poison being introduced into a bite or a wound, or a crack in the skin. This is also the case with the suppurative micrococci. Diphtheria is often, and syphilis occasionally, spread by direct transference of mucous secretion from one individual to another. Again, punctures made by insects may sometimes be the means of conveying the virus; cattle, for instance, are liable to be infected with anthrax by gadflies, and we shall find that mosquitoes are important transveyors of the living contagion of ague from water to a human host.

Some specific diseases, as enteric fever and cholera, are transmitted by means of *drinking-water.* The contagia of enterica and of scarlatina may be spread by means of milk, and that of tubercle by milk, and less frequently by solid food. It is probable that, even in water, the specific microzymes multiply in the interval between its contamination and its reaching the human stomach. But there can be little doubt that milk affords more favourable conditions for their growth.

Most bacteria when swallowed are destroyed by the gastric juice, but, unfortunately, those of enteric fever, and of tuberculosis, particularly in children, pass safely on into the intestines and there multiply.

The conditions which favour infective bacteria being conveyed by the *air* vary greatly.

In the case of microbes which live in earth, the first condition of their development is the presence of water in the interior or upon the surface of the ground, and the presence in this water of substances fit to serve as pabulum for the microbes. Further, it is necessary that the level of the water should vary from time to time, so that particles of the soil itself, or the stems and the leaves of plants growing upon it, should undergo desiccation.[*]

The contagia of smallpox (dried and powdered pus), of scarlatina (epidermic scales), and of diphtheria and tubercle (desiccated mucus) gain an entrance by the fauces and the air-passages generally.

Contagious microbes leave the body mainly in the fluid excretions—the saliva, the urine, the fæces, and the sweat. They may also escape with the cuticle which is constantly being shed from the surface; in the case of scarlet fever and in that of smallpox this mode of diffusion of the poison is of the first importance.

The microbes which adhere to fragments of cuticle are already dry; those which are contained in fæces, in urine, saliva, or sweat must undergo desiccation before they can reach the air.

Free ventilation and exposure to direct sunlight are deleterious to bacteria, whereas nothing is more favourable to their disposal than soaking linen or other fabrics with any of the infected secretions. The microbes remain upon the surface of the cloth when it dries, and are afterwards shaken off by the slightest movement.

The escape of microbes in the course of an infective disease is accidental, not due to a process of "elimination," and of no advantage whatever to the patient. Indeed, the microbes which find their way out of the body are probably altogether insignificant in number when compared with those which remain in the blood or in the tissues.

Persistence of contagia.—How long a patient remains capable of infecting others after his recovery from a contagious disease is a most important question, but difficult to answer. Almost as soon as the pyrexia is at an end, *spirilla* disappear from the blood in relapsing fever. Typhus and smallpox, when the last symptoms have disappeared, appear to lose their power of contagion. On the other hand, the desquamation of scarlatina prolongs the chance of infection for many weeks, and that of diphtheria may long exist in the fauces when it has disappeared from all other parts, and when the patient himself is perfectly well.

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[*] If any value is to be attached to the observations which Pettenkofer made at Munich with regard to enteric fever and to cholera, microbes concerned in the propagation of these "miasmatic-contagious" diseases must be assumed to come in some cases from the soil at a depth of several feet. The conditions which may be supposed to render this possible are very carefully argued out by Nägeli. In the first place, he thinks that in the ground-water which saturates a porous soil below a certain level the growth of microphytes is likely to be especially active towards the surface of this water. Hence the slightest fall in the level of the water must cause an abundant settlement of microbes upon stones, sand, and fragments of clay and humus, which come into contact with air as soon as the water recedes from them. In this subterranean atmosphere he conceives that currents are produced by various causes—changes of temperature, changes of pressure, winds, and lastly, the suction action of warm air into the interior of houses having deep foundations. Such currents, he thinks, may easily carry away the dry microbes, especially if there is no precipitate of colloid matter from the water to make them adhere closely to the substances on which they are deposited. A further condition, at least so far as concerns their escape into the air above, is that the superficial layers of the soil through which they must pass should not be damp, and should not be covered with a uniform carpet of vegetation.—C. H. F.

Contagia sometimes adhere with great tenacity to the walls of a room or a house which has been tenanted by a patient,* and to bedding or clothes which he has used.

Woollen substances seem to afford the most favourable conditions for the preservation of microbes in a state of activity, short of their being enclosed in sealed glass tubes. By the hygrometric properties of such substances, contagia adherent to them may probably be prevented from undergoing too complete a desiccation, while they are at the same time protected from currents of air.† Of the contagious diseases plague and erysipelas are most apt to cling to walls and furniture.

Destruction of contagia.—Disinfection of persons, of their clothes, and of their dwellings after a contagious disease was enjoined by the Mosaic laws, and was carried out in modern Europe after epidemics of the plague. But only since the presence and natural history of living contagia have been discovered, have such methods been capable of scientific direction.

The patient himself, his physician, and his nurses, the linen of his bed or body, his excreta, the furniture, and the walls of his chamber are each and all sources of possible infection.

The patient should be isolated in a separate room or suite of rooms, if possible, at the top of the house. An intervening passage, of which the windows can be kept open, is a great advantage. Ventilation should be carefully attended to, and there should be a fire, so as to maintain a draught up the chimney. The doors should be kept closed, and outside them there should be hung an old sheet saturated with a liquid solution of carbolic acid (1 in 40), and never allowed to become dry. The addition of glycerine is useful for the latter object. It is useless to expose vessels containing carbolic acid in a sick-room itself, or to scatter chloride of lime upon the floor.

Those who nurse or wait upon the patient should not be allowed to enter other parts of the house, and, if possible, persons should be chosen for this duty who have already had the disease. The medical attendant should take every possible care to avoid carrying contagion abroad by change of clothing, and by exposing himself freely to the open air after each visit, or by covering his ordinary dress with a long mackintosh or cotton gown. He should never see a patient with an infectious fever while fasting, and should scrupulously cleanse his hands after each visit.

All curtains, carpets, and padded chairs, all articles of wearing apparel in cupboards or in drawers, all unnecessary furniture, should be removed from the sick-room before the patient is placed in it. Only such books or toys should be allowed as are not too valuable to be burnt when they are done with. Any food or drink which the patient or the nurse may leave should be thrown away. Cups, plates, and spoons should be put in boiling water before they are allowed to go back to the kitchen; but, as far as possible, it is well to keep the same articles from day to day in use in the sick-room, and to wash them there.

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* Nägeli mentions that at Munich several masons fell ill with smallpox after scraping the ceiling of a room in which smallpox cases had been treated six or seven years before, and which had then been whitewashed.

† Watson's lectures contain the following instance:—A house in which several persons had been attacked by scarlet fever was left empty for a year. When the family returned, a drawer in one of the bedrooms resisted for some time attempts to pull it open. A strip of flannel had got between the drawer and its frame, and had made the drawer stick. This piece of flannel the housemaid put playfully round her neck. An old nurse who was present, recognising it as having been used as an application to the throat of one of the subjects of scarlet fever, snatched it away and burnt it. The girl, however, soon sickened with the disease.

All the excreta of the patient should be rendered innocuous by the addition of a disinfectant, crude sulphate of iron or permanganate of potash or chloride of lime. Other effectual disinfectants are thymol and other aromatic compounds, phenol ("carbolic acid"), sulphurous acid, chlorine, and—in some respects the most valuable of all—corrosive sublimate.

A point on which Baxter laid stress,[*] as Nägeli had before him, is that the addition of disinfectants in too small quantities may do harm instead of good. Probably all the substances that can destroy contagia are also antiseptic agents; that is, they can prevent putrefaction. And it appears that they are capable of doing this even when present in too small a quantity to disinfect. So that if a disinfectant is added in small quantity, it may easily happen that by preventing putrefaction it keeps the contagious microbes alive.

For disinfecting linen and utensils and sterilising instruments heat is the most effectual agent. There is reason to believe that a degree of heat below 212° may be sufficient. Davaine found that the virus of anthrax diluted with water was destroyed in five minutes by a temperature of 131° F., and Baxter that even dry vaccine was rendered inert in thirty minutes by a temperature of 185° F. In the dry state contagia resist heat much better than when they are suspended in water; so that steam saturated with watery vapour is better than dry steam.

Some hospitals for infective diseases have hot-air chambers, erected for the purpose, in which there is no difficulty in raising the temperature, even in the centre of a flock-bed, to 220°, 240°, or 300° F.

The room which has been occupied by the patient during the disease must be purified by removal of wall-papers, scrubbing the floor, and scraping and whitewashing the ceiling.[†]

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[*] Sixth volume of the new series of Sir John Simon's 'Reports to the Privy Council.'

[†] Since the first edition of this book appeared many excellent treatises on the subject of microphytes and contagion generally have appeared in England. Of these may be particularly mentioned Dr Klein's 'Micro-organisms in Relation to Disease,' Dr Crookshank's 'Introduction to Practical Bacteriology,' Mr Watson Cheyne's original papers and his translation of German essays on 'Bacteria in Relation to Disease' (published by the New Sydenham Society, 1886), the chapters on contagion and on bacteria in Professor Hamilton's text-book of pathology, and in the admirable 'Student's Manual of General Pathology' by Dr Payne (1888); also Dr Kanthack's able treatise in the first volume of the 'System of Medicine' edited by Professor Allbutt, and the practical directions in Drs Wooldridge and Goodall's 'Manual of Infectious Diseases.' The best short account of bacteria recently published is that by Dr Muir and Dr Reid (2nd edit., 1899).

FEVER

Præterea minimus gelido jam in corpore sanguis
Febre calet sola.—Juv., Sat., x, 217.

Pyrexia—Theory of fever—Thermolysis and thermogenesis—Thermotaxic nervous centres—Thermometry—Clinical and pathological course of pyrexia—Hyperpyrexia and paradoxical temperatures—Idiopathic and symptomatic fever—Raised temperature without fever—Pathological forms of pyrexia and its clinical varieties—Concomitant phenomena of pyrexia: nutrition, pulse, respiration, &c.—Subnormal temperature.

Galen defined fever as *cœlor præter naturam*, and fever was rightly believed by the physicians of Greece and Rome to be essentially a condition of unnatural heat (*πυρετός, πῦρ; febris, ferveo*). The touch of the fevered patient's body, his thirst, and the consumption of his tissues were regarded as due to an "inflammation" of the blood, and the whole process was regarded as an exaltation of the "vital heat" which was supposed to be the cause, not the effect, of the movements of a living body. By Aristotle and his successors down to the experiments of Hooke, Boyle, and Mayow in the latter part of the seventeenth century, the method of moderating the vital heat was supposed to be the cooling action of the air in respiration, and hence dyspnœa and fever were naturally associated.*

We now know that the bodily temperature in health is maintained at a nearly constant point, partly but only in a subordinate degree by the lungs, and principally by evaporation from the skin. We know that radiation and conduction are prevented by clothing, and that perspiration is checked when the surrounding air is colder than usual, while it is given free play and the skin becomes hot and moist, when the air rises in temperature.

Rightly regarding the increased temperature of the body as the essential and dominating symptom of fever, of which thirst, high-coloured scanty urine, dry tongue, burning skin, anorexia, insomnia, muscular weakness, emaciation, delirium, and coma are all results, modern pathologists have concentrated their attention upon this fundamental fact.

Now it is quite clear that a higher mean temperature in a living body, as much as in a greenhouse or an oven, can be the result of only three causes, viz. increased production of heat, diminished loss or dissipation of heat, or the two acting together.

* See Dr Wm. Ogle's introduction to his translation of Aristotle's treatise on 'Respiration,' 1897.

It is convenient to make a distinction between Fever the complex pathological state known under that name, and Pyrexia the condition of raised temperature as a fact by itself. The word fever should never be used as a nosological term without a qualifying adjective.

When Liebermeister, in 1870, said that in fever Nature strives to keep the mean temperature at 103° (or whatever the pyrexial state is), just as in health she strives to keep it at 98·4°, he spoke metaphorically; but he was right in holding that the same causes produce "morbid" pyrexia as those which produce normal or "healthy" apyrexia.* In other words, the pathology of fever is only a chapter in the physiology of animal temperature.

Physiology, then, teaches us that the high mean temperature of fever must depend on increased oxidation, or on diminished output of heat from the skin or the lungs, or on both causes acting together.

Traube, who was the first to study fever physiologically (1863), believed that the primary fact in pyrexia was the contraction of the capillaries of the skin, which led to the whole blood becoming hotter. The production of heat was, he thought, no greater than in health, but its dissipation was checked. He also first proved that the excretion of nitrogen is increased, chiefly as urea. The excretion of carbon dioxide was at one time believed from experiments on animals not to be increased during pyrexia, but it has now been satisfactorily proved to undergo marked increase during fever in the human subject by Leyden, and the same result has been arrived at by Pflüger and his pupils in the case of animals also. Rosenthal ('Berlin. klin. Wochenschrift,' August 10, 1891) has since repeated Traube's experiments, and come to the same conclusion.

That pyrexia does not always depend on diminished loss of heat is proved by the profuse sweats of rheumatic fever, and by the inefficacy of hot-air baths in reducing high temperature. Again, as Cohnheim remarked, a febrile patient may be thrown into profuse perspiration by the subcutaneous injection of pilocarpine, when the loss of heat from the skin must be enormously increased, and yet no fall in his temperature follows. In fact, the heat passed off in fever may be more, not less, than in health. It seems, therefore, clear from both experiment and clinical observation that in some, if not in all cases, there is an increased production of heat, associated with diminished muscular movements, with increased oxidation, and with increased excretion of nitrogen, of water, and of carbonic anhydride.

In fever, as in health, the regulation of temperature still goes on, but the mean point is a higher one. Dr Burdon Sanderson, in his lectures on fever in 1876, gave abundant evidence that pyrexia does not depend on diminished dissipation of heat, but on disorder of the thermotaxic mechanism. That is to say, that, as Virchow surmised in 1853, fever is essentially a disorder of the nervous system. The loss of heat, normally regulated by thermolytic nerves which govern the cutaneous blood-vessels and sweat-glands, and the respiratory movements, the muscles and the glands throughout the body—this loss of heat may in pyrexia be less than in health, or it may be and often is greater; but the production of heat regulated by thermogenic nerves is always increased, so as to leave the temperature of the body above that of health. This view, though ably disputed by Swatow, who adhered to Traube's doctrine, has been confirmed by Cohnheim ('Allgemeine Pathologie,' Bd. ii, S. 539), Leyden, May, and other German pathologists. The difficulties of observation and experiment, as well as of theoretical adaptation of the physical laws of production and dissipation of heat to the human body, are so great, that it is not wonderful that it is not yet clear whether there is in this or that particular case of fever decreased thermolysis in addition to increased thermogenesis.

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* Liebermeister, in 1864, took advantage of an attack of tonsillitis in his own person to demonstrate this fact. His temperature having risen to between 102° and 103°, he exposed his body to cold air, and washed himself with cold water; and he found that this caused a slight rise of the axillary temperature from contraction of the superficial vessels and diminution of loss of heat from the skin, exactly as would have occurred in health. The same observer, in 1868, by noting the change in the temperature of the water of a cold bath, in which he placed a man suffering from acute pneumonia, was able to calculate that the immersion caused a greatly increased proportion of heat, far more heat being given off than would have corresponded with the mere lowering of the temperature of the patient. The accuracy of this calculation has since been disputed by Winternitz ("Der Einfluss von Wärmeentziehungen auf die Wärmeproduction," 'Med. Jahrb.' Wien, [illegible], and 'Virch. Arch.' Bd. lvi. S. 181).

The heat of fever, like that of warm-blooded animals in health, depends ultimately on oxidation of the combustible elements of food (and other subordinate chemical processes). This oxidation takes place chiefly in the muscles, and next in the liver and secreting glands, but to some extent in all living tissues. The dissipation of this heat takes place only to a small extent by conduction and radiation, chiefly by evaporation, and this partly from the lungs, but chiefly from the surface of the body. The regulation of thermolysis, or loss of heat, is accomplished partly by the nervous mechanism of respiration, but chiefly by the vaso-motor and vasodilator nerves diminishing the amount of blood which circulates in the liver and hot internal organs on the one hand, and increasing the flow through the comparatively cool integuments on the other. In many cases of fever, as above stated, more not less heat than in health is given off; but the essential point is that more heat is produced than in a healthy person, lying as still and taking as little food as a febrile patient does. The regulation of both thermogenic and thermolytic processes is thrown out of gear. The thermogenesis is increased beyond the power of the thermolysis to cope with. The nervous mechanism of the latter process is known: that of the former is more obscure, but there is reason to believe that both catabolic and anabolic nerves exist, to increase and to check the thermogenesis of the muscles.*

The next step was to attempt to ascertain the nervous centres in the brain or cord which "preside over," or reflexly control, or "inhibit" the production and loss of heat. The localisation of such thermogenic, thermolytic and (possibly distinct) thermotaxic centres was first attempted by Dr H. C. Wood, of Philadelphia (1880), who found pyrexia (or a "hyperthermic state," as it has been called to distinguish it from idiopathic "fever") to follow injury to the cerebral cortex near the cranial sulcus in the dog. These observations have been since confirmed and extended by Zuntz and other experimental pathologists in Germany and France, and by Dr Hale White in this country. The pyrexia caused by injury to the spinal cord in man, observed many years ago by Sir Benjamin Brodie, and since by Mr Hutchinson and other surgeons, is thus explained: the inhibitory thermotaxic influence of the corpora striata and other parts of the cerebrum is cut off. This experimental or traumatic pyrexia is unaccompanied by rigor, does not lead to vascular changes, and is only temporary in its course. Nevertheless we have every reason to believe that toxic or "idiopathic" pyrexia also depends on derangement of the thermotaxic mechanism, so that thermotaxis overbalances thermolysis.†

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* This part of the theory of fever was ably discussed by Dr Donald MacAlister in the Gulstonian Lectures for 1887.

† Dr Hale White published in the 'Guy's Hosp. Reports' for 1884 (vol. xliv, p. 49) an

The next question is, on what does this nervous disorder depend apart from direct injury or disease of the centres in the cortex, striated body, or mesencephalon? The answer is that it depends on poisoning (intoxication, as the French pathologists call it) with the chemical products of infective microbes, as described in the chapter on Infection. Fever has again and again been produced by introduction of pyogenic streptococci, of putrid liquids free from microbes, of ptomaines, of co-called "tuberculin," and of albumoses. Whether ultimately there is only one pyretogenic compound, or whether many have the power—as many micrococci can produce suppuration, and many poisons can cause myosis—remains to be discovered. At present it may be said that the state of fever is in its essence a disturbance of the nervous mechanism which regulates the production and dissipation of heat, and that this disturbance is due to the operation of chemical poisons.

Thermometry.—It was not until the second half of the present century that the clinical thermometer was introduced. About a hundred years before, the thermometer had been used by Boerhaave and by his pupils Van Swieten and De Haen. The latter, a physician in Vienna, discovered the striking facts that in ague, during the cold stage, the temperature of the blood is increased, and that the temperature of the body may sometimes rise after death. Hunter made many thermometric observations, and Dr John Davy published extensive tables of temperatures in animals and in man, under varied conditions of age and climate, in health and in disease. But when, in 1850-51, Traube and Bärensprung independently called attention to clinical thermometry, the practice was new. Wunderlich devoted infinite pains to the study of temperature in disease. In this country the clinical thermometer was soon adopted by Parkes, Ringer, Aitken, and many others; and its value is now universally acknowledged.

Methods.—The temperature which we want to ascertain is not that of the surface, but of the deeper structures of the body, an inch or more below the skin. To determine the surface temperature of any region is quite another, and a most difficult matter. Instruments called surface thermometers have been constructed, with the receptacle for the mercury of such a shape that one side can lie flat on the skin while the other side is protected with a non-conducting material, so as to diminish as much as possible the loss of heat. But to cover up any part of the surface of the body is, of itself, to raise its temperature for the time being.

When the bulb of a registering thermometer is placed deeply in the axilla, the mercury goes on rising until it indicates a temperature corresponding very closely with that of the body at the same distance from the surface as that at which the instrument is placed; but it is uncertain how long such an observation may take for its completion. Much depends upon whether the parts were closely in contact before the thermometer was introduced. Thus, if the patient is lying on one side in bed, one should always choose the more dependent of the two armpits, since it will give a fixed temperature far more quickly than the other.

It is essential in placing the bulb in the axilla that no clothes should

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interesting essay illustrating the theory of one or more "heat centres" in the brain and cord by a series of clinical cases, some of increased, others of subnormal temperature. See also his Croonian Lectures ('Lancet,' June, 1897) and Riegel's original paper, "Über den Einfluss des Centralnervensystems auf die thierische Wärme" ('Pflüger's Archiv,' 1872). Also Dr Isaac Ott's review in the 'Proceedings of the American Neurological Association' be allowed to remain in contact with it, and that the skin should grasp it firmly. If there be any perspiration, the hollow must first be wiped dry. The arm should then be folded across the chest, and the hand may be made to take hold of the opposite arm, while the opposite hand supports the elbow of the side on which the thermometer is.

It is a very different matter in a thin person, or if the arm has been separated from the chest, so that the axilla has contained air, and perhaps a fold of underclothing. The skin itself must then rise through several degrees before it can bring the thermometer to a stationary point: and the length of time required for this to take place will vary indefinitely, according as the circulation of blood in the peripheral parts of the body is active or otherwise. When the skin is hot and turgid, as in scarlet fever, a comparatively short period will suffice; in the cold stage of ague, and still more during the collapse of cholera, it is doubtful whether an axillary temperature can ever be relied on.

Prof. Bäumler of Freiburg ('Brit. Med. Journ.,' 1864) found by direct experiments that, even when ordinary precautions are taken, it may happen that too low a reading by ·3 to ·8 of a degree Fahrenheit is arrived at if the thermometer is withdrawn from the axilla at the end of five minutes, and that from eleven to twenty-four minutes are required to give an absolutely constant result. Liebermeister laid down the rule that the instrument should be observed to remain stationary for several minutes before it is removed. In practice, however, this is impossible, and in ordinary cases we may with due precautions be satisfied that after five minutes only a small fraction of the total increase of temperature is unregistered, while with some modern instruments the time is three minutes or even less.

When a thermometer has once risen to its full height in the axilla, a second one placed in the same spot immediately afterwards will rise to the same point in from three to five minutes.

In the case of infants and young children the *groin* is better adapted than the armpit, for the parts are fatter, and children who resent the arm being held will often allow the thigh to be kept close to the abdomen without moving.

The *mouth* is a suitable place if one can depend upon the patient keeping it constantly closed and breathing entirely through the nose. The bulb may be placed either beneath the side of the tongue or at the back of the cheek.

Of all localities for thermometric observations, the *rectum* is theoretically the best, and though it cannot be often used in practice, yet it should be chosen when there is doubt as to the accuracy of an axillary or oral observation; and it is particularly suited for children.

By far the most rapid method of taking the temperature of the body accurately is for the patient to hold the bulb of the thermometer in the *stream of urine* when emptying his bladder. The glass and mercury are rapidly heated, for the cooled fluid passes at once away. The method is most applicable for taking the temperature in the evening before going to bed, and also for ascertaining the presence or absence of pyrexia when a patient is going about or visiting his physician. (See a short paper by Dr Oertmann in 'Pflüger's Archiv,' Bd. xvi.)*

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* When, after the withdrawal of the instrument, the index has been read, it is common to dot down the result on a sheet of paper ruled for the purpose. Observations are repeated at regular intervals twice a day, every two hours, or even oftener, and each successive

The course of pyrexia is naturally divisible into certain periods or stages, which, however, vary greatly in length in different diseases. First comes the "pyrogenic" or "initial" stage; during its continuance the temperature rises more or less steadily, the rise being interrupted by the ordinary daily fluctuations. When the rise is at first rapid, there is commonly a shivering fit or *rigor*—the first indication of a severe disturbance of the central nervous system. This is often replaced by a convulsion in the case of children, or by the act of vomiting.

The second stage is the *fastigium*, when the temperature reaches its *acme* or highest point; but not infrequently, since this stage may last for days or weeks, it is characterised (independently of the daily fluctuations) by a series of irregular slight ascents and descents, so that the highest point or one very near it may be touched again and again at intervals.

The third stage is that of *defervescence*, during which the temperature falls again to normal. This is sometimes protracted, sometimes short and sudden. In the former case the pyrexia is said to end by *lysis*, in the latter by *crisis*. When defervescence is rapid, it is frequently accompanied by profuse sweating or by an abundant flow of urine.

After defervescence the temperature often remains for some days slightly subnormal. It is also less stable than in health, being easily disturbed by slight causes; for example, a rise of one or two degrees is apt to follow the first solid meal.

The following is the pathological explanation which Dr Fagge gave of these clinical facts concerning the origin, continuance, and subsidence of the febrile state.

In the *initial stage* of fever the loss of heat from the cutaneous surface is greatly diminished; and as there is no reason to suppose that the evaporation from the lungs undergoes a proportionate increase, it is probable the total loss of heat is less than under ordinary circumstances. As De Haen long ago discovered, the temperature of the deeper parts of the body during a rigor is actually higher than normal, and in fact rises rapidly. Nor is the feeling of coldness of the limbs, and of the skin generally, merely a subjective sensation produced by the state of the peripheral nerves. A man in the cold stage of ague is under no illusion when he complains of being cold, and a surface thermometer confirms his feelings by the slowness with which it rises above the temperature of the air, and the comparatively low point which it reaches. Thus Schülein, among certain observations of surface temperatures (published in Virchow's 'Archiv' for 1876), gives a case of tertian ague; and in his chart it is most striking to notice, how, during each attack, at the precise moment when the

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reading is recorded in a similar manner. Lines are then often traced from dot to dot so as to form what is termed a "chart." It is, however, important to remember that the result is artificial. However short may be the intervals at which the thermometer is applied, there is no reason for supposing that the patient's temperature moves straight upwards or straight downwards from one point to another. And when the intervals are long, as when the instrument is employed only twice in the twenty-four hours, there is not even a probability that the dots marked on the chart each day represent respectively the true maximum and the true minimum. No doubt in disease as in health there are, as a rule, certain daily fluctuations, the temperature being generally highest at a particular hour in the evening and lowest in the morning. But even when the observations are made at those times, it may be that the rule is not observed in that special case; and it is always probable that between each pair of observations there may have been two or three or even several ascents and descents of the temperature which escape notice. Some therefore prefer to make no chart at all, but to place the figures in two vertical lines, one for the morning and the other temperature in the axilla was rising from 98° to 104° or 105° F., that between the toes fell still more sharply, from 95° to 86°, or even below 77°. The cause of the chilliness experienced by such a patient is that the peripheral arteries are contracted and do not allow of the passage of a sufficient quantity of blood to compensate for the loss which is always going on by radiation and conduction. A necessary consequence is a great fall in the temperature of the skin, and this in turn involves a lowering of the amount of heat-loss, and so helps to cause the rapid rise of temperature during rigor.

During the *fastigium* the temperature of the internal parts may remain stationary, or undergoes slight oscillations; but while the axillary or the rectal temperature is high the patient's skin sometimes may feel cool. On the other hand, there are certain diseases in which the hand upon the patient's skin feels it to be pungent and burning. Addison used to teach that in acute pneumonia the skin had a pungent heat which was not observed in any other disease except perhaps scarlet fever. This fact—that in acute pneumonia the temperature of the surface is maintained at a point nearly as high as that of the deeper parts, instead of being far below it—was since established by Schülein, as the result of a series of careful observations on the relation between surface and internal temperature in febrile diseases. His method was to insert a thermometer with a very small bulb between the first two toes; and he found that whereas in enteric fever, acute rheumatism, and phthisis the instrument in this position always indicated a much lower temperature than one placed in the axilla, there were three diseases in which the difference was very slight indeed, namely, acute pneumonia, measles, and scarlet fever. Another disease may be added to those recognised by Addison and by Schülein, namely, acute rheumatism when complicated by hyperpyrexia.

It is evident that in order to maintain such a uniform high temperature of the superficial as well as of the deeper parts, in spite of the great loss of heat from the surface, the generation of heat must be far greater in pneumonia than in cases in which only the interior of the body is raised to a similar degree of heat. So, also, when the skin perspires freely during the *fastigium*, while the rectal temperature remains high, the production of heat must no doubt be proportionately increased.

During the stage of *defervescence* the profuse sweating which so often accompanies it, when a crisis takes place, probably helps in bringing about the rapid fall of internal temperature. But the true cause of defervescence is that the nervous centres which regulate heat are no longer disturbed.—C. H. F.

Daily fluctuation in fever.—A striking proof that the physiological process of heat-regulation is still in action during the course of pyrexia is afforded by the persistence of the regular daily fluctuations of temperature. In pyrexia, as in health, the rule is for the temperature to rise more or less constantly during the day, and to fall during the night. The minimum occurs at about 6 or 7 a.m., the maximum at about 6 p.m. The range of fluctuation appears to be generally as much as 1° C., or even a little more, so that it approaches 2° Fahr.

Degrees of pyrexia.—It is often convenient to be able to express in general terms the degree of pyrexia without giving the actual temperature; and for this purpose Wunderlich proposed the following scheme.

1. *Subfebrile*, temperature in axilla 99·5°—100° Fahr.; or 37·5°—38° C.

2. *Slightly febrile*, temp. 100·4°—101·3°; or 38°—38·5° C.

3. *Moderately febrile*, temp. 101·3°—102·2° in morning, 101·3°—103·1° in evening; or 38·5°—39° C. in morning, 38·5°—39·5° C. in evening.

4. *Decidedly febrile*, temp. about 103·1° in morning, about 104·9° in evening; or 39·5° C. in morning, 40·5° C. in evening.

5. *Hyperpyretic*, temp. approaching 107·6° or even higher (42° C.).

A less elaborate but possibly as useful a nomenclature is—Feverish, 99·5°—101° Fahr. Febrile, 101°—104°. High fever, 104°—106°. Hyperpyrexia above this. At what point the term hyperpyrexia should be used is somewhat uncertain; but it is generally understood to mean such a temperature as is sufficient of itself, if continued, to endanger life.

We must bear in mind that in the case of children fever is set up very easily; an evening temperature of 104° may be due to comparatively trifling causes; and on the following morning the thermometer may not rise far above the normal point. So, again, sensitive women sometimes show a "highly febrile" condition, when the result proves that no apparently adequate cause for it has been present. On the other hand, in old people the temperature is apt to be below what one would expect from the gravity of the case. So that high temperature in children is often of no ill omen, while even slight pyrexia is serious in an elderly patient.

For the thermometer to rise to 109° or 110° (43·5° C.) is very exceptional. Just before the death of a patient from tetanus Wunderlich obtained a temperature of 112·55° (44·75° C.); and this perhaps remains the highest recorded point that has certainly been reached.

Paradoxical temperatures.—A few instances have, however, been recorded in this country which, if they can be relied on, seem to show that far higher temperatures have occurred, not only without being followed by death, but sometimes without being attended with serious symptoms.

Cases.—The first case of this kind was observed by Mr J. W. Teale ('Clin. Soc. Trans.,' 1875) in the person of a young lady, who by a severe accident had several ribs broken, and afterwards suffered from great tenderness over the dorsal vertebræ. Two months later her temperature was one day taken at 110°; and afterwards the index of the thermometer was on four occasions buried in the bulb at the top of the instrument, above 122°. Sometimes these extraordinary temperatures were taken in the axillæ, sometimes between the thighs, or even in the rectum. She was at first in an exceedingly weak state, but she gradually improved and regained fair health.

Other examples of "paradoxical temperatures," as they have been called, were recorded by Dr Donkin ('Brit. Med. Journ.,' 1879). His first case was that of a nurse who was recovering from enteric fever, when the thermometer was found one night to register 110°. Afterwards very high temperatures were repeatedly taken, on a single occasion one of 111·6°, yet no symptoms could be discovered accompanying this reading beyond a feeling described by the patient as one of "flushing" or "rushes of heat." But perhaps the most singular circumstance of all was the evanescent character of this pyrexia, if it deserves that name; once the thermometer rose to 107·2° in the right axilla, whereas five minutes later it stood at 98·6°. In the mouth a temperature of 106° was once observed. It does not appear that the instrument was ever held *in situ* while such extraordinary results were being obtained, but the patient's hands were watched, and the idea of imposture was present. Seven other cases were cited by Dr Donkin, all but one of them being in females.

In 1879 a remarkable instance of this kind occurred in Guy's Hospital under Dr Moxon. The patient, a girl of 22, had been in the ward for phthisis during ten months, when on the evening of July 25th her temperature was taken at 107·4°, and about an hour afterwards at 110·8°. She appeared to be suffering somewhat from dyspnœa. On the following

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gant variations of temperature were recorded. On one occasion Dr Mahomed obtained simultaneously a reading of 102° in one axilla, of about 114° in the other axilla, and of 107° in the month. On changing over the instruments the highest temperature was obtained in the axilla, where it had before been lowest, that of the mouth being now 104°. Another day a small registering thermometer gave 102·6° in one axilla, while another one in the other axilla gave 109·4°; but directly afterwards, when two large instruments without indices were used, and when the patient's arms were held all the time, the temperature stood at 103° on each side. Dr Mahomed noted that the skin always felt moist and of the ordinary temperature, even when a very high reading of the thermometer had just before been obtained. He never got a high temperature with a non-registering thermometer, when he himself held the instrument in the axilla, keeping his hand pressed against the patient's arm.

It is certainly difficult to avoid the conclusion that, in this case, some deception was practised, although its nature was never discovered. Can a patient, without being observed, rub the bulb of a thermometer, so as to drive the mercury up, or actually squeeze the glass with the same result?

In these cases of abnormally high temperatures the pulse and respiration did not rise in any like proportion, there was no delirium or febrile condition of the urine, and the patient did not die.

Pathological varieties of pyrexia.—Fever used to be distinguished as "symptomatic" when it is secondary to a local inflammation, and "idiopathic" or "essential" when it depends on the entrance of a contagion into the blood. It has been experimentally shown that the division of the nerves of the limb of an animal is without effect in preventing the development of fever as the result of local inflammation. The inference—that inflammatory fever is caused by the entrance of a morbid agent into the blood from the inflamed tissues—entirely accords with the early observations of Billroth and Otto Weber, who in 1864, and Sanderson in 1875, showed that pyrexia could be produced by injecting into the blood either fresh pus or decomposing products. It may be stated generally that the inflammatory fevers are caused by pyogenic micrococci, the essential fevers by specific microphytes.

We have, however, examples in human subjects of pyrexia which is not toxic, but directly nervous in origin. Thus cerebral hæmorrhage or the status epilepticus causes symptomatic pyrexia without inflammation. Again, there may be an absence of pyrexia notwithstanding intense inflammation (especially of the meninges and of the peritoneum), when we must suppose that some stimulus acts upon the thermotaxic centres by which they hold the thermogenesis of the body in check.

The irregular fever of idiopathic anæmia, of leuchæmia, and of Hodgkin's disease has not yet received a satisfactory explanation. That which accompanies the various forms of acute tuberculous affections probably depends upon rapid multiplication of the specific bacilli, or in some cases on extensive concomitant inflammation.

There are certain rare cases of pyrexia which agree in having no local inflammation as a cause, and in not being specific and contagious, which run an irregular course and are not accompanied by a corresponding disturbance of the pulse, appetite, and other functions. These cases are more common in women than in men, and in young adults than in others; but differ from the paradoxical cases above mentioned, in the pyrexia being moderate and continuous. The writer has had more than one case of this kind which ended favourably, without their true origin being discovered.

In tetanus the temperature is sometimes exceedingly high without other febrile symptoms. The centre affected by the toxine of tetanus is probably spinal only, not cerebral.

Cohnheim excluded the phenomena of sunstroke from pyrexia. Here the body is directly heated, as by a fire, beyond the power of thermolysis to keep in check, and perhaps the thermolytic mechanism is, so to speak, strained and out of gear. The same view may perhaps be taken of hyperpyrexia in general, because the temperature neither remains stationary at a very high point, nor continues to oscillate backwards and forwards, but either goes on rising until death occurs, or falls again to a moderate level.

Clinical varieties of fever.—We may provisionally arrange the conditions under which we meet with raised temperature in practice somewhat as follows:

1. Irritative inflammatory fever, due to poisoning of the regulating nervous centres by soluble toxines derived from an inflamed part. The pyrexia is moderate when the skin or mucous membranes are affected; moderate or sometimes absent in idiopathic or "simple" inflammation of the serous membranes, and even in acute inflammations of certain viscera, as the brain, the liver, and the kidneys; but always present and usually high in inflammation of the lungs and in acute tonsillitis. Pyrexia is usually absent in chronic inflammations, and cannot be produced by the most severe pain of neuralgia or angina pectoris, although it may apparently be increased by pain, as in cases of acute synovitis, of iritis, of syphilitic periostitis, and of orchitis or inflammation of the ovary.

2. Suppurative or septic inflammatory fever. The temperature is much higher and more constant than when the inflammation is non-purulent. It is most marked when pus is closely confined and subsides on its liberation; but is rarely absent with purulent inflammation, whether of the connective tissues or bones, the brain, liver, and kidneys, or the serous and synovial membranes. It may be regarded as partly due to the same causes as the "irritative" form, partly to absorption of septic micrococci.

3. Pyæmic fever, where there is superadded to the pyrexia of suppurative inflammation that due to intense septicæmia with embolism.

4. Specific primary or idiopathic fever, produced by the swarms of special microphytes (or their secretions) affecting the heat-regulating centres. With these cases we must range by analogy fevers like typhus and measles, in which no microphytes have been certainly discovered, and also, perhaps, rheumatic fever.

5. The irregular fever of the most severe forms of anæmia—Addison's idiopathic or "pernicious" variety, Virchow's leuchæmic or splenic, and Hodgkin's lymphatic Anæmia. Pyrexia is strikingly absent in cases of secondary anæmia and of chlorosis.

6. Those febrile conditions which appear to be primarily nervous in pathology, including *tetanus*, *heat-stroke*, any genuine cases of *paradoxical temperature*, and, lastly, those cases of continued but not excessive pyrexia above described which may provisionally be grouped together as *febris nervosa*.

Concomitants of pyrexia: tissue-change and wasting.—The sources of febrile heat are, as above stated, identical with those which maintain the normal temperature of the body. The elevation of temperature in pyrexia does not arise from an increased tissue-change, as Virchow in 1854 supposed.

urea excreted in the urine during fever surpasses by at least 70 per cent. that which is voided by a healthy person living on the same diet, and careful observations by Ringer in a case of ague established the fact of the same excess of excretion of urea for intermittent pyrexia. But the production of heat may depend upon the increased oxidation of carbo-hydrates which have never formed part of the tissues. Both Leyden and Liebermeister have proved that the excretion of carbonic acid gas is excessive in fever, the increase amounting probably to 50 per cent. But neither the quantity of urea, nor that of carbonic acid, is so great as may be discharged without any rise of temperature after taking active exercise, or after eating a large meal of animal food with fat.

There is, however, no more striking feature of pyrexia than the wasting of the body which accompanies it. This is quite independent of loss of blood or albumen or pus. A patient of Liebermeister's, who had an abscess discharging about three ounces of pus daily, lost from fifteen to twenty pounds in weight during two months, while his temperature was at 101° or 102°, but regained it afterwards when he had no fever, notwithstanding continued suppuration. No doubt inability to digest food helps in bringing about the wasting; it is well known that the secretion of milk ceases during pyrexia, and it is therefore probable that there is a similar lack of saliva (to which, in part, the dryness of the mouth is due); the gastric and pancreatic secretions are also diminished in fever. That it is not merely a question of loss of appetite seems to be shown from an observation of Niemeyer's, who, comparing together a healthy man and one suffering from fever during a period of two days in which they lived on exactly the same food, found that the latter lost much more weight than the former. According to a calculation made by Leyden, the daily loss of substance in fever amounts on an average to 7 parts per 1000 of the whole body-weight. On this Cohnheim remarked that, if this be correct, eight weeks of severe fever should suffice to kill a man by the mere destruction of his tissues; at least, if one may appeal to the observations of Chossat, which showed that to the higher animals a loss of ·40 per cent. of their weight is directly fatal.

Increased rapidity of pulse.—Before the introduction of the clinical thermometer, acceleration of the pulse, rapidity of breathing, diminution of the secretions, thirst, and loss of appetite were the characteristic symptoms of fever. Probably these all depend on the increased temperature of the patient. Liebermeister laid down as the rule that for each degree centigrade above the normal temperature there should be a rise of the pulse by eight beats above eighty. But no doubt various other circumstances may affect the pulse-rate of febrile as of healthy persons. In basilar meningitis the pulse is often infrequent in spite of high fever, possibly owing to stimulation of the vagi. Occasionally a severe case of peritonitis, diphtheria, or meningitis runs its course without elevation of temperature, and then the pulse is often, though not always, quickened.

It is important to observe that the rapidity with which the blood flows in pyrexia, and the arterial *blood-tension*, are by no means proportioned to the acceleration of the heart's action. Early in a fever, when the pulse feels full and hard, there is no doubt obstruction in the arterioles or capillaries. These were the cases which used to be called "*sthenic*" fever. In the 'Med. Times and Gazette' for 1873 the late Dr Mahomed gave sphygmographic tracings of the pulse in fever, showing that in certain cases the vessels are full and the arterial tension is high. But when pyrexia has lasted a few days, and in many instances from the very first, it is of an "*asthenic*" type. The circulation is more sluggish than natural, and the blood stagnates in the more dependent parts of the body, so that hypostatic congestion occurs in the lower lobes of the lungs, and beneath the integument of the back and buttocks, as the patient lies in bed. The pulse still remains frequent, but it is now soft, feeble, and often small. The sphygmograph shows a condition of arterial relaxation, with low pressure and marked dicrotism. At this stage of fever the heart, although its beats are so frequent, empties itself incompletely. Thus the velocity of the blood stream and the arterial pressure are diminished, and thrombi form in the heart's chambers.

The cardiac muscle, like all the other tissues, is ill-nourished as pyrexia goes on. But besides its wasted condition there are definite morbid changes in its texture, due to the fibres being affected with the same "parenchymatous degeneration," or "cloudy swelling," which is found in the liver, in the kidneys, in the heart, and in the voluntary muscles. It is the direct result of elevation of the body-heat, reaching a certain degree of intensity, and protracted over a somewhat long period of time.

Other febrile symptoms.—The increased frequency of the *breathing* in pyrexia is also probably due to the action of heated blood upon the respiratory centre; and this view is supported by Goldstein's experiment of warming the carotid blood alone; he found that the respiration was thereby accelerated. Cohnheim adduced, in illustration of this theory, the fact that in acute pneumonia the breathing ceases to be rapid after the crisis of the fever, notwithstanding that the affected part of the lung still remains hepatised.

It is generally believed that the *cerebral symptoms* of fever also depend on the increased temperature of the blood. This probably explains the marked effect of a cold bath in restoring the mental faculties of a patient suffering from fever, though this may be partly due to the improvement of circulation.

Obscure in some points as is the connection between pyrexia and these disturbances of other functions, there is no doubt of their practical importance at the bedside; and it may be well briefly to enumerate them before leaving the subject.

In addition, then, to the raised temperature which is the constant and governing phenomenon of fever, we observe to a greater or less extent, according to the severity and duration of the pyrexial state—(1) acceleration of the pulse and respiration; (2) thirst; (3) disturbance of secretion, and probably as results, (4) loss of appetite, a dry and furred state of the tongue and constipation; (5) scanty and high-coloured urine, rich in urea, often albuminous, and depositing lithates; (6) pains, particularly headache, not specially frontal, vertical or occipital, but central and deep-seated; and also muscular pains in the loins, back, and limbs; (7) delirium; (8) emaciation.

The *treatment* of pyrexia by baths and antipyretic remedies will be most usefully discussed in the sections on enteric fever and acute rheumatism, which offer the most frequent and urgent occasions for their use.

Subnormal temperature.—It is an interesting question whether the temperature of the body ever undergoes a continuous change in the directhe normal instead of above it. There are many instances in which a thermometer placed in the axilla remains low, but probably in these cases the internal organs maintain a temperature nearly as high as ever, at least until death is close at hand. Cohnheim says that this is the case with those who suffer from inanition as the result of stricture of the œsophagus, of starvation from other causes, or of extreme anæmia. Lowering of temperature, due to inanition and cold (by "*force majeure*," as Liebermeister called it), is well seen in persons who are picked up insensible in the streets during cold weather, when the thermometer in the rectum may be only 86° or even 79° F. Their pupils are dilated and sluggish, while the pulse and the respiration are greatly reduced in frequency; but frequently such patients under suitable treatment regain their normal temperature within a few hours, and sometimes pass into a condition of reactive pyrexia.

The two conditions, in which (apart from inanition and from the reaction after pyrexia) the temperature is most frequently found subnormal, are diabetes, and the stage of collapse in cholera. It is also habitually subnormal in the curious affection described by Sir William Gull as a cretinoid condition in adults, and since named myxœdema. The lowest temperatures, however, have been observed in certain cases of injury to the cord: in one recorded by Mr Hutchinson the temperature in the rectum was not above 80·6° F.

INFLAMMATION

Notæ vero inflammationis sunt quatuor: rubor et tumor, cum calore et dolore.
CELSUS.

Introductory—Successive theories of Inflammation—Observation of reaction to irritants in the frog, in mammals, in non-vascular tissues—The significance of the redness, swelling, pain, and heat—of the pyrexia—of the exudation and particularly of the migration of leucocytes—Chemiotaxis and Phagocytosis—Clinical varieties of Inflammation—catarrhal—serous—suppurative—plastic—membranous—gangrenous—Local varieties: inflammation of skin, mucous and serous membranes, connective tissues, cartilage and bone, muscles and nerves—viscera—Events of inflammation—Its ætiology and uses—Chronic fibrous inflammation—Granuloma—Possibility of retaining the term inflammation.

WHEN an injury is inflicted on the skin or a visible mucous membrane, there is, after the immediate pain has moderated or disappeared, an interval, followed by renewed *pain* of a throbbing character, *i. e.* increased with each beat of the heart.

At the same time the injured skin or conjunctiva becomes *red*, it *swells* both visibly and palpably, it feels *hotter* than the surrounding parts, and if the surface affected is extensive, *fever* supervenes, *i. e.* there is thirst, anorexia, and the other symptoms described in the last chapter, together with rise in the temperature of the body.

The term Inflammation was applied to the whole process; and the word was only gradually extended to presumed similar affections of bones and other accessible parts, such as the tongue, the lymph-glands, or the testicle. Finally it was assumed as the cause of most diseases of which the local seat was discovered or assumed. The presence of the "four signs" was no longer insisted on, and the fact of fever was regarded as evidence enough of internal inflammation. The term was then applied to such conditions as Bronchitis or Cystitis, Peritonitis, and even Phrenitis. Suppuration, Phagedæna, and Gangrene were included as forms or results of inflammation; and lastly, the phrase was made wide enough to include the processes now known as chronic inflammation, such as cirrhosis of the liver, chronic contracting pneumonia, interstitial nephritis, and grey degeneration of the cord. We shall afterwards discuss how far this extension of the term from its primary significance is justifiable or expedient.

Past theories of inflammation.—One of the earliest hypotheses as to visible, traumatic inflammation was that its essence consisted in the redness, which was assumed to be due to a primary dilatation of the blood-vesse [illegible] rapidity of flow and increased heat of the blood. We now know that the calibre of the arteries is regulated by vaso-motor nerves derived from the spinal cord, and that mere dilatation of the blood-vessels (experimentally produced, for instance, by division of the cervical sympathetics in the rabbit's ear) does not produce inflammation even after weeks and months.*

It has also been settled, after long discussion, which dated from the first observation of Hunter, that the increased temperature of an inflamed part is not due to any local production of heat, but to the greater amount of blood admitted from the internal organs; the *calor*, in other words, is the mere consequence of the *rubor*. As soon as local inflammation sets up fever, the blood rises from 98·4° to 100° or 104°, and this, of course, makes the inflamed parts so much the hotter.

The next theory, that of exudation, may be regarded as resting on the third Galenic symptom, *tumor*; for the swelling of an inflamed part, though at first due to increased fulness of the blood-vessels, is chiefly caused by the speedy exudation from these vessels of *plasma*—*liquor sanguinis*, or "inflammatory lymph"—consisting of water, salts, pigment, and proteids in solution (serum), together with fibrinogen and leucocytes (lymph-corpuscles, white blood-corpuscles, exudation cells). This "inflammatory œdema" becomes, if the corpuscles are so numerous as to make the exudation opaque, "suppurative;" or, if coagulation is active and the resulting fibrin abundant, "plastic inflammation," the clot being still often called "coagulated lymph," and, when it forms a layer on a surface, "false membrane." We shall see that the most constant and therefore the most essential event is the passage of these cells from the vessels into the tissues.

Present condition of the question.—Accepting Burdon Sanderson's definition of the process of inflammation as "the succession of changes which occur in a living tissue when it is injured, provided that the injury is not of such degree as at once to destroy its structure and vitality," we may say that this reaction of an organism, still living though injured, consists essentially in the presence of leucocytes in the irritated or inflamed part. The dilatation of the blood-vessels is secondary to this in vascular parts, and may even be absent, as proved by the study of the effects of injury on non-vascular tissues like the cornea and articular cartilages. This was the great contribution made to the subject by Virchow in Germany, and by Goodsir and Redfern in this country between 1850 and 1860. The same conclusion was arrived at by the observation of the effects of irritation on the embryo of Echinoderms by Metschnikoff.

The heat is the result of increased afflux of hot blood, and the pain is the result of pressure on the nerves of the part by this afflux, which causes the characteristic inflammatory throbbing, and by the exudation of corpuscles and "lymph" into the tissues.

It was formerly supposed that the inflammatory corpuscles formed *de novo* in the exuded lymph; and, when this spontaneous formation of living things was disproved, Virchow taught that these corpuscles were the offspring of the existing living cells of connective tissue. That such "proliferation" does take place is certain, but it is a process belonging rather to new growths and to the more chronic forms of inflammation and repair than to the most acute and characteristic form of inflammation, that of suppuration.

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* The only effect of this "active congestion" is to increase the liability to inflame when the ear is injured, and to increase the œdema when it is inflamed ('Journal of Physiology,' vol. viii, p. 25).

In acute suppuration Cohnheim, following up earlier observations of William Addison and Waller (1846), conclusively showed that pus-corpuscles are nothing but leucocytes which have wandered out of the capillaries; probably through the minute stomata between the endothelial plates of the intima. By what force this is accomplished—whether by their own amœboid movements, whether by increased pressure forcing them through, or whether owing to some precedent damage to the vascular wall—is still unsettled. The first seems the most probable hypothesis, for when intra-vascular pressure is much increased, or when the walls of the minute blood-vessels are diseased, the result is extravasation of red blood-discs, without the characteristic events of inflammation.

Observation of inflamed tissues in the frog.—Coming now to the facts actually observed under the microscope, we may state the phenomena which follow irritation of transparent vascular membranes in cold-blooded animals as follows:—When the web of a frog's foot, the mesentery of a toad, the tail of a tadpole, or the thinnest parts of a small fish's tail are touched by nitrate of silver, croton oil, or other chemical or mechanical irritant, or even if left exposed to the drying of the air, in from fifteen to twenty minutes the arteries are observed slowly to dilate, until at the end of an hour or two their diameter is nearly twice as great as before. A little later a similar change begins in the veins. At first the blood-stream perhaps flows more quickly than before, but very soon its rapidity begins to diminish, so that the oval red blood-discs can be distinctly seen in the arteries, at least during diastole. A striking change is now noticeable in the veins. Leucocytes begin to fall out of the middle of the current, and loiter against the sides of the vessel, and stick from time to time, until the channel of the veins becomes lined by a thick layer of these bodies, which lie at rest while the central current of red discs goes on as before. In the capillaries, too, the leucocytes are arrested here and there; but between them there are seen many red discs also adhering to the walls. Outside the wall of these vessels there gradually appear a number of minute rounded bodies, each of which is connected with a leucocyte within. Thus the leucocytes may be said to have assumed a dumb-bell form. By an imperceptible process, the extra-vascular part keeps growing larger, and the intra-vascular part smaller, until at length the latter altogether disappears; whereupon the leucocyte resumes a more or less globular shape, and now lies close to the vein, but outside it. The result is, that large numbers of leucocytes are soon collected not only in the immediate neighbourhood of the vessels, but in all the interstices of the surrounding tissues. At the same time a quantity of fluid exudes, which reaches the free surface of the mesentery and coagulates there, forming a membranous layer, thickly set with leucocytes.*

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* The important fact of the white corpuscles adhering to the sides of the vessels in the web of the frog's foot had been observed and figured by Dr C. J. B. Williams, in his 'Principles of Medicine' (1843). Mr Wharton Jones had detected the same phenomena and accurately described the dilatation of the vessels and stasis of the blood ('Guy's Hospital Reports,' New Series, vol. vii, 1850), and Mr (now Sir Joseph) Lister had confirmed and extended these observations by his own (also carried out on the web of the frog's foot) in the 'Philosophical Transactions' for 1858. One of the important points established by Lister was that the dilatation of the arteries depends on paralysis of the vaso-motor nerves.

In cold-blooded animals severe irritants, as croton oil, cause a condition known as *stasis*. The circulation is arrested, and the blood coagulates in the capillaries and vesicles of the injured part. In the middle all the injured tissues are killed, and an *eschar* is formed. Next comes an area of stasis. Further outwards the blood-stream is almost stagnant, and the capillaries are choked with red discs, which escape from them in large numbers. Beyond this again the emigration of leucocytes is the principal change. Last comes a zone in which the vessels are merely dilated, while the circulation through them is somewhat retarded.*

Inflammation in mammalia.—In warm-blooded animals the attempt to watch the inflammatory process directly with the microscope was attended with considerable difficulties; but these were gradually overcome. Wharton Jones had observed in the bat's wing that the vessels when inflamed dilated, that the blood-stream slackened, and that the corpuscles crowded together to the sides of the vessel ('Med.-Chir. Trans.,' 1853). Paget, in his 'Lectures on Surgical Pathology' published in the same year, also used the bat's wing, and observed the very short period of initial contraction, the subsequent dilatation, and other phenomena as afterwards described, excepting the emigration of leucocytes. In 1870 Sanderson and Stricker, of Vienna, contrived an admirable method of studying the circulation in the mesentery of the guinea-pig, which was demonstrated at the meeting of the British Association in that year at Liverpool, and this method was afterwards applied to the observation of inflammation. A few years later Thoma published in 'Virchow's Archiv' for 1878 a series of microscopical observations on inflammation in the mesentery or the omentum of dogs, cats, rabbits, and guinea-pigs. Emigration of corpuscles and exudation of coagulable fluid occur in precisely the same way as in frogs.†

The separation of the leucocytes from the red discs flowing through the mid-channel of the veins, and their collecting against the walls of these vessels, are direct physical results of the slowing of the blood-current. The process of *emigration* must be referred to the power of spontaneous locomotion possessed by the leucocytes themselves.

Inflammation in non-vascular tissues.—Before the time of Virchow it was doubted whether parts into which no vessels penetrate could undergo inflammation. He placed the question on its right footing by showing that the distinction between vascular and non-vascular tissues was after all only one of degree, and that a minute islet of liver substance is outside the blood-current, no less than the cornea or the cartilage of a joint. About the same time Goodsir in Edinburgh, and Redfern in Belfast, were demonstrating the possibility of inflammation and ulceration in articular cartilage and other non-vascular tissues. Cohnheim's investigations went a step further; for he proved that leucocytes could penetrate into the interior of the cornea from the blood. It had been shown by Von Recklinghausen that when finely divided vermilion was injected into a lymph-cavity in the frog, the leucocytes would take this substance into their interior, or "feed" upon it; and

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* Ryneck, of Gratz, as long ago as 1870, showed that stasis could be set up by irritation in the web of a frog, after defibrinated mammalian blood, or even milk, had been substituted for the natural circulating fluid of the animal; but that when a solution of chromic acid or sulphate of copper had once been passed through the blood-vessels, even for a few moments, no such effect could be produced. The same is true of a frog in which the blood is replaced by normal salt solution.

† According to Prof. Thoma, in mammals the veins only dilate, not the arteries also as in frogs ('Virchow's Archiv,' 1878).

Cohnheim found that, after injecting pigment into one of the aortæ, many of the leucocytes which appeared in the cornea under inflammation were coloured, so that they had evidently been derived from the circulatory fluid.

The phenomena of inflammation.—We may now proceed to indicate such explanations of the symptoms of inflammation as accord best with our present stage of knowledge.

1. *Rubor.*—The redness of inflammation depends partly upon the dilatation of the veins and other blood-vessels of the affected area, partly upon the accumulation of blood-discs in its capillaries. If the separate vessels are visible, the redness is spoken of as "injection;" if they cannot be seen, it is said to be "diffused." When stasis occurs, this also causes redness; and in the later stages hæmorrhage helps to deepen the tint, which acquires a punctiform character, and no longer fades beneath the pressure of the finger. Free exudation tends to conceal the red colour, even where it does not actually compress and empty the vessels. When non-vascular structures become inflamed, the redness can only show itself in the vascular parts around, from which they derive their nutriment; for instance, in the conjunctiva at the margin of the cornea, and in the synovial membrane about articular cartilages. After death inflammatory redness disappears, or is greatly diminished.

That redness is not essential to Inflammation is proved by its absence in Invertebrates, which nevertheless show the same phenomena of exudation and chemiotaxis as do Vertebrates.

2. *Tumor.*—This may, to some extent, be due to vascular turgescence, but the chief cause of it is exudation. It was long ago experimentally shown that the stream which flows away through the lymphatics of an inflamed part is greatly augmented. The swelling of inflammation corresponds, not with the whole amount of exudation, but with the excess of it, as compared with what is taken up again.

In distributing itself through the inflamed structures, exudation follows the lines of least resistance. In connective tissue, and in the stroma of a parenchymatous organ, it fills the natural interstices. Wherever there is a gap or interspace it accumulates. It covers the free surfaces of serous membranes. Mucous membranes throw it off, to mix with their natural secretions. In the skin it penetrates through the deeper layers of the cuticle, and it often raises the horny layer to form vesicles and pustules.

3. *Dolor.*—The pain of inflammation is usually, and no doubt correctly, attributed to the compression of the sensory nerves by distended vessels and afterwards by exudation. Its throbbing character is due to the pressure being increased by each pulsation of the heart. It is generally accompanied by "tenderness;" or (in other words) it is increased by pressure from without. Some tissues, such as ligaments and bones, may become exceedingly painful when inflamed, although they are not naturally very sensitive. The pain of periostitis and of orchitis and glaucoma appears to be caused by the resistance to distension and swelling which is offered by unyielding fibrous structures.

4. *Calor.*—That those parts which are most accessible to observation become hotter than natural when inflamed, is matter of common knowledge; anyone may satisfy himself of it by placing one hand over an acutely swollen joint or a recently formed abscess, and the other upon the corresponding

spot on the opposite side of the body. But contradictory statements have been made as to the relation between the temperature of an inflamed part and that of the blood and of the deep internal organs.

In 1860 Sir John Simon made a series of observations with a small thermo-electric apparatus, so shaped that it could be thrust like a pin into the soft tissues, or even into the interior of large vessels; and he concluded that the arterial blood flowing towards a severely injured limb in a dog was always less warm than the venous blood flowing away from it. But Jacobson, of Königsberg, who afterwards investigated this question with more accurate apparatus, found (' Virchow's Arch.,' 1870) that the temperature of the skin and of the muscles down to the bone never, even under conditions of the most intense inflammation, reaches that which exists in the rectum, the vagina, or the peritoneal cavity at the same time. The difference was generally as much as 2° or 3·5° Fahr. As for the relation between the temperature of an inflamed part and that of the opposite side of the body, the difference was always less in proportion as the distance from the surface was greater. Thus, whereas a rabbit's ear when inflamed was warmer than the other ear by 7° or 8° Fahr., the temperature of the deep muscles of a limb under the same circumstances seldom exceeded that of the same structures on the opposite side by more than 1°. It would follow that the internal organs under inflammation continue to be of a temperature approximately the same as that of the blood. Jacobson also showed by direct experiment that in pleurisy the affected cavity was not hotter than the other one.

In December, 1879, Peter brought before the Paris Academy of Medicine a series of observations, from which he drew the conclusion that there was a considerable local production of heat during inflammation. But what he really showed was nothing more than that the temperature of the abdominal wall may in such cases exceed that of the axilla by as much as 3·5° Fahr. If Jacobson's observations are correct, the real increase of temperature in the superficial structures when inflamed may receive a simple explanation. For the blood-current is a great warming apparatus, which carries to the tissues all over the body heat that is chiefly generated in the liver and other glands, and in the muscles. If there be no additional local production of heat, the temperature of each part must be the resultant of four varying conditions: (1) the temperature of the arterial blood supplied to it; (2) the proportion between the space occupied by its blood-vessels and that occupied by its extra-vascular material; (3) the rapidity with which its vessels are traversed by the blood; (4) the greater or less extent to which heat is dissipated from it by conduction, by evaporation, or otherwise. Of these conditions the most liable to great fluctuations are the second and third. The vessels of an inflamed part are dilated, and the flow of blood through an inflamed part is much augmented. Sir William Laurence is said to have roughly demonstrated this long ago by bleeding from both arms simultaneously a patient whose hand and forearm on one side were inflamed. But Cohnheim firmly established the same fact by careful experiments. Having set up inflammation in the paw of a dog, he measured the amount of blood which afterwards escaped through a cannula from the principal vein of the limb; and he found that it was sometimes more than twice as much as flowed from a corresponding vein on the opposite side in the same length of time. The only exceptions were, when gangrene set in, and when there was profuse suppuration. Increased velocity in the blood-current of an inflamed part, with dilation of its vessels, must necessarily raise its temperature.*

That the heat of inflammation is not essential to the process is proved by its absence in artificial inflammation in the frog. Even in human pathology we occasionally meet with "cold abscesses."

Moreover, we know by experiment that division of the vaso-motor nerves of a part will cause great dilatation of the arteries, great redness, and decided increase of temperature; and yet there is no inflammation—no pain, no swelling, no exudation.

5. *Pyrexia.*—To the four Galenical signs of inflammation we may add fever as a constant result in injury, inflammation, and repair, and thus a constant companion of pain, heat, redness, swelling, and exudation, which constitute the process of acute inflammation. As we shall see, it is difficult to bring the more chronic forms of inflammation under the same definition as acute suppuration, though it is still more difficult to draw a line between them. But in proportion to the acuteness of the process is the height of the fever, and where no active exudation takes place the fever is absent. Like heat and redness, pyrexia is only observed in warm-blooded animals, and must depend, as we saw in the chapter on Fever, upon disturbance of the thermotaxic centres of the brain and cord (p. 32). We therefore look to some toxic disturbing cause, and this we find in the microbes or their poisonous products, which take part in the process of Infection (p. 22), particularly streptococci, staphylococci, and other pyogenic organisms. Fever is highest when there is most suppuration, lower where there is a low degree of infection, and absent after an aseptic operation, followed by repair of the tissues without any active exudation at all. If this be so, we must regard the pyrexia of inflammation not as its consequent, but as a collateral, phenomenon, complicating the process of repair when the irritation which causes inflammation is accompanied by toxic effects.

6. *Inflammatory exudation.*—We must now turn to the products of the inflammatory condition, to the solids and liquids which exude from the blood. The solid bodies exuded are leucocytes, or white blood-corpuscles. Hæmorrhagic inflammation, in which diapedesis of red blood-discs also takes place, is an exceptional and probably a complex process. But the leucocytes are by no means all alike, even in healthy blood, and in the exudate of inflammation they present important differences, which have been minutely studied during the last few years.†

There may be distinguished the following emigrated white corpuscles or exudative cells:

a. Lymph corpuscles, characterised by their small size, clear protoplasm, and large single nucleus—young cells.

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* No doubt if superficial vessels are involved in the inflammatory process, more heat is at the same time dissipated, and this tends to prevent the surface temperature from approaching that of the internal organs. But a point of considerable importance is that such an increased dissipation of heat actually makes the surface feel hotter to one's hand than it would otherwise feel.

† The pioneer in this as in other questions of the physiology of the blood and circulation was Mr Wharton Jones, who in 1846 distinguished between clear nucleated cells (lymphocytes) and coarsely or finely granular cells. But his observations, like those of Wm. Addison on Emigration, were not followed up in England, and were continued by Rindfleisch and Max Schultze. Recently Ehrlich in Germany, Metschnikoff in France, and many other observers have greatly advanced this part of the subject.

b. A clear hyaline uninuclear cell like the last, but larger—the large non-granular leucocyte, possibly the mature stage of the first variety. Like it, the nucleus stains readily; but, unlike it, this kind of leucocyte is endowed with the power of destroying bacteria. These "phagocytes" are not found in lymph, and are far less common in blood than the preceding variety. They are found in passive serous effusion in greater numbers than in the blood.

The protoplasm of the above clear leucocytes does not readily stain, in contrast to the nucleus. This distinguishes them from those in which the protoplasm is granular and stains readily with aniline dyes.

c. In one group of the granular easily stained corpuscles, the granules are minute and punctate and the nucleus is bi- or tri-lobed. These are common in human blood and in inflammatory exudations. They exhibit active movement on the warm stage, and are phagocytes. The granules are probably proteid in chemical character. In some cells, mostly the larger in size, they stain with acid and alkaline dyes ("amphiphile" or "neutrophile;" in others, mostly smaller, they stain only with alkaline dyes (basophile).

d. The leucocytes with coarse granules (which in part at least are fatty in character) are scarce in human blood, but are found in inflammatory exudations and in pus, and also in lymph spaces. They are large. with a highly refracting nucleus, which comes out boldly when acetic acid is run in under the cover-glass. They appear not to be phagocytic in habit, although they exhibit amœboid movement when warmed. Some of these "coarsely granular" cells stain with acid dyes (eosinophile); others, which are said not to occur at all in human blood, with basic dyes.

Passing to the fluid part of inflammatory exudation, this may contain: (1) water, holding salts and albumen in solution—serous exudations, inflammatory œdema; (2) mucin, constituting the catarrhal or mucous variety; (3) fibrinogen, which in the "coagulable lymph" forms a fibrinous clot—the "plastic" inflammation of the serous cavities, of the air-vesicles, and occasionally of the mucous membranes; (4) corpuscles or leucocytes particularly finely and coarsely granular, mostly eosinophile. As these are more abundant they render the clear or opalescent exudation thick and turbid; when very numerous they make it purulent (*pus laudabile*)—as opaque as milk, and for the same cause, the solid particles interrupting and reflecting the rays of light so as to be dark by transmitted and light by incident illumination. These four varieties may be variously mingled, so as to form sero-purulent (*e. g.* in pleurisy), muco-purulent (*e. g.* in bronchitis), or fibrino-purulent (*e. g.* in pericarditis) exudations. Pure pus is produced by connective tissues, by the skin, by mucous membranes occasionally, by bone, brain, joints, and solid viscera under special forms of irritation—usually infection with micrococci.

Not only may an exudation which begins as a serous or mucous become a pustular one, as, for instance, in cystitis and eczema, but one which at first is fluid may become more solid by the gradual coagulation of the fibrinogen. Fibrin must often be formed very rapidly, for it appears as a thin uniform layer upon the sloping surface of a serous cavity.

Inflammatory exudation used to be spoken of as inflammatory lymph. and, like lymph, it consists of water, with pigment, salts, and albumen in solution, and leucocytes in suspension. But it differs (1) in there being added sometimes mucin, sometimes peptones, and sometimes fibrin-forming proteids; (2) in the kind of living cells, those of lymph being smaller and clear, those of exudation larger, finely or coarsely granular, and for the most part eosinophile; (3) in the proportion of the chemical constituents. Lymph and dropsical fluid is of lower specific gravity (1010—1015), contains less albumen and globulin, and forms scarcely any fibrin; inflammatory exudation is of higher specific gravity (1020—1025), though never so high as blood-serum, with twice as much proteids and produces more or less abundant fibrin.

It is observable that inflammatory exudation from serous membranes is richer in albumen, globulin, and fibrinogen, than that from mucous; and that, with the abundance of leucocytes, peptones come to form a larger proportion of the proteids held in solution. Being comparatively diffusible, they appear first in the urine, to be followed by albumen and globulin when there is not only local suppuration present, but also a condition of general pyrexia.

Theory of inflammatory exudation.—We have seen that the pain of inflammation is secondary to pressure, *i. e.* to swelling, that redness is due to dilatation of the blood-vessels, that heat is caused by increased amount and increased velocity of the blood in the injured part, and that fever is a collateral effect of blood-poisoning.

The swelling, *i. e.* the exudation of plasma and leucocytes from the vessels, remains as the essential phenomenon of reaction after injury, alike in man and mammals, in cold-blooded and in bloodless animals (as Aristotle called the invertebrates, meaning without red blood), so that the next step in the inquiry is what causes exudation? Cohnheim adopted and enforced the hypothesis of Samuel, that the answer is to be found in lesions of the vascular wall which allow the escape of liquids and solids impossible in health. But a sufficient answer to this seems to be that this presumed morbid permeability of the blood-vessels ought to lead to hæmorrhage, whereas with rare exceptions inflammatory exudation is unstained by red blood-discs; and, on the other hand, the capillaries are often so much damaged that they allow of escape of blood, without any of the signs or symptoms of inflammation.

We must therefore look for some cause why the leucocytes leave the blood-vessels for the tissues, or, to speak more scientifically, for some evidence of the immediate conditions under which they exert their amœboid energy in this way. We have seen that they cluster together and arrange themselves at the injured or irritated spot, that they collect outside the vessel when they have traversed it, and are then active in loosing foreign bodies or eschars formed by dead tissues, in circumscribing and limiting destructive changes, and in "englobing" the hostile organisms of bacterial invasion. The former process is known as *chemiotaxis;* the latter is the work of the modified leucocytes, known as *phagocytes* (cf. p. 24). The "explanation." then, is to regard the process of inflammation as on the whole conservative in its results, and to refer its essential phenomenon of exudation, *i. e.* chemiotaxis and phagocytosis, to the energy of living amœbiform protoplasm, which "instinctively"—if the word may be conceded—strives against invasion, and struggles for the preservation of the organism to which it belongs. The process would then be only one case of the great "struggle for existence," to which all living things are subjected.

One advantage of this theory (or way of looking at the facts) is that it includes under "the reaction of an organism to injury" not only traumatic and micrococcal inflammation in mammals, but similar reaction in all classes of animals down to the lowest, and even the processes of repair or reaction to irritation and to poisons which are observed in the vegetable creation—in the healing of wounds in the bark of a tree, and the formation of a gall in response to an invading insect.*

Clinically we may recognise the following chief varieties of inflammation:

Catarrhal inflammation.—When mucous membranes are irritated they throw off an abundance of more or less altered epithelial cells. In the lungs or in the kidneys such "catarrhal products" are often seen in immense numbers; not only do they accumulate in and fill up the pulmonary alveoli or the renal tubes, but a great many of them are also carried away in the sputum or in the urine. With this may be grouped dry inflammation, seen in the skin when similar proliferation of epithelium takes place, but without the flow of liquid to wash them away, as in cases of psoriasis.

Serous inflammation.—When a joint or serous cavity is affected with inflammation of moderate severity, it pours out a transparent fluid which accumulates in its cavity. There may be a thin layer of fibrin upon the free surfaces of the membrane; or shreds and flocculent masses may be floating in the fluid. Sometimes not a trace of fibrin is to be seen; fibrinogen is present, but for some reason remains uncoagulated. Virchow many years ago showed that fluid effusion, after its removal from the pleura by paracentesis, would often throw down a coagulum of fibrin, and that if this were removed, a fresh one might form in the course of the following day, and so on for several days in succession. This kind of inflammation is found not only in closed serous and synovial cavities, but also in the endocardium, in the iris, in the lymph-spaces of areolar tissue, and in the air-vesicles of the lungs, all of them lined with a flat single layer of epithelium and in connection with lymphatics.

Suppuration.—In the more intense inflammations of joints or serous membranes the exudation is purulent, and does not coagulate. Intermediate conditions, however, are frequent, in which the surfaces are coated with fibrin, while the cavity contains liquid pus; in the pericardium, pus unmixed with fibrin is seldom, if ever, seen, and it is rare in the pleura or the pia mater. Occasionally in the pleura or peritoneum it may happen that every part of the surface retains its natural smoothness and lustre, notwithstanding that pus is present in large amount.

The quantity of pus which collects in a large serous cavity, such as the pleura, is often enormous; and since it may be rapidly secreted, one wonders how the blood can yield a sufficient number of leucocytes. But, as Cohnheim remarks, this difficulty applies far less to the emigration theory than to that of pus-formation by proliferation—from the cells of connective tissue, as Virchow taught. While inflammation is going on, the blood throughout the body contains an excess of leucocytes as well as of fibrin.

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* The theory is clearly and attractively set forth in Metschnikoff's lectures on the 'Comparative Pathology of Inflammation,' delivered at the Pasteur Institute in 1891. His observations on Daphnia, Hydrozoa, Axolotl, and other animals have been confirmed by Lieberkühn, Leber, Hafkine, and many others, and correspond to the observations on the behaviour of the lower vegetable organisms by Pfeffer, De Bary, and Mr Arthur Lister.

Probably the lymph-glands and the spleen throw into the circulating fluid a largely increased supply of leucocytes to make up for the drain that is going on. Moreover exudation cells themselves multiply by fission. Dr Thin once figured a dumb-bell shaped leucocyte which seemed to be dividing into two, from the inflamed cornea of a rabbit; and similar appearances have been observed in cold-blooded animals by Stricker, Klein, and Ranvier.

In the great majority of cases of suppurative inflammation, micrococci of some kind are present (strepto-, staphylo-, or diplococci—cf. *supra*, p. 19), and this applies not only to pure suppuration, but to cases where it complicates other forms of infective inflammation, as diphtheria and tubercle.

Membranous exudation.—This remarkable variety was formerly called "plastic," afterwards by an unfortunate confusion of clinical and pathological terms, "croupous," and by a still more mischievous confusion of a specific disease with a common morbid process, "diphtheritic." Why it should be so rare in mucous membranes, in solid organs, and in connective tissue, and so common in serous and other endothelial surfaces, is hard to say. The exceptions, however, are not few, nor are they limited to the case of specific inflammation like that of diphtheria. In all likelihood a plastic inflammatory exudation is but one case of fibrinogen becoming fibrin in the plasma, and will only be understood when physiologists have solved the hitherto insoluble problem of the coagulation of the blood.

Membranous necrosis.—The process which combines fibrinous exudation with loss of vitality in the affected tissues is what has been called "diphtheritic." The term is an unfortunate one, since it implies a connection with the disease Diphtheria, by no means generally the case.

A free surface, usually a mucous membrane, looks dry, and of a slate-grey or pale yellowish colour. It feels rough, granular, and tough. In the dead body, if an incision is made, the dry grey appearance is found to penetrate to some depth, and to cease somewhat abruptly. Under the microscope is to be seen a more or less distinctly fibrillated tissue, containing in its meshes altered epithelial cells and leucocytes. Glistening fibres which resolve themselves into chains of micrococci pervade the tissue. The only way in which the disease can end is by the separation and shedding of all the dead tissues. This detachment is effected by *ulceration*. From the first there was an inflammatory action of more or less intensity in the living structures beneath. But now leucocytes collect in large numbers along the boundary line, and probably the connecting material is eaten away and absorbed by phagocytes.

The bladder is very liable to necrosis, involving the whole thickness of its mucous coat; and it is not uncommon for the *mucosa* to be cast off almost entire. An autopsy was once made by Dr Fagge in the case of a woman from whose bladder during life a piece of considerable size had been removed. All the rest of the vesical mucous membrane lay loose, as a thick ash-grey substance, with markings on one surface corresponding with those of the hypertrophied muscular trabeculæ.

The other parts most liable to this membranous necrosis are the mucous membranes of the mouth, gums, and cheeks, of the fauces and soft palate;

and sometimes in addition the specific microbes of an infectious disease. Besides isolated cases of this form of inflammation, which are often difficult to assign to their proper pathological places, the most important diseases in which it is met with are diphtheria and dysentery.

Gangrenous inflammation.—Gangrene, sphacelus, or mortification of a part of a living body, has been recognised from an early period; but only in recent times has the part inflammation plays in its production been ascertained. Living tissues may be directly killed in various ways without inflammation; as, for instance, by escharotics, or by a high degree of either heat or cold. When the death of a part is due to a failure of its supply of blood, it does not always undergo inflammation first; but surgeons are now familiar with the fact that in many instances senile gangrene of the foot, which used to be attributed merely to obstruction of the arteries, arises from slight injury to a toe, and begins as an inflammatory process. Even when a limb undergoes mortification as the direct result of embolism of its main artery, there is always active inflammation at the margin of the gangrenous part. Indeed, Cohnheim showed that the effects of a temporary ligature applied round the ear of a rabbit depend entirely upon the duration of the constriction. After a certain length of time the blood fails to penetrate into the vessels when the ligature is removed, and the part dies; but if the ligature is removed at earlier periods, the result is only an intense inflammatory swelling of the ear with hæmorrhage. In those forms of gangrene which seem to be determined by the occurrence of pressure, there are always further conditions: a previous enfeeblement of the circulation, or some slight local injury, or some affection of the trophic nerves. Thus bedsores occur chiefly in those cases in which there is hypostatic congestion of every dependent part, and the gangrene is preceded by inflammation that can be attributed to irritation of the skin by excreta, or washing and drying, or friction against folds in the sheets. Thus, excluding the immediate or instantaneous occurrence of gangrene, most of its forms arise out of an inflammatory process. The chief exceptions are two: cases of strangulation, which cut off the supply of arterial and at the same time prevent the return of venous blood (as in hernia and twist of the spermatic cord); and cases of acute bedsore.

Putrefaction is an essential part of gangrene. Gangrenous pneumonia is, as a rule, the result of particles of animal food being drawn into the bronchioles and there decomposing. The condition may be compared to necrosis of a tooth from lodgment of fragments of food.

In "senile" gangrene the parts dry up and shrivel into a hard material like that of a mummy; and "dry" gangrene was formerly described as the characteristic effect of obstruction of the arterial blood-supply. But it is now known that the desiccation depends upon the escape of fluid from the surface in consequence of detachment of the epidermis. It is only when mortification spreads very slowly through the whole of a limb that it can "mummify." The gangrene which ensues upon embolism of a large artery is more or less moist. But it often happens that deep structures remain soft, while superficial ones dry up into a horny mass, which is termed a hard slough or *eschar*. The colour of gangrenous parts is generally greenish or purplish black, but sometimes grey; it is due to chemical changes in hæmoglobin which has escaped from the blood-discs, and has diffused itself through the tissues. The horrible odours often emitted are attributed to the formation of sulphuretted hydrogen and volatile fatty acids. Gases sometimes collect until the mortified tissues become emphysematous, and crepitate when they are touched. In other cases they are saturated with a thin, dark red fluid, which raises the cuticle into bullæ. That structures which have undergone mortification are devoid of sensibility is well known. They feel cold to the hand, since their supply of warmth is cut off with their supply of blood.

Gangrene is often from the first "circumscribed," or limited to a certain part; and when it spreads, its progress, if arrested at all, is limited by a "line of demarcation." Here active inflammation develops, abundant exudation of leucocytes takes place, and the connection between the living and the dead tissues is gradually eaten away, until the latter become completely detached and are cast off.

Local varieties of inflammation.—It will be well to summarise the various forms which inflammation assumes according to the organs which it affects.

1. The skin is very readily liable to inflammation. This may be slight and transient without visible exudation; or the exudation may produce general swelling—*inflammatory œdema;* or it may appear as clear plasma in vesicles, bullæ, or on a raw weeping surface. Or the exudation may become rich in corpuscles and pustules; pus will result, with the formation of scabs when the pus dries up. Or the inflammation may be chronic, and lead to fibroid thickening from "hyperplasia" of the deeper parts of the skin. When similar proliferation affects the inflamed epidermis, epithelial scales instead of spindle-cells and fibrous tissues are produced, and the inflammation becomes desquamative. Or the inflammation may be destructive, and cause loss of substance in the form of *ulceration.* If this is rapidly progressive it is called *phagedæna.* If visible fragments of dead tissue result, it is called, not molecular necrosis or ulceration, but *gangrene* or necrosis in mass.

2. The mucous membranes rarely form vesicles or pustules. Serous exudation, with more or less admixture of pus, is combined with mucin and with desquamation of the superficial epithelium. The whole process is called *catarrhal* inflammation, and the exudation mucous or muco-purulent, or purulent, *e. g.* ophthalmia. This is the characteristic inflammation of mucous membranes, and the term catarrhal is best kept for it, although it has been extended to any watery discharge, such as the exudation of serous inflammation. Under infection from a specific microbe some mucous membranes are liable to a true diphtherial *membranous* form of exudation. This may affect the respiratory surfaces, including the larynx, pharynx, and fauces, nasal fossæ and conjunctiva, and the trachea and bronchi.

Again, mucous membranes are very liable to *ulceration*, especially the mouth, stomach, colon, rectum, and larynx. Much less frequently they exhibit membranous necrosis, particularly the large intestine, the bladder and uterus.

3. The exudation of serous membranes consists of serum or of fibrin, or, more frequently, a mixture of the two. Occasionally, especially in children, the exudation is purulent; probably this is always due to infection with septic bacteria. The membranes lining the great pleuro-peritoneal sac—pleura, pericardium, peritoneum, and tunica vaginalis—are also liable to chronic plastic inflammation with adhesions and thickening. Other surfaces covered by endothelium and hollowed out of mesoblast which undergo plastic inflammation, are the lining membrane of the heart and arteries

(endocardium and intima), the air-vesicles of the lungs, and the iris ; also, under some conditions, the urinary tubules. Many of these forms of inflammation are bacterial, as ulcerative endocarditis.

4. Connective tissues. Areolar tissue is readily susceptible of suppuration : but this is almost always secondary to pustules or ulceration of the skin, or perforating abscesses from internal cavities, or general pyæmia. It is also liable to acute serous exudation, as in the anasarca of Bright's disease. Fat very rarely inflames—tendons scarcely ever; ligaments only from strains and ruptures, or from synovitis.

5. Cartilage is little liable to inflame, but is capable of ulceration—chronic, or acute and infective ; it may also undergo interstitial inflammatory changes, which make it opaque and calcareous. The cornea resembles articular cartilage in its inflammations, which are either acute and ulcerative or chronic and interstitial. The fibro-cartilages, both yellow and white, seem almost exempt from inflammation.

6. Bone is frequently attacked by acute suppurative inflammation, which usually leads to either molecular ulceration or caries, or else massive sloughing or necrosis. The chronic inflammation of bone is characterised by hardening (osteoporosis), with hypertrophy and obliteration of the cancelli.

7. Muscles are rarely liable to inflammation, and probably never to suppuration. Myocarditis leads to fatty degeneration. Chronic inflammation of the muscles is supposed to be the cause of myalgia, or muscular rheumatism.

8. Peripheral nerves are almost exempt from suppuration. Neuritis, whether parenchymatous or interstitial in origin, ends in thickening of the perineurium and destruction of the myelin.

9. The solid viscera are liable to three chief varieties of inflammation. These are—

a. Infective, suppurative, and acute : traumatic or secondary to other suppuration, and probably always of bacterial origin.

β. Irritative, non-suppurative, parenchymatous, softening, acute or subacute, varying with each organ.

γ. Interstitial, fibrous, contracting, hardening, and very chronic.

Under the first head come abscess of the brain (usually from caries or pyæmia), pyæmic pneumonia, abscess of the liver, "surgical" kidney, and pyæmic abscess of the spleen.

Under the first head come abscess of the brain (usually from caries or brain is inflammatory or hæmorrhagic, and whether acute myelitis is always truly inflammatory ; but some cases of the latter are proved to be so by the meningitis which goes with them. True acute lobar fibrinous pneumonia is the typical instance of this group : but its inflammatory nature has also been called in question ; at least it is never traumatic, or suppurative, or chronic. Acute yellow atrophy of the liver should probably be classed as a diffuse parenchymatous hepatitis. Glomerular and tubal nephritis, acute, subacute, and chronic, is an example of this group. The spleen does not appear liable to a similar affection, nor the lymph-glands, nor the testes, nor the ovary.

Under the third head falls the grey degeneration or, as it is now called, sclerosis (hardening and shrinking) of the brain and spinal cord, varying in its symptoms according to its locality, but always showing the same histological features. The corresponding process in the lung is chronic interstitial pneumonia, iron-grey induration with contraction, also called pulmonary cirrhosis. That in the liver is the chronic interstitial hepatitis, named cirrhosis by Laennec. That in the kidneys is the chronic interstitial form of Bright's disease. A very similar process is seen in the fibroid testis, which results from chronic syphilitic orchitis, and which, when gummata are also present, closely resembles syphilitic cirrhosis of the liver.

Apart from the difference manifested by inflammation as it attacks the several tissues and organs, or the greater or less intensity of the irritation which was set up, or the specific nature of the microbe involved, there are yet other differences which have no relation to the *irritans*, but to the *irritabile*, not to the cause but to the subject of inflammation. Many years ago Paget examined the fluid contained in blisters which had been used in the treatment of thirty patients. In some cases it formed a firm, fibrinous coagulum, in others it was purulent. The former condition was observed in persons who were in sound health, the latter in those who were suffering from phthisis or other chronic disease. Patients affected with renal dropsy are especially liable to sloughing, and the same is true of diabetes.

Children are certainly as a rule particularly apt to suffer from suppuration, and old people from mortification.

Events of inflammation.—If the injury and the resulting inflammation are but slight, *resolution* ends the process. The circulation through the vessels of the affected parts gradually resumes its natural condition; leucocytes which had escaped pass off through the lymphatics, and coagulated fibrin undergoes fatty degeneration and absorption.

If pus cells have accumulated in a serous cavity, or in the sac of an abscess, they die there and break into granules. The fluid is then absorbed, while the solid matter remains as an opaque yellowish mass. This process is known as *caseation*. Such cheesy residues often become hard by deposit of insoluble earthy salts.

When a slough becomes detached, or when an abscess discharges its pus, a hollow space is left, which is filled up by a process of granulation. Ziegler embedded in the subcutaneous tissues of dogs a series of minute flat chambers, each consisting of a pair of glass plates, with a capillary space between them, cemented together at the corners, but open at the sides. These he removed after an interval, and found not only that leucocytes had penetrated into the space between the plates, but sometimes that tissue elements and even blood-vessels had developed there. The earliest step was the formation of large granular cells with vesicular nuclei. They are generally round, but sometimes oval or irregular in shape. Their size is variable, but they are larger than the epithelial cells of the tongue, and go on developing until they become *giant cells*—irregular masses of protoplasm, each containing a large number of nuclei.

Pear-shaped or fusiform giant-cells give off bundles of delicate fibrils, which unite and form connective tissue. New blood-vessels arise as buds and loops from the sides of already existing capillaries.

The connective tissue which is developed out of inflammatory exudation is sometimes of an enormous thickness, as in the pleura, and very slowly reaches its permanent condition.

Inflammatory adhesions and cicatrices have a strong tendency to contract, sometimes with serious consequences, or after a long time they may soften and disappear. Paget cites the following observation of Bichat's: A man had made from twelve to fifteen attempts at suicide, at different times, by stabbing himself in the abdomen. In the situation of the more recent wounds it was found that the intestines adhered to the parietes; but the adhesions corresponding with the older ones were reduced to narrow bands, or had even become divided and were hanging free. In cicatrices also a marked "loosening" may at length take place. These facts, and others, like the ultimate subsidence of sclerodermia, deserve to be borne in mind in reference to analogous changes in internal organs, which we are too apt to regard as hopelessly permanent.

Regeneration of *epithelium* during the subsidence of inflammation takes place after ulceration of the skin or a mucous membrane. That the cuticle which ultimately covers a granulating wound always arises in contact with pre-existing epidermis is well known; Reverdin's practice of skin-grafting is an illustration of it. But the reconstruction of epithelium does not extend to the formation of glandular tissue.

Ætiology of acute inflammation.—That many, probably most, cases are caused by a direct injury or irritant is a matter of common experience. The reaction to a wound, or to chemical, thermal, or electrical injury constitutes "simple" or traumatic inflammation. In another natural ætiological group a specific microbe produces local inflammation as part of a general infective process. A third depends upon infection by pus-producing micrococci; and this may be primary or superadded to a previous infection which it complicates. To the first group belong not only external injuries, but the action of poisons absorbed from the alimentary canal or otherwise. To the second belong the inflammations which accompany smallpox, enterica, pneumonia, phthisis. To the third belong suppurative inflammations.*

There remain examples of inflammation which cannot be assigned to any definite injury of the organ or tissue affected, and in which the presence of specific microbes has been vainly sought—pleurisy, bronchitis, and acute inflammation of the kidneys or spinal cord. They are attributed in popular belief to exposure to cold, but even if this be admitted it is very difficult to explain in what a "chill" consists, and how it produces an internal inflammation.

Again, the inflammation tending to rheumatism and to gout cannot be classed with the specific inflammations of infective disease, and may hereafter be found to have a very different origin.

It is often said that internal inflammations depend on a predisposing as well as an exciting cause; that a chill which has no effect on a person in health will produce pleurisy, pneumonia, diarrhœa, ar acute Bright's disease in one who is exhausted by want of sleep or by want of food. This is probably true, but does not help in explaining the way in which the chill acts. It is more to the purpose to notice that whether predisposed or no, the different organs react with very different readiness to an external chill. There are some organs which appear never to inflame from a chill: the brain and its membranes, the bones, the peritoneum and pericardium, the liver (in temperate climates at least), the spleen, pancreas, adrenals, testes, and ovaries. Of the mucous membranes, those of the entire respiratory tract, including the conjunctiva, frontal sinuses, larynx, and bronchi, are most prone to inflammation "from cold;" next the fauces and intestines; rarely the bladder; while the mouth, œsophagus, and stomach are exempt.

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* There are certain cases, such as suppuration from the irritation of abrus bean, which appear to show that suppuration may take place without the presence of micrococci.

Uses of inflammation.—As in the case of fever, so in that of inflammation, we may inquire whether any beneficial effect can be attributed to the process. While repudiating the assumption of a *vis medicatrix naturæ*, we may admit that the phagocytes have a beneficial action in destroying invading bacteria, that serum has itself a destructive influence in the same direction, and that œdema and suppuration soften the parts and favour the separation of dead tissue.

One useful effect of inflammation is in causing adhesions of serous membranes, which shut off the foci of disease, and prevent circumscribed empyema or peritonitis from becoming general. But adhesions are also a frequent cause of fatal strangulation.

Chronic inflammation.—This term, though often objected to, is inevitable; for although it includes some processes in which the four cardinal signs and the fever which forms the fifth sign of inflammation are absent, yet we are led insensibly from the most recent and typical forms to those where all the signs fail us. Suppuration—plastic and catarrhal inflammation—subacute and chronic inflammation form a series in which it is impossible to draw an absolute distinction at any step. The one event which is common to every step of the series is exudation of leucocytes; the one condition which precedes this process is irritation; the one event which follows in all is the cicatricial inflammatory thickening.

At one end of the scale we have suppuration with softening of the organ; at the other, formation of fibrous (or, as it is often called by a barbarous synonym, fibroid) tissue with hardening of the organ.

Chronic inflammations are the result of slow, long-continued slight irritation; they are unaccompanied by fever; the leucocytes are few and the effused lymph scanty; hence there is no visible pus and no calcareous remains; the natural termination is the formation of fibrous scar-tissue, with thickening of thin, and opacity of transparent membranes, contraction, induration, and adhesions of connective tissue, and atrophy of solid organs.

In most cases in which the ætiology is ascertained, chronic, fibrous, interstitial, atrophic inflammation is due to chemical poisons rather than to mechanical injury or microbial invasion—to plumbism, alcohol, or syphilis.

The familiar examples are chronic inflammation of the several portions of the great serous sac or cœlom, of the pleura, pericardium, peritoneum, and tunica vaginalis; chronic interstitial inflammation of the liver (cirrhosis of Laennec), of the kidneys (contracting atrophic form of Bright's disease), and of the lung (Corrigan's pulmonary cirrhosis); and sclerosis, or grey induration of the brain and spinal cord. All of these are more or less closely connected with subacute forms of inflammation of the same organs, and histologically and physiologically agree with the formation of a cicatrix after a burn or a wound, with the false membrane which surrounds an abscess, and with the tough membrane lining an old tuberculous vomica.

There is a form of fibrous thickening which accompanies long-continued venous congestion with local "asphyxia," or lymphatic obstruction, and which is clearly passive and not inflammatory. Examples are the clubbed bulbous fingers of heart disease and empyema, and the enormously hyper- [illegible] scrotum and legs of elephantiasis.

Granuloma.—We have seen how difficult it is to draw a clear line between acute and chronic inflammation on the one hand, and between chronic inflammation and hypertrophy or fibroid degeneration on the other.

There is, however, another form of chronic inflammation, which ends not in fibrous thickening or hypertrophy, but in the formation of a new growth of cellular tissue which resembles the granulations of an ulcer. This process appears to be intermediate between inflammation and the production of neoplasm or tumours, and the diseases which come under this head were included by Virchow in his great work on morbid growths. Anatomically they consist of young spheroidal cells (leucocytes), often with " epithelioid " or " giant cells " among them. There is no fibrous tissue and a rather scanty vascular supply.

The term *infective granuloma*, which is now commonly applied to the pathological group in question, includes Tubercle, Lupus, Syphilis, Glanders, Leprosy, and the rarer forms of disease known as Mycosis fungoides and Frambœsia. They all agree in their histological characters, and more or less in their clinical course. We now know, moreover, that many (and suspect that all) owe their peculiarities to the presence of microphytes which act as permanent irritants, and keep up a " specific " form of inflammation. This interesting pathological group has therefore relations to acute and chronic inflammation in one aspect, to specific fevers in another, and to tumours and neoplasms in a third. Its several members will be discussed hereafter.

Tuberculosis and syphilis take their place among specific febrile diseases, inasmuch as apart from the local granuloma, there is pyrexia from infection (or, as the French pathologists call it, " intoxication ") of the whole body. Glanders combines local inflammation of a granuloma type, specific infection, generalisation, and fever, and, as a matter of convenience, will be described under the general head of specific fevers along with others of animal origin. Lupus and leprosy will be noticed among affections of the skin.

Several eminent pathologists, Thoma, Ziegler, Cornil and Ranvier, with whom Dr Payne has given his voice in this country, not only give up the attempt to define Inflammation and content themselves with describing the several events observed, but more or less decidedly give up the term itself as obsolete and obstructive rather than helpful. No doubt many of the conceptions associated with the word at different periods must be given up, but regarded as reaction of a living organism against injury, and defined as exudation of leucocytes, Inflammation may still have a place in scientific as well as practical medicine. Doubtless suppuration is very different from serous effusion, and both from chronic fibrous induration. But the points of resemblance are perhaps greater than those of difference, and it is impossible to draw a satisfactory limiting line between them.

This, however, may be freely admitted: that the mere diagnosis of inflammation of an organ, dermatitis, peritonitis, nephritis, is of little scientific or practical value. We must ascertain what is its immediate cause, infective, pyogenic, microbial, tuberculous, traumatic, syphilitic, leprous; and until we have done so must regard the term primary or idiopathic inflammation as of only negative value.

PYÆMIA AND SEPTICÆMIA *

> Τέχνῃ κρατοῦμεν ὧν φύσει νικώμεθα.
>
> ANTIPHON. *fragm.*

Pyæmia—Varieties of blood-poisoning—Sapræmia—Septicæmia—Metastasis—Phlebitis—Embolism—Infection—Microphytes—Practical application—Chemistry of pyæmia—Internal pyæmia—Channels of infection—Local distribution of secondary abscesses—Idiopathic pyæmia—Slighter forms—Diagnosis: especially from enteric fever and tuberculosis—Prognosis.

It is remarkable that little attention was formerly paid to the malignant process which often rendered slight wounds and injuries mortal, and which up to the present day was the great danger to be feared after the most skilful surgical operations. The very names of "blood-poisoning," "septicæmia," and "pyæmia" are recent; while Tetanus, a far less common sequel of wounds and injuries, was familiar from ancient times.

Probably one reason for this was that the immediate effect of wounds in producing what was called "irritative fever" was confounded with the later appearance of pyæmia, and that this was not distinguished from the still later "hectic" (*i. e.* continued) fever which accompanied prolonged suppuration. Another reason may have been that in the hotter climates with which the physicians of ancient Greece were familiar (and those of Italy in the sixteenth and seventeenth centuries) cases of tetanus are more common than in the north of Europe. Lastly, whatever other miseries followed an ancient or mediæval battle, the wounded were at least free from the dangers which made the crowded wards of a city hospital, until recent times, a hotbed of pyæmia.

Pyæmia (wound fever, surgical infection) is happily of much diminished practical interest to the modern surgeon; but its pathological interest is as great as ever, and it enters so far into certain forms of internal disease that a treatise on the principles of medicine would be incomplete without some notice of this general morbid process. Its place is naturally after inflammation and infection, and before specific contagious diseases.

As we shall presently see, the disease known as pyæmia is a complex condition, consisting of—(1) a mechanical process, plugging of an artery by a clot of blood, or sometimes by a mass of microphytes; (2) a biological process, the entrance of a parasitic microbe, which multiplies in the lymph, blood, and tissues, as described in the chapter on Infection; (3) a chemical process, the poisoning of the lymph and blood by the chemical products secreted by these microbes. It is convenient, while keeping the term pyæmia as a convenient or at least an indispensable term for the whole process, to distinguish its several parts, as (1) Embolism—a hydraulic process, (2) Septicæmia—an infective process, and (3) Sapræmia—a toxic process.

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* *Synonyms.*—Infection purulente—Résorption purulente—Pyohémie—Fièvre traumatique ou purulente—Diathèse purulente—Infection putride—Intoxication chirurgicale (Maisonneuve, 1867)—Tabes purulenta (Armstrong, 1732)—Suppurative fever (Braidwood, 'Astley Cooper Essay,' 1868)—Pyæmia (1844, Marcuse, 'De pyæmia quædam,' Königsberg, and Rak, 'Pyæmia,' Prag) quoted in U.S.A. Army Library Catalogue—Putride intoxication [illegible] 1868)—Septicæmia (Urfer, 'De septicæmia,' Bonn, 1864)

Sapræmia.—This condition by itself is seen in cases where a dose of a chemical poison, produced by decomposition of animal tissues, either within or without the patient (*σάπρος*, *putridus*), finds an entrance through the skin or mucous membrane into a lymph-space or vein.

A single dose is rarely dangerous to life; for the products of putrefaction, whether ptomaines or albumoses (cf. *supra*, p. 22), are seldom received in lethal quantity. They become at once diluted by the lymph and blood, and if not reinforced by fresh additions, the danger, if present, is soon over. We may compare such an event to the injection of the poison of a mosquito, a bee, an adder, or a cobra, and the effects vary no less in degree; or we may compare it to the admission of a large single dose of alcohol, of hydrocyanic acid, of belladonna, or of arsenic. In all these cases, if the immediate effects of the poison are withstood, it is gradually eliminated from the organism, and no permanent harm ensues.

But it is very different when a source of "intoxication" is constantly present and efficient. A decomposing fragment of placenta in the uterus, a clot breaking down in a vein, a cavity containing foul pus or putrid blood or sloughing tissues, is a source of constantly renewed supplies of poison. We may then compare the condition of sapræmia to that of chronic alcoholism, to chronic plumbism, or poisoning with arsenic, and the gravity of the case is no less increased.

Nevertheless, just as the physician who can trace the symptoms of a patient to drink or to lead poisoning is able to cure the disease by cutting off the cause, so the obstetric physician can clear out and irrigate the contents of the uterus, and the surgeon can open up and drain the "pockets" and sinuses which supplied the poison to the blood.

The symptoms of sapræmia are pyrexia, muscular weakness, anorexia, diarrhœa and sweating, with sometimes sore throat, and hæmorrhage from the skin and mucous membranes. If the poison starts from the skin, the lymphatics become red and tender, and the lymph-glands into which they pour the lymph are swollen and painful. If death ensues, no points of suppuration are found; but the spleen is swollen and soft, the blood-vessels are stained with hæmoglobin, and petechiæ or larger ecchymoses stud the serous membranes or the muscles. The tissues, like the blood, undergo rapid decomposition, and rigor mortis is early, brief, and imperfect.

Septicæmia.—This term has been used in various senses. It is applied to a specific disease in mice marked by the presence of cocci, often arranged in fours. It has been used as synonymous with sapræmia, or with the distinction that the former denotes blood-poisoning from an external source, the latter the same toxæmia from a source within the body. But it is now most often restricted to any general condition of toxæmia in which the poisons are secreted by parasitic living organisms within the body. From the practical point of view the great distinction is that the supply of virus is not limited, but may be indefinitely continued; moreover, pure septicæmia does not produce embolism.

The symptoms are not always more severe than those of sapræmia, but the disease is far more dangerous, since the source of the mischief is not removable, but consists in countless microbes scattered throughout the blood and tissues. In addition to the fever and hæmorrhage there is often albuminuria, rigors are more constant, and abscesses may occur. The number of organisms found in the blood and tissues of a patient suffering from septicæmia are few, much fewer than in the lower animals, and their character various—bacilli, staphylo- and strepto-cocci. Occasionally they appear to clump together and form an obstruction in a capillary or minute artery, but there are no fibrinous emboli.

The diagnosis from sapræmia, from pyæmia, and from specific diseases like hæmorrhagic variola, typhus, and diphtheria, is often difficult or impossible during life, and not always easy even after anatomical investigation, and cultivation of the blood.

The prognosis is almost hopeless in severe cases; in many, however, the infection is stopped by the lymphatic glands, and the patient escapes, as with an abscess in the axilla after a dissection wound.

Pyæmia.—The discrimination of septicæmia and sapræmia as just defined from a third still more complicated, and at one period more fatal form of blood-poisoning, has been the result of long and instructive investigation.

When an unhealthy-looking wound was accompanied by fever, with wasting, sweats, and more or less jaundiced tint of skin, the earlier pathologists supposed that an "ill habit of body" prevented the wound from healing; and when abscesses were discovered after death in the lungs, liver, and other organs (a fact known to Ambrose Paré in the sixteenth century), they were ascribed to *metastasis*, or transference of "disease" or "inflammation" from the wound to the internal organs. Even the strongest humoralists seem not to have supposed that the ill habit of body was due to poisonous humours absorbed from the wound.

The opinion of Boerhaave in the eighteenth century was that pus entered the veins from a suppurating wound, and produced the metastatic abscesses of the viscera. This theory of pyo-hæmia or pyæmia was, however, not the only one current when pathology revived after the Napoleonic wars; for Hunter had (in 1784) distinguished suppurative from adhesive phlebitis, and attributed the effects of wound fever to the former process of local inflammation. Hence for many years surgeons dreaded injury or even ligature of veins.* Hunter's opinion was adopted by Cruveilhier (1826), who taught that the "pus in the blood" was not derived from the wound, but from the inflamed vein. He ascertained that globules of mercury when injected acted as irritants, and produced inflammation and abscess around the point where they stuck in the capillaries.

One of the achievements of Virchow was to discover the true meaning of this phlegmonous, suppurative, or infective phlebitis. He showed that what was supposed to be a mass of pus-corpuscles, the product of inflammation in the lining tunic of a vein, is really a discoloured clot composed of leucocytes and fibrin. He showed that fragments of this clot may be detached and carried by the still open veins to the right side of the heart, and thence to the lungs; and that in like manner *thrombi* (*coagula*, clot may be carried from the left side of the heart to the systemic capillarie This remarkable process of *embolism*, by which detached particles of cl are carried from its original seat by the blood-stream, had been ind pendently discovered by Dr Kirkes, of St. Bartholomew's Hospit It was extended by the great pathologist of Berlin not only explain the mechanical effects of a plug of fibrin, but also the mo dangerous results when the emboli are derived from a septic or putr thrombus, and act not only as mechanical obstructions to the bloo vessel in which they lodged, but also as centres of infection to the surroun ing tissues. The mechanical effect of emboli upon the hydraulics of t circulation will come under consideration when embolism of the cerebr arteries is discussed. It is of practical importance in this case, and al when it occurs in the retina, in the kidney, or in the spleen; and it most important of all when it affects the lesser circulation and obstructs a main branch of the pulmonary artery. When the clot is septic, two pathological processes are combined, the mechanical effects of the embolism and the infective power of the poisonous embolus. This latter condition was distinguished by Virchow as "septicæmia," not exactly with the same meaning as that above defined.

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* The refutation of the hypothesis that suppurative phlebitis spread by continuity from the wound to the heart was due to Arnott and Dance, who wrote before 1830 (see Virchow's 'Gesammelte Abhandlungen,' S. 637).

The effects of pyæmia could no longer be ascribed to actual contamination of the blood by pus. When pus-cells were ascertained to be only, as Virchow believed, young cells, like white blood-corpuscles, or, as we have seen in the last chapter they are now proved to be, true blood-corpuscles out of place, it was clear that the mere presence of these elements in the blood could no longer explain the process of pyæmia. Moreover the white cells found in the thrombus are not pus (for they have never left their habitation), but leucocytes aggregated by coagulation and not by inflammation. Accordingly, various terms, such as septicæmia, ichoræmia and sapræmia (since applied in special senses) were invented or adopted from earlier pathology to express the element of "blood-poisoning" which complicated that of embolism.

In this as in other departments of pathology, direct experiment upon the lower animals was at once the herald and the guide of speculation,—speculation based on the exact facts of minute anatomy, and controlled by clinical experience. It was found, first, that minute fragments of healthy blood-clot, introduced into the circulation, acted like seeds or any other non-irritant foreign bodies, as mechanical obstructions leading to anæmia, to hæmorrhage, and other local results, but not to suppuration or to fever. Secondly, it was found that pure *pus laudabile* when injected into a vein either mingled with the blood, the corpuscles gradually breaking up and no effect following, or if in large quantity formed an ordinary non-infective thrombus. Thus "pyæmia" was proved not to be what the name signified, pus in the blood. Thirdly, it was found that when ichorous, putrid, unhealthy pus, carefully filtered so as to get rid of all fragments of solid matter, or even dialysed so as to leave corpuscles and most of the albumen and globulin behind, was injected into a vein, the result was pyrexia, disturbance of the nervous system, diarrhœa, and congestion of the lungs; but neither during life nor on dissection after death were "metastatic" abscesses discovered, nor lobular pneumonia, nor embolic "wedges."

Thus the result of observation and experiment up to 1867 was that pyæmia or wound fever was not due to "metastasis" of suppuration from the seat of injury to internal organs; that it was not due to suppurative, still less to ordinary adhesive, phlebitis; that it was not due to the entrance of pus into the blood, nor to mere embolism, nor to poisoning of the blood with chemical compounds in solution—such alkaline products as have lately been recognised as ptomaines; but that it was made up of two pathological processes, embolism and septicæmia. An infective or septic thrombus gives rise to infective emboli, and these reproduce the parent suppuration, not by a mere deposit of pus, but by acting as an irritant, and exciting around them as foci true inflammation which ends in the formation of pus. Every "pyæmic" abscess of the lungs is preceded by lobular pneumonia, every such abscess of the liver or kidneys by corresponding inflammation. The "secondary" abscesses resemble the primary suppuration as the secondary "deposits" of cancer resemble the primary tumour.

But all these observations and experiments were, in the words of Bacon, "light-bearing" rather than "fruit-bearing." Knowledge of the process of pyæmia had come, but power to prevent it lingered. The mortality from pyæmia after operations in well-ventilated, cleanly hospitals was grievous, but in hospitals like the old Hôtel Dieu, of Paris, or the old hospital at Halle, it was terrific. In order to avoid this dreadful scourge the most elaborate dressings were devised, as complicated, as cumbersome, and as useless as the armour of the fifteenth century; breasts were removed by *cautérisation à flèches*, wedges of chloride of zinc were thrust into the tissues and allowed to eat away the organ, tumours were grubbed out with the fingers, and even limbs were amputated by an *écraseur*. Meantime thoughtful surgeons noted that pyæmia, so fearful under all the appliances of great cities and the dexterity of the most skilful operators, was far less frequent in country places, in open tents and in cottage hospitals.

At length the explanation came. It was not phlebitis, nor access of air, or oxygen, or pus itself, but the invasion of certain lowly organised plants which made the utmost skill and the most elaborate precautions unavailing, and which had almost led some distinguished surgeons to abjure operations altogether. A brief account of these microphytes was given in the chapter on Contagion. They have been divided by their physiological effects, rather for convenience than as a scientific distinction, into those which are fermentative (secreting enzymes), those which form pigment (chromatogenic), those which give rise to specific diseases (pathogenic), those which produce putrefaction (saprogenic), and those which cause suppuration (pyogenic). The members of the last group, the septic bacteria and micrococci, are those which cause pyæmia. When they are precluded from gaining access to a wound, no infective thrombi are formed, no septicæmia occurs, no fever results, and no secondary abscesses are produced.

The recognition and classification of these perilous microscopic organisms was the work of many observers—chemists and botanists, physiologists and pathologists—among whom Pasteur is entitled to the foremost place. The application of this knowledge to the practical end of preventing the mischief they do was due to the scientific training and the philosophical insight, the skilful ingenuity and the untiring efforts towards perfection of methods which enabled our illustrious surgeon, Lister, to effect a revolution in practice.*

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* "Putrefaction in wounds may be avoided without excluding the air, by dressing them with some agent capable of destroying the vitality of the atmospheric organisms, provided that it does not act with too great violence upon the human tissues."—LISTER.

Pyæmia, then, though like other terms it may be retained when its original significance has been disproved, is a somewhat complex condition. It combines (1) sapræmia or absorption of soluble chemical products; (2) thrombosis and embolism or transference of minute clots from the veins to the lungs and thence to the systemic capillaries; (3) the presence of infective microbes, surviving their transference in the blood-stream, and propagating in the spot to which they are conveyed; (4) local inflammation of an infective and virulent character, which leads to (5) necrosis and abscess.

Probably the locality of a pyæmic focus is often determined by the size of the capillaries of the part; those of the lung are comparatively large, and transmit objects which will block the more minute vessels of the joints. But beside this, Wilks and Moxon ('Path. Anat.,' p. 626) argue with much force that local stagnation (as in the back of the lung when a patient is in bed, in the cerebral sinuses, in the large veins of the prostate, and in the recesses of the valves of veins) will cause an accumulation of infective particles, and thus determine the local foci of their earliest and greatest activity.

Chemistry of pyæmia.—The first scientific attempt to determine the chemical aspect of pyæmia or blood-poisoning was made by Panum, the physiologist, at Copenhagen in 1856. He not only injected foul secretions from wounds into the veins of animals and obtained symptoms of sapræmia, as had been done before him, but he ascertained that the virus was not destroyed by heat, and so was not a living organism; that it was not left upon a filter, and so was not participate; and that it was active after being precipitated by alcohol and redissolved in water. Selmi in Italy, and Brieger in Germany, ascertained further that the virus was a soluble alkaline compound belonging to the groups of amides, and identical with ammoniacal products of putrefaction of albumen, which had been isolated by Armand Gautier in 1872. On these the general term *ptomaine** was imposed, while separate kinds were distinguished as cadaverine, sepsine, &c.

These toxic products of the animal body are closely allied to the vegetable poisonous alkaloids, such as atropine, digitaline, nicotine, and muscarine. Indeed, the last-named physiological product of the fly-fungus has been obtained by Brieger from the decomposition of fish.

Beside these alkaline toxines it is now discovered that there are albuminous poisons which are also the products of fermentation and secretion. Such are the albumoses which are present in snake venom, and the proteid discovered by the late Dr Wooldridge in the thymus and testis, which produces coagulation when injected into a vein.

Internal pyæmia.—In the great majority of cases pyæmia results from an external wound, accidental or operative, and so far it belongs to what is called surgical pathology rather than to internal medicine. But not unfrequently we meet with cases where the origin of the pyæmic process is internal and not traumatic. There is probably always a breach of surface; but it is not always a mechanical breach in the skin or accessible mucous membranes.

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* This barbarous word is derived from πτῶμα, *cadaver*. A still more grotesque term was invented to indicate toxines secreted from proteids in the living body—leucomaine, from λεύκωμα, *albumen*.

One variety of internal septicæmia, and the most important by its frequency, is when infection takes place from the uterus after delivery. The peritonitis, and other symptoms which follow the formation of septic thrombi in the great uterine sinuses, constitute one form of the dreaded puerperal fever. Here also—as in the treatment of an amputation-stump, to which Simpson compared the uterus after parturition—antiseptic precautions have been successfully introduced.

Another form of internal pyæmia is when the origin of the process is in bones, most often in the petrosal bone of the skull; such cases will be described under diseases of the brain. A third depends on suppuration of the urinary tract, but this is most frequently the result of stone in the bladder or of stricture, diseases which admit of mechanical treatment; so that urinary pyæmia, or, as it has been called, "erysipelatous," "diphtheritic," or "infective" inflammation of the urethra, bladder, and kidney, falls for the most part within the arbitrary limits of surgical pathology.

Again, when there is suppuration in the alimentary canal (as in dysentery), a local or a general infection of the blood may result. If local it is confined to the portal system, and thus Pylephlebitis is recognised as a portal pyæmia starting in the intestine, the gall-passages, the stomach, or (very rarely) in the spleen. In another local form of acute pyæmia the origin of the infection is in the bones—malignant osteomyelitis.

In most of these cases the primary seat of inflammation is more or less directly in comunication with the exterior of the body, and therefore liable to contamination from bacteria on the surface. But in another remarkable group of cases the original focus of infection is in the interior of the vascular system. These cases of ulcerative, infective, or malignant endocarditis are in the fullest sense internal pyæmia, and here, as was first shown by Professor Heiberg ('Virchow's Archiv,' Bd. lvi), the primary ulcer is characterised by the presence of septic micrococci. The microbe is not always the same; it may be a strepto- or staphylo-coccus, or the diplococcus of pneumonia, or the rod-shaped organisms of enteric fever or tubercle. This remarkable variety of disease will be described under Diseases of the Heart in the second volume of this work. How microphytes gain entrance to the interior of the cardiac chambers is most difficult to understand, but not more so than their access to the bones in cases of acute osteomyelitis. Infective endocarditis is the only—or almost the only—instance in which the infective thrombus starts, not from the veins or sinuses, but from the left side of the heart. It was, therefore, aptly named "arterial pyæmia" by Sir Samuel Wilks.

Mode of infection.—The virulent, infective or septic, bacterial inflammations which accompany pyæmia appear to spread in three ways:

(1) By continuity: chiefly seen in the case of mucous surfaces, as when a gonorrhœal urethritis extends to the bladder and then along the ureter to the pelvis of the kidney; or when scarlatinal angina passes along the Eustachian tube and infects the middle ear; or when septic metritis travels up the Fallopian tube to the peritoneum.

(2) By lymphatic channels: as when an unhealthy sore on the finger sets up inflammation of the lymphatics of the arm and causes a glandular abscess in the axilla. By a somewhat different route, but probably still by means of the large lymphatic channels which unite the areolæ of connective tissue with each other and with the great serous lymph-sacs of the

body, an acute suppurative pleurisy spreads to the pericardium, or infective parametritis to the peritoneum, or bacterial peritonitis to the pleura.

(3) By blood-vessels: in this process alone of the three, septicæmia and conveyance of septic organisms is combined with thrombosis and embolism. It is the commonest and most fatal of all forms of pyæmia. It may infect the veins of the extremities after amputation, the sinuses of the dura mater from caries of the bones of the skull, the portal system of veins from dysentery, or the systemic capillaries from ulcerative endocarditis. The last is the most important case of arterial pyæmia, and causes suppurating thrombi and bacterial abscesses in the brain, spleen, or kidneys, exactly as venous pyæmia causes bacterial abscesses in the lungs and portal pyæmia in the liver.

Apart from these forms of local pyæmia, which can be explained by the anatomical relations of the parts involved, certain remarkable facts have been observed which appear to point to the great principle that, when morbid influences affect the whole body in common, each tissue and each organ has its own greater or less proclivity to suffer, and each has, to some extent, an individual power of modifying the general process in its own case. We see this law in operation when the organs and tissues are affected by starvation, by overfeeding, by lead poisoning, by alcohol, by the diseases which, like poisons, affect not the blood only, but blood and bone and flesh—tubercle, syphilis and cancer, lardaceous and fatty degeneration, scarlatina, and malaria. Every organ and tissue must be acted on by the same exciting agent in each of these diseases, but all react differently. Some are suitable harbours for infection, others are refractory and resist the invasion.

So with pyæmia. Percival Pott in the last century noticed that abscesses in the liver were particularly apt to follow injuries to the skull. The serous membranes suffer most in puerperal septicæmia. The lungs and liver are more prone to pyæmic suppuration than the brain. The pancreas and mamma and testis almost always escape. Arterial pyæmia affects the spleen, the kidneys, and the brain more often than other organs. Infection starting from a bone is most apt to produce secondary abscesses in the heart and in the kidneys, as Wilks and Moxon discovered. Mr Watson Cheyne believes that in the case of the kidney, infective microbes may pass from the blood into the tubules with the urine, and there develop freely and form abscesses. He would apply the same hypothesis to the curious cases of suppurative parotitis after operations on the peritoneum to which Mr Stephen Paget has drawn attention.

Idiopathic pyæmia.—Ulcerative endocarditis, portal pyæmia, urinary pyæmia, and cerebral pyæmia, with the results of infective caries of the vertebræ and pelvis, are the most frequent kinds of internal or, as it may be styled, "medical" pyæmia. Occasionally it results from an empyema or from some internal abscess, from typhlitis, or from sloughing ulceration of the fauces, the rectum, or the skin.

The most puzzling cases to account for are those which we are forced to call Idiopathic Pyæmia (see case below, p. 72). It occasionally happens that on a *post-mortem* examination unmistakable signs of general infective embolism are found, lobular pneumonia with pulmonary abscesses, ecchymoses of the pleura and pericardium, staining of the inner surface of the aorta and multiple abscesses in the liver or kidneys or spleen, or suppuration in the joints; and yet it is impossible to discover the source of the infection. There is no external injury or wound, the urethra is found healthy, there are no anal fissures or inflamed piles, no chronic ulcer of the fauces or pharynx or nasal fossæ, no necrosis of the long bones, or caries of the vertebræ or ilium or internal ear. In one case, the late Dr Moxon, after laboriously searching these and other parts, extracted every tooth from its socket to make sure that there was no alveolar abscess or caries; yet all was in vain. Sometimes we are rewarded by discovering the source of the mischief in unexpected situations,—a pin or other foreign body in the appendix cæci, caries of the ethmoid bone or of the sacro-iliac joint, pyosalpinx as the result of gonorrhœal inflammation of the vagina. But in a few cases we are compelled at the end of the search to confess our failure, and to call the case one of idiopathic or spontaneous pyæmia.

Probably no pathologist will admit that bacteria appear in the blood spontaneously. They must, either as minute micrococci, or possibly as still more minute bacterial spores, gain an entrance by some undiscovered breach of surface.

Slighter forms of pyæmia.—It is probable that under this head should be classed certain cases, common in surgical practice, and not unknown in the medical wards of a hospital, where a patient, suffering from some internal suppuration which has been relieved either naturally or by incision, is from time to time attacked by more or less marked rigors, rise of temperature, quick pulse, loss of appetite, and sometimes profuse sweats. We can usually trace this to the flow of pus being interrupted, and when free exit is restored the symptoms disappear without further inconvenience.

Such accidents we see in the course of an empyema which has been opened, of purulent pyelitis, of suppurating hydatid of the liver, and of otorrhœa from caries of the petrosal bone. In many instances an abscess has been opened under strict antiseptic precautions, and there is no reason to suppose the entrance of impure air or secretions afterwards. The symptoms referred to are certainly connected with the retention of pus, and disappear when free discharge is established. They are quite different from the mere effects of pain when healthy secretions are retained, or when an over-full cyst or an inflamed testis produce general disturbance by pressure, which disappears when the tension is relieved. Hence these symptoms cannot be referred to the mechanical effects of pressure upon nerves. They seem more likely to be caused by the increased pressure leading to entrance of chemical products of inflammation, or possibly of septic organisms themselves, into the adjacent lymph-spaces or blood-vessels.

Of the same character are probably the rigors and pyrexia which follow operations on the urinary organs, and sometimes even the first passage of a catheter. They are not pyæmic, as is the severer form of urinary infection, but sapræmic; and the toxins are probably, according to Bouchard and Harrison, derived from the urine itself.

The most favourable form of true pyæmia is when the secondary suppuration is confined to the subcutaneous tissues or the joints. These cases of chronic or benign pyæmia were admirably described by Sir James Paget, and have more than a pathological interest, since, by early evacuation of the pus, recovery may be often secured.

Diagnosis.—The symptoms of pyæmia as it occurs after a surgical operation are happily less familiar to the present generation than they were before the introduction of antiseptic and aseptic surgery. Still, they occasionally occur, and are well recognised. The difficulty arises when no wound or injury is found to account for the symptoms.

The cases of internal or non-traumatic pyæmia present themselves to the physician under the form of fever, and a high temperature is the only constant symptom. It may be accompanied by local pain, by tenderness, or by œdema, which are of the utmost value in leading to the source of mischief. But these are often absent. Headache, delirium or stupor, muscular twitchings, diarrhœa, sweating, jaundice, are none of them constant symptoms, and all may be present in cases of typhus, of enterica, or of tuberculosis; but a history of rigors, the presence of profuse sweats and an icteric tint of the skin or conjunctiva are more distinctive signs of pyæmia. Pain and swelling of the joints may also be present and simulate rheumatic fever. In cases of ulcerative endocarditis the cardiac murmurs with pyrexia sometimes make this last resemblance extremely close, and pericarditis and pleurisy may rather increase than diminish the difficulty of diagnosis, while the fact of previous attacks of rheumatism is far from making the more serious alternative unlikely. This difficulty will be discussed in the chapter on Rheumatism in the second volume of this work. The age of the patient, the persistence of local synovitis, the presence of jaundice, and signs of embolism in the brain, spleen, kidneys, or retina, may more or less conclusively decide the diagnosis, but in some cases it is impossible to decide until time has developed the course of the disease. Moreover, rheumatism occurring repeatedly with concomitant lesions of the valves may at last appear in combination with a new and infective form of endocarditis.

The most characteristic single symptom of pyæmia is pyrexia, which is of an intermittent type, falling every now and then to normal or even lower, but rising suddenly and irregularly, to fall again with equal abruptness. It has none of the regular daily variations and steady general rise, culmination, and subsidence which we observe in typhus, enterica, and the exanthems. It has not the regular evening rise of hectic fever, which in many respects it resembles, nor the frequently remittent temperature, alternating with irregular elevation, characteristic of some forms of acute tuberculosis. Hyperpyrexia is more common than in enteric fever or in phthisis. The resemblance to a regular form of remittent malarial fever is sometimes remarkably close and deceptive. Appetite is sometimes retained, and the mind often remains clear till nearly the end. The pulse is febrile, the tongue furred, and the breath often has a sweetish smell.

When a patient is found suffering from fever not due to external inflammation, we first ascertain the absence of internal acute disease, and especially of pneumonia and of phthisis. We may then have to wait the appearance of a characteristic exanthem, as the rash of scarlatina, the mulberry eruption of typhus, or the erythematous blush of erysipelas—which is often delayed for a day or two after the temperature has risen. If the first week of fever has passed without these appearances, and if there are no physical signs of pneumonia—which sometimes only appear after two or three days of pyrexia—we then reconsider the possibility of enteric fever and of acute tuberculosis, and lastly the question of internal pyæmia.

Enterica usually begins insidiously, the temperature rises gradually, and the state of the tongue and of the bowels make diagnosis easy even before the rose-rash appears. Occasionally it begins more abruptly, the temperature rises high in the first week, the rash is absent or has passed unnoticed before the patient was seen, the bowels are constipated, and diagnosis may then become extremely difficult.

Acute tuberculosis can usually be traced to previous tubercular disease of the lungs or lymph-glands or abdomen, of which the signs remain in evidence, or we may find a caseous testis or symptoms of "strumous" pyelitis. Again, we may have evidence of meningitis, or may by chance discover a tubercle in the choroid. The lungs are in these cases almost always the seat of disseminated tubercles, and this may be recognised, not so much by physical signs, which are often limited to a slight occasional rhonchus, but by the high ratio of respirations to pulse and temperature, by slight duskiness or marked cyanosis, by the alæ nasi working in aid of the chest, and by dyspnœa unaccounted for by examination of the chest.

When combined with tuberculous enteritis in a child, it may be almost impossible to distinguish acute tuberculosis from enteric fever until the subsequent course of the disease makes it clear.

When we suspect pyæmia, our first investigation is directed to the ear for a purulent discharge, to any of the bones or joints which may be tender or œdematous, to the genito-urinary organs, and to the heart. Symptoms of pyæmia, with a cardiac murmur, point almost certainly to ulcerative endocarditis; this is the more probable if previous valvular disease has existed, and particularly if the febrile symptoms have come on during convalescence from pneumonia, as was stated by Dr Osler in his lectures before the College of Physicians; and the signs of embolism in other organs may confirm this diagnosis. In the rare case of this disease affecting the right side of the heart, of which I once had a characteristic example, the lungs are the first organs to be affected, and the diagnosis is comparatively easy. When from arterial pyæmia infective emboli lodge in the spleen it becomes enlarged, partly by the formation of hæmorrhagic wedges (*infarcta*), partly by the febrile intumescence of the whole organ.

The splenic dulness being increased, and the viscus being felt below the ribs on inspiration, does not in itself distinguish the case of pyæmia from one of enteric fever or of ague, but the "typhoid spleen" and the "ague-cake" are not usually sensitive, while the pyæmic spleen is tender to the touch, probably by reason of acute local peritonitis. Hæmaturia, hemiplegia, and, above all, the presence of icterus would confirm the diagnosis.

When otorrhœa, especially fetid otorrhœa, with a perforated tympanic membrane, is present, we may discover tenderness or œdema over the mastoid process or down the neck. The infection travels by the internal jugular vein to the heart, and then affects the lungs, setting up suppurative lobular pneumonia and acute pleurisy.

On the whole, it may be said that in most cases internal pyæmia has an origin which with care and patience can be discovered during life; that enterica is the most variable of all specific fevers in its course, and is the one most often unrecognised; and that in doubtful cases we may remember that common things most commonly occur, or, to state a converse truism, that we rarely meet with rare diseases.

The following example of idiopathic pyæmia shows the difficulty of diagnosis, or at least the fact that a wrong diagnosis may be made.

A girl twenty years of age, who had been confined four months earlier, was admitted into Mary Ward on December 6th, 1886. She was in high fever, with more or less constant delirium and severe headache, and photophobia. She had, we were told, complained only that morning, had gone out without eating her breakfast, and was found moaning and almost insensible in a corner of the room where she had gone to sell bottles to the hospital patients. The pupils were contracted, there was broncho-pneumonia on both sides, and the temperature rose, notwithstanding cold affusion and ice-bags, to 105·8°. Her mother and a brother had died of consumption.

Next day she was duller and lay curled up in bed. There was a faint apex-systolic murmur. On the third day a pleuritic rub was heard, and the respirations rose to 54, with temperature 104·8°, and pulse 140. The next day the fever rose to its highest point, and was once more reduced by ice; albumen appeared in the urine, and she died after three and a half days' illness. I supposed that she was suffering from acute tuberculous meningitis with similar disease of the lungs. The brain, however, was found normal. There was recent plastic pleurisy and a vomica in the apex of the right lung, but no miliary tubercles in the lungs. There was no ulcerative endocarditis, but a little recent lymph on the mitral curtains. One small abscess was found on the surface of the liver. The spleen was swollen but contained no embolic foci. Both kidneys contained numerous small abscesses as in ascending pyelitis, but the pelves were normal, as were the ureter and bladder. The vagina, uterus, and Fallopian tubes were healthy; one ovary contained a serous cyst. The skull and internal ear, the sinuses, the vertebræ and sternum, and all the large joints were searched and all the internal organs, but nothing further was found except one small ulcer in the duodenum. The portal vein was perfectly normal.—C. H. F.

Prognosis.—As a rule cases of pyæmia are hopeless, and the exceptions are very few. But there are cases of chronic pyæmia above mentioned where the fever is never high, internal organs are spared, and the joints are successively attacked by suppuration. These cases sometimes do well, and it appears certain that some forms of bacterial infection can be dealt with successfully by phagocytes when the number of microbes admitted is not too large. So that we may say that, even with our present means of treatment, pyæmia is not an absolutely fatal disease.

The indication is to ascertain, if possible, the source of infection, and give free exit to the septic materials. When an empyema has been laid freely open and drained, a mastoid bone trephined and the pus given exit, a sequestrum of bone cut down upon, or a joint freely incised, infection may not only be avoided, but even a pyæmic process already begun may be prevented from becoming general.

The discovery of the antitoxic power of serum from immune animals has been applied to the treatment of septicæmia, and anti-streptococcic serum has been injected in cases of internal pyæmia with promising results. Unfortunately its action appears to be limited to cases of infection by the same microbe as that which was present in the animal from which it was prepared.

NEW GROWTHS

Καρκίνωμα ἐστιν ὄγκος κακοήθης, καὶ περίσκληρος, ἀνέλκωτος ἢ ἡλκώμενος· εἴρηται δὲ ἀπὸ τοῦ ζώου καρκίνωμα.*—GALEN.

New growths—Definition from hypertrophy and from granulomata—Innocent and malignant tumours—Virchow's characters of malignancy—Malignancy not an attribute of structure—Histological classification of tumours—Mesoblastic growths—Sarcoma: round, spindle, and giant cells; hæmorrhagic and alveolar sarcoma: melanotic sarcoma—multiple sarcomata as a general disorder—osteitis deformans—Carcinoma: glandular, colloid, and squamous—Distribution of primary and secondary carcinoma—Histogenesis—Heredity—age and sex, etc.—Theory of cancer.

THE term Tumour was once applied in modern languages (as *tumor* in Latin) to all swellings which were not obviously inflammatory, so that it included hydroceles, hydatids, hypertrophy of the spleen, enlargement of the liver, and even the swelling of a dropsical limb. One of the first lessons in morbid anatomy was that circumscribed solid masses, like external wens, are to be met with in the internal organs as well as on the surface. From a pathological point of view these solid outgrowths were distinguished as *pseudoplasms*, *neoplasms*, or *new growths*.

When a tumour grows on mucous membrane, it often projects from the surface, and may hang by a distinct pedicle; it is then (by a curious perversion of an ancient medical term) called a *polypus*.† When it forms a rounded mass in a solid organ it is called a *nodule* or a *tuber*.

The essence of a tumour, in the modern sense of the word, is that it should grow by its own impulse, on the common nutriment conveyed to it and to the surrounding healthy tissues by the blood.

In practice it is sometimes difficult to decide whether a new growth of a normal tissue should be called a tumour or a hypertrophy. A lipoma that can be shelled out from the subcutaneous fat around it or a pedunculated exostosis growing from a phalanx is a tumour; but hypertrophy of fat in one region or overgrowth of the bones of the face (known as *Leontiasis ossea*) may with equal propriety be termed a local hypertrophy or a diffuse new growth.

Again, it is sometimes difficult to distinguish a chronic inflammatory swelling from a new growth. The group of morbid structures which

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* "Cancer is a hard and malignant swelling, either with or without ulceration, and derives its name from the animal" (*cancer*, a crab).

† Polypus was a term originally applied to the branching blood-clots of fibrin found in the heart.

Virchow collected and named granuloma was included in his great work on morbid growths. It includes syphilitic nodes, tuberculous nodules, and fungating masses, such as are seen in glanders, in leprosy, in rhinoscleroma, in mycosis fungoides, or in frambœsia, and ulcerations which will not heal and yet are not cancerous, like lupus of the skin or larynx.

These granulomata consist of large round nucleated cells, such as are found in certain kinds of sarcoma, but also in healing ulcers, of inflammatory or exudation cells, of smaller lymph-cells, and of giant-cells, each with several nuclei. None of these cells are distinctive or peculiar, but are common results of long-continued irritation. Purulent secretion takes place, and some amount of pyrexia is often present, so that these granulation-growths closely resemble chronic inflammatory products.

Since Virchow's work was published, it has been ascertained that tubercle, glanders, leprosy, and lupus are the products of microphytes. In all probability the same is the case with the other members of the group, and if this proves to be the case, they will thus be definitely separated from ordinary acute or chronic inflammation on the one hand, and from new growths on the other.

Benign and malignant growths.—It was early noticed that whereas some tumours remain for years without affecting the health or causing any inconvenience, others rapidly destroy the patient's life. The distinction between "innocent" and "malignant" growths was therefore natural and obvious. Further observation showed that whereas those of the former kind are like the natural tissues of the body—fat, or cartilage, or bone—most of the latter are unlike any healthy tissue. The microscope confirmed this distinction, and thus an anatomical division was made between *homologous* growths, generally innocent, and *heterologous* growths, generally malignant. It was further supposed that, although there were several varieties of malignant growths, differing in character and appearance, yet all these belonged to a single disease, which was termed Cancer.* Its tendency to destroy the surrounding tissues rather than its anatomical structure was regarded as its fundamental character.

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* Cancer is merely the Latin translation of *καρκίνος*, a crab (see the quotation from Galen at the head of this chapter). This word was applied by the Greek physicians to any eating, gnawing sore. Both *καρκίνος* and *καρκίνωμα* occur in this sense in Hippocrates ('Aph.,' 1257 *et passim*) as well as in later writers. Celsus speaks of diseases "cum quid intra se ipsum corruptum est, ut in *Cancro*," in close proximity to those which result "cum quid extrinsecus læsit ut in vulneribus" (lib. v, cap. xxvi, 1). A little further on (ibid., xxvi, 31) he says that a cancer ensues from too great inflammation, or too great heat or cold, or too tight binding of a sore, or because the patient is old or of an ill habit of body. "Omnis autem cancer non solum id corrumpit quod occupavit, sed etiam serpit." He goes on to say that the Greek physicians divided cancers into species for which there were no Latin equivalents: as erysipelas, gangrene, &c. He describes (ibid., xxxviii, 2) *καρκίνωμα* as chiefly affecting the upper parts of the body, the nose, ears, lips, and the breasts of women.

Galen admits, however, of *καρκίνος χωρὶς ἑλκώσεως* ('De tumoribus præter naturam,' c. 12), and Paulus Ægineta says "Cancer tumor est inæqualis—interdum sine ulceratione, quem Hippocrates latentem (*κρυπτὸν*) nominavit." This author derived the name from the distended veins of a cancerous breast resembling the legs of a crab (lib. vi, cap. 45), and so Galen; others from the difficulty of getting rid of it ('Expos. vocum medicinalium,' 1564).

Our English word *canker* is but an older form of "cancer," and more generally applied. It is used in the Authorised Version of the New Testament to translate the Greek *γαγγραίνα* (2 Tim. ii, 17).

In the same way the venereal ulcer, called in French "chancre," bears the same name slightly modified.

Lastly, "cancrum oris," or noma, a sloughing ulceration of the mouth in children, shows how widely the term was once used.

Lebert and the early pathological histologists who supposed that the tissue of a new growth differed completely from the normal tissues of the body, made it their aim to discover some particular specific element or "cancer-cell," the presence of which might be an infallible criterion, and solve the frequent difficulties which arose in the anatomical diagnosis of tumours.

In 1847, and the years which followed, the genius of Virchow placed this question in an altogether new light. It had already been shown by Johannes Müller that the structure of every growth, however heterologous in appearance, always corresponded in its ultimate elements with some natural tissue, either permanent or embryonic. Virchow carried the same idea into full detail. As he pointed out, the physiological type of the cells which had been supposed to characterise cancer is to be found in the epithelium of mucous membranes; while the round or spindle-shaped cells which really make up many malignant tumours correspond with those which are seen during the development of connective tissue. He insisted that the structure of a tumour in man is always human; so that, for instance, such a tumour might contain hairs, but never feathers; whereas in a bird a tumour might contain feathers, but not hairs. For Virchow, therefore, no growth was *heterologous* in the sense hitherto attached to that term. The distinction, as he drew it, was between homologous tumours which resemble in structure the tissues in which they occur, and heterologous tumours which differ from these, though they resemble other tissues of the human body. Thus the same growth, which in one situation would be homologous, would in another be heterologous. He further showed that between certain tissues there exist close relations which are wanting between them and other tissues, and that such relations have important bearings upon pathology. Thus, all the structures belonging to the connective-tissue series, being related to one another by common origin from the mesoblast, by histological structure and by function, the presence of bone, or of cartilage or of fat in fibrous tissue would not be inconsistent with homology, whereas that of hair or of glands would be.

A still greater advance was made by Virchow in regard to the mode of development of tumours. It had before been generally held that cancer was something deposited from the blood. Chemists had made analyses in the hope of finding in it some peculiar principle for which the name "carcinomatin" had been invented in advance. Its cause was believed to be a "dyscrasia" or ill-mixture of the blood. The fact that active tubercle and cancer are but seldom found in the bodies of the same individuals was thought to support this notion, it being supposed, first, that tubercle is deposited from the blood, and secondly, that the blood could hardly present two different dyscrasiæ at the same time. Yet the fact that wounds and injuries undergo repair in exactly the same way in those affected with cancer as in other persons should have led to doubt of this hypothesis.

Virchow, in accordance with his dogma, *omnis cellula e cellulâ*, maintained that the substance of a cancer was developed *in situ* by the growth of the cells and other structures of which it was composed. He was mistaken in the view that they arise by a proliferation of the connective-tissue corpuscles. But this was unimportant in comparison with his rejection of the notion that cancerous and other tumours were "deposits from the blood."

One necessary consequence of Virchow's conception of Cancer was that the so-called cancerous cachexia, instead of preceding the formation of malignant growths within the body, must be a result of their presence; and this is quite in accordance with clinical experience. No doubt a cancerous patient often becomes cachectic, his skin acquires a waxy yellow colour, and his features look pinched. But in such cases the tumour wears down its victim by pain even if it does not affect a vital organ or become complicated by ulceration or hæmorrhage. Surgeons who were not blinded by dogma always recognised that some women with cancer of the breast were well-nourished, ruddy, and in all other respects healthy. Moreover, some innocent growths, when they cause severe pain and much bleeding, are accompanied by well-marked cachexia; for instance, certain uterine polypi, and even hæmorrhoids.

Virchow defined the term *malignancy*, apart from a tendency to destroy life, by the following four characters.

1. *The tendency of a growth to extend into the tissues around it.* Malignant tumours show little or no respect to the natural boundary lines marking off one kind of tissue from another. Thus in a case of a cancer of the bladder, recorded by Dr Fagge, the tumour having doubtless started in the mucous membrane, ate its way through the whole substance of the organ, through both layers of the peritoneum, through the muscles, aponeuroses, and other structures of the abdominal wall, until it formed an immense ulcerated cavity, reaching from the pubes to the umbilicus.

An exception to this power of overcoming the resistance offered by the different tissues is the fact that malignant growths seldom penetrate the walls of the larger arteries, although they frequently extend into the interior of veins, even into the venæ cavæ. Cicatricial structures also sometimes form a barrier to the spread of a cancer, so that when recurrence takes place after an operation the disease will creep along one side of the scar without crossing it.

It is a point of interest that cancers are capable of passing across a serous cavity without previous union between the two surfaces. Of this Dr Fagge met with the following remarkable instance. The fundus of the uterus being occupied by a malignant growth, the great omentum hung down into the pelvis, so as to lie in contact with it: these parts were not in the slightest degree adherent to one another, but the lower edge of the omentum contained a cancerous mass. It is possible that infection in such cases is the result of active amœboid movements on the part of the cells of the primary tumour; Waldeyer ('Virch. Arch.,' lv) detected slow changes of form in cells from a cancer of the breast, and in those of a round-celled sarcoma of the axilla, when placed on a warm stage.

2. *Its tendency to return* after having been extirpated by the surgeon. This is one of the facts which used to be urged in favour of the constitutional origin of cancer. It really depends on the fact that a malignant tumour always has outgrowths far beyond the area in which the tissues can be seen by the naked eye to be affected. It is true that surgeons are seldom able to prevent the return of the disease by increasing the size of the mass which they excise; but this depends upon the apparently healthy surrounding tissues being infiltrated with malignant cells. For in the case of double organs, like the breasts, the removal of the whole of one gland for a

malignant growth is not followed by recurrence in the organ on the opposite side, as might have been expected if cancer were a constitutional disease. The same experience applies to the testes.

Since it has become the practice to remove the whole breast or the whole tongue instead of the growth in it, and to operate before the lymph-glands have become implicated, the results of operations for cancer have been much improved.

3. *Its spreading to lymph-glands.* As a rule, the glands first attacked are those which immediately receive the lymphatic vessels from the diseased part; for the lymph-spaces round the margin of a tumour take up from it something which is carried into the lymph-stream and deposited in the next gland. There is no difficulty in supposing that this something consists of nuclei, or even of cells from the growth; for Virchow has shown that sometimes an entire lymphatic network—beneath the pulmonary pleura, for example, or the peritoneum—may become filled with such materials, as if it had been artificially injected ('Kr. Geschw.,' p. 52, fig. 4). Whether lymphatic channels exist in the interior of tumours was doubted by Cohnheim, although Van der Kolk long ago, as he believed, discovered them. Most histologists now believe that there are lymph-spaces as well as blood-vessels between the cells of sarcomata and in the fibrous stroma of cancers, but not in its "alveoli," tubules, acini, and ducts.

4. *Its spreading to distant organs and tissues by infection, or, as was formerly said, by "metastasis."*

It is not the mere multiplicity of tumours which indicates that they are malignant. Molluscum contagiosum, fatty and fibrous tumours, and exostoses are sometimes present in great number, and yet are perfectly innocent. But all such innocent multiple tumours have their seat in some one tissue, and never appear elsewhere. On the other hand, secondary cancerous growths may appear in almost every tissue of the body at the same time.

Again, in most cases of death from cancer it is easy to recognise some one tumour as the starting-point of all the others. A skilled pathologist recognises this "primary growth" either by its anatomical character, or from what he knows of the origin and distribution of the particular kind of tumour. The anatomical difference consists not so much in its greater size as in its being of firmer consistence from slower growth; or an advanced state of degeneration, or extensive ulceration, may show its earlier date. Locality is another important criterion; for primary cancers, like almost all other diseases, do not arise at random through the body, but are common in certain regions, rare in others, and almost unheard of in the rest (*v. infra*, p. 95). As a matter of fact it is exceedingly rare to find in the same case two malignant tumours with equal claims to priority.*

It is remarkable that organs and tissues which are especially apt to be the seats of primary growths are seldom affected with secondary or "metastatic" growths of the same kind. For instance, a primary cancer is often developed in one of the breasts, but secondary growths avoid the breast; nor are they met with in the cervix uteri, another locality from which cancer frequently starts.

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* An old woman died in Guy's Hospital with cerebral symptoms, and I discovered three tumours in the brain or in its membranes. I felt convinced that they were secondary to a primary growth elsewhere. But I searched every part of the body in vain, until I noticed that the end of the rectum had not been taken out. This was found to contain a large ulcer with prominent fungating edges, which evidently was the lesion for which I was looking, although it had given rise to no symptoms during life. A microscopic examination of the secondary cerebral tumours showed that these were "columnar epitheliomata." Now, if I had been able to examine them previously, my task would have been very much more easy, for I should have known almost exactly where to look for the primary affection which gave rise to them.—C. H. F.

The most common seats of primary cancer are the lower lip, the tongue and pharynx, the œsophagus, stomach, colon, and rectum; the mamma, uterus, penis, and testes; and in these regions secondary cancers are almost unknown. On the other hand, the lymph-glands, liver, lungs, bones, and serous membranes, so frequently attacked by secondary cancer, are very rarely the starting-points of the disease. Nor is this a mere matter of structure, for the whole epithelial surface of the small intestine is all but exempt from cancer; and some glands, as the parotid, are equally so. With sarcomata no such rule holds.

In many cases the distribution of secondary nodules or tubera is so limited as clearly to indicate by what channel they were derived from the primary growth. For example, a malignant tumour in the stomach or in the intestine sometimes causes secondary nodules in no other organ but the liver, showing that something was carried by the blood of the portal vein, which served to propagate the disease; or the primary growth being in one of the limbs, the secondary growths may occur only in the lungs, so that the infection obviously took place through the systemic veins and the pulmonary artery. Malignant tumours often penetrate into the interior of veins; there is, therefore, no difficulty in understanding how nuclei, or cells, or even pieces of the growth, may get washed away by the blood-current, and carried to distant parts. Cancerous thrombi form soft colourless masses, parts of which are blended inseparably with the coats of the vessel, while other parts, often much elongated and lobulated, project into its channel, either perfectly smooth on the surface, or coated with a layer of fibrin.* Dr Moxon mentions in the 'Guy's Hospital Reports,' vol. xviii (3rd series), that he had twice seen cancer growing in clots which lay within the cavity of the right ventricle at its apex, brought, no doubt, by the blood of the systemic veins. In 1871 Dr Payne showed to the Pathological Society a heart in which both the right auricular appendix and the apex of the left ventricle contained similar cancerous thrombi projecting from between the muscular trabeculæ. In that instance the infecting agent must have traversed the pulmonary capillaries from the right side of the heart to the left.

Secondary tumours most often spread by lymphatics to the lymph-glands, less frequently by the veins to the lungs, or the liver. In some cases, however, their distribution is different from what we should have expected on anatomical grounds. Thus, in a case of epithelioma of the œsophagus, the lungs were found healthy, but a secondary nodule existed in one kidney; and in a woman with cancer of the breast, almost every bone may become cancerous, while the viscera remain free from the disease.

We ought to add, as an authentic although rare way in which malignant growths may multiply in the body, the possibility of portions of a tumour becoming *transplanted* when set free by ulceration. In 1868 Dr Moxon showed to the Pathological Society a specimen in which he thought that an epithelioma of the œsophagus extending into the trachea had in this way infected the lungs; secondary nodules were seated in the interior of the lower lobes, and not beneath the pleura, as is usually the case; they occupied the centres of lobules, and small tubes could be traced up to them. He there mentions that Mr Simon had exhibited specimens in which "cancer germs" had appeared to take root in the bladder after descending the ureter from the kidney. In a case observed by Dr Bristowe, a malignant growth of the interior of the skull had apparently infected the cord low down by fragments which had fallen; and in the abdomen we sometimes find a nodule just opposite to the primary tumour and in contact with it. So several cases are on record in which cancer has started in one labium of the vulva and been apparently transferred by contact to the other. In a case of cancer of the duodenum shown before the Pathological Society there were two ulcers which were so placed that one readily came in contact with the other at the bend of the gut ('Path. Trans.,' 1894, p. 63).

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* Some time ago I saw an instance in which, the primary growth being in the liver, and the hepatic veins extensively involved, the lower border of one lung contained a whitish-yellow wedge-shaped mass, exactly like an infarctus due to embolism from ordinary thrombosis; Dr Pye-Smith, however, under whose charge the case had been during life, found that it was really a secondary nodule of the tumour.—C. H. F.

This direct transfer must, however, take place under exceptional conditions, for such cases as those just cited are admittedly rare. The particles detached from a malignant growth are, as a rule, dead and already decayed, and no authentic case appears to have been recorded of cancer of the vagina or os uteri in a woman producing the same disease in her husband.

In some cases it has been possible to produce cancerous disease in an animal by "grafting" a portion of a similar growth in another animal of the same species. But it has never yet been possible to inoculate one of the lower animals with cancer from a human being or from a different animal, although the attempt has been frequently made.

Malignant behaviour and cancerous structure.—The above characters which Virchow laid down as distinguishing malignant from innocent new growths belong to other infective processes, such as septicæmia, pyæmia, and tubercle. These exhibit "local progression," and sometimes pay little more respect to the natural boundaries of the tissues than does cancer itself. They spread to lymphatic glands, and may undergo "generalisation" or "metastasis" through the blood. It is not, therefore, surprising that these malignant, invading, infective properties are not exclusively found in one anatomical kind of tumour. It has long been recognised that no absolute line of histological distinction can be drawn between innocent and malignant growths. Paget described in 1853, under the name of "*recurrent fibroid*," a growth characterised by an inveterate tendency to return *in situ* after removal, without infecting distant structures. Wilks, in his 'Lectures on Pathology,' published in 1859, made a separate group of "semi-malignant" tumours, including, besides the "recurrent fibroid" of soft structures, the "osteosarcoma" of bone; and he specially recorded instances in which "myeloid" or "enchondromatous" growths, which were then generally regarded as innocent, appeared metastatically in the lungs.

Varieties of cancer.—The older surgeons had recognised a great variety of cancerous or malignant growths "solanoid" (*i. e.* cutting firm like a raw potato), fungus hæmatodes (sprouting rapidly and bleeding readily), villous or cauliflower excrescences, white and soft like brain or marrow, and so on.

The result of microscopic investigation was to limit these varieties to the following:—Scirrhus, or hard cancer; Encephaloid, medullary, or soft cancer; Epithelioma, or horny cancer; Melanotic, or black cancer; Osteoid, or bony cancer; and Colloid, or gelatinous cancer. Virchow showed that most instances of "encephaloid cancer," "melanotic" and "osteoid cancer," really belonged to the *connective-tissue* series of growths, for which he revived the old name of Sarcoma. There are other soft tumours, however, which have the alveolar, glandular, epithelial character of true cancer, and these are now closely associated with the scirrhous tumours, the only difference being the density and comparative abundance of the fibrous network compared with the soft cellular contents of the acini. In essential character carcinoma agrees with growths of the tongue, lip, and skin, which used to be called epithelioma or cancroid, but are now recognised as the horny or ceratoid variety of carcinoma.

Classification.—No one has yet framed a completely satisfactory classification of tumours. The following arrangement, like all others now in use, is a modification of Virchow's. So-called cystic tumours are excluded. If merely dilated cavities containing fluid (retention-cysts), they are no more "tumours" in the modern sense than is a hydrocele, or an œsophageal pouch. If due to an animal parasite (hydatids) they are not neoplasma, but foreign bodies. If degenerations of solid tumours, as glioma, their place is with the structure of which they are the outcome.

I. Tumours formed on the type of connective tissue: mesoblastic.
  (α) Following fully developed connective tissues.
    1. *Fibroma*, or fibrous tumour, innocent.
    1. *Fibroma*, or fibrous tumour, innocent.
    3. *Glioma*, like neuroglia of brain, spinal cord, and retina.
    4. *Chondroma*, or cartilaginous tumour, sometimes malignant.
    5. *Osteoma*, or bony tumour.
    6. *Odontoma*, or ivory tumour.
    7. *Lymphoma*, or lymphadenoma.
  (β) Following embryonic connective tissues.
    8. *Myxoma*, resembling the tissue of the umbilical cord, or the vitreous body—nasal and aural polypi;* subcuticular and intermuscular tissue; uterine moles. Sometimes recurrent.
  (γ) 9. *Sarcoma*, resembling undifferentiated mesoblastic tissue, recurrent and more or less malignant.
    10. *Compound growths, as Fibro-lipoma, Glio-sarcoma, Myxo-sarcoma.*
II. Of epithelial type: epiblastic and hypoblastic.
    1. *Papilloma*, and other usually innocent growths of skin and mucous membrane.
    2. *Adenoma*, or innocent glandular tumour.
    3. *Carcinoma*, or cancer: glandular, alveolar—malignant.
III. Of the type of higher tissues.
    1. *Myoma*, or muscular tumour: rhabdomyoma, leiomyoma.
    2. *Neuroma*, or nerve tumour.
    3. *Angioma*, or vascular tumour.
    4. *Lymphangioma*, or lymphatic tumour.
IV. *Teratoma*, or embryonic tumour, ranging from dermoid cysts to complete parasitic organisms in the ovary or on the surface.

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* See Jacobson, 'Guy's Hosp. Rep.,' vol. xli, p. 217.

Many of these forms of tumour have surgical rather than medical interest, and do not need more than mention; but others which affect numerous internal organs will be most conveniently treated here.

I. Mesoblastic Tumours.—1. *Fibroma.*—This is made up of fibrous tissue, either arranged in loose meshes or tightly felted together. In the former case its substance is soft and succulent; in the latter it may be exceedingly tough, so as to creak when cut through.

True fibrous tumours are far from common. The most important are the multiple fibromata of nerve-sheaths, often called *neuroma.* Similar round or oblong small fibrous nodules occur in tendons and periosteum, particularly in children after rheumatism. The remarkable multiple pedunculated fibrous growths, which are sometimes found covering the skin in immense number, will be described with other cutaneous tumours, as fibroma molluscum.

Similar growths of the mucosa of the intestine, uterus, or bladder are usually pedunculated, and are called fibrous *polypi.* Fibroma of the breast or ovary is a very rare disease. It is not uncommon for several small fibrous growths to be found in the kidneys, which appear as minute soft white nodules, and are apt to be taken for secondary tumours when there is a growth in some part, or for tubercles.

Fibrous tumours are small, and very rarely, if ever, recur after removal. Paget's recurrent fibrous tumour is now recognised as a spindle-celled sarcoma. English writers separate, under the name of *Myoma,* a common variety of tumour containing much fibrous tissue, on account of the presence in it of more or less numerous bands of smooth muscle; but most German pathologists replace these growths, which are seen in the uterus and in the prostate, among the fibromata. Some small "fibroids," however, have certainly rod-shaped nuclei and other characters of muscle. Dr Fagge described a myoma from the œsophagus ('Path. Trans.,' vol. xxvi, p. 94).

There are true myomata (*rhabdomyomata*), or tumours of striated muscle, sometimes found in the kidney and elsewhere. They are of purely pathological interest.

2. *Lipoma.*—This is a tumour consisting of adipose tissue like the subcutaneous fat, but circumscribed, and generally enclosed in a well-marked capsule. It is the largest of all innocent growths, often multiple, and common on the surface of the body. When they occur in the interior of the body fatty tumours do not produce symptoms so as to come under medical observation, unless they are so large as to press upon important organs, which is very seldom the case. But Dr Frederick Taylor ('Path. Trans.,' vol. xxvii) met with a case in which a lipoma grew in the post-pharyngeal space of a child, and caused death by suffocation. In the abdomen such growths have occasionally reached an immense size, and have been mistaken for ovarian tumours.

3. The modified connective tissue of the central nervous system, called neuroglia by Virchow, gives rise to a characteristic kind of new growth confined to the brain and (as a rare event) to the cord and the retina, and named *Glioma.* It will be described as the most frequent among "simple" tumours of the brain.

4. *Enchondroma.*—Tumours made up of cartilage were so named by Johannes Müller. Histologically they differ widely in different cases; the matrix may be either hyaline or fibrous, and the cells may present various

characters. They usually begin in bone, occasionally in the testis or parotid, and are sometimes malignant in their course. They scarcely come under the notice of the physician. Virchow describes multiple chondromata of the lungs, situated chiefly near their roots, and probably bearing some relation to the cartilages of the bronchia, found accidentally in the *post-mortem* room. The cartilaginous tumours, which are not infrequently found in the testis and parotid gland, are mixed tumours best described as chondrosarcoma.

5. *Osteoma.*—This is the technical name for growths which consist of osseous tissue, but it is not often used. For when a tumour projects outwards from a bone it is called an *exostosis*, and when it extends inwards (as into a frontal sinus) it is sometimes called an *endostosis*. Most other bony growths contain soft structures as well—osteochondroma, osteosarcoma.

A remarkable kind of growth, to which the name *Malignant Osteoid Tumour* has been appropriated, comes under the notice of the physician rather than that of the surgeon. By Paget it was called "Osteoid Cancer." Virchow's term, "Osteoid chondroma," was unfortunate, since (as he himself pointed out) the structure of the non-calcified part of the growth is not that of cartilage but of periosteum. Moreover, confusion has arisen from its being supposed to be identical with an "ossifying enchondroma," which is a tumour really consisting of cartilage, the deeper and older parts of which have undergone conversion into bone. Wilks and Moxon term this growth "Periosteoma," and define it as representing the varied tissues of ossifying bone.

These malignant osteoid tumours are made up of an exceedingly dense, firm, and tough material, which is of a pale greyish colour, and cannot be teased out. Under the microscope it may appear more or less distinctly fibrous. Paget describes the fibres as "crisp and stiff," or as moderately broad, "with uneven thorny edges," and arranged in bundles, "looking like fagots." Embedded in this substance are cells, which are rather small, of round or oval shape, without alveoli. When calcification takes place, earthy salts are deposited in hard granular masses, but in some parts more or less perfectly formed bone may be seen. The calcified growth has a peculiar dull white aspect like chalk or mortar. It cannot be cut, and must be sawn through, but it may be "rubbed or scraped into a fine dry powder."

Malignant osteoid tumours are most frequent in young subjects. Of nineteen cases collected by Paget, five occurred in persons between ten and twenty years old, nine in those who were between twenty and thirty, and none over fifty. They are generally attended with severe pain. Their favourite seat is the lower end of the femur. One in Guy's Hospital affected the humerus close to the shoulder. This kind of growth forms a very large, hard mass, surrounding the whole circumference of the bone, slightly if at all nodulated on the surface, and gradually sinking down to the level of the rest of the shaft. Thus it might seem to be outside the bone, but on section the medullary cavity and the cancellous tissue are found to be completely occupied by the opaque, hard substance above described. When a flat bone is attacked, the growth is said by Paget always to project from both surfaces. We had a remarkable case in which the two iliac bones were affected symmetrically, each having a large bossy prominence projecting from both the dorsum and the venter, which might have been felt during life. There may be many such tumours growing from different bones in the same patient, and the clinical aspect of the case may be that of a cerebral tumour or of paraplegia from compression of the cord. Or, again, the symptoms may be thoracic, from an immense mass lying at the root of the lungs, or from numerous nodules scattered in their substance and beneath the pleuræ. The secondary growths in lymph-glands and viscera look like those of the bones. They may be calcified until only a narrow white, fibrous-looking substance remains round their edges.

6. *New growths of the type of lymph-glands.*—Among the primary forms of tumour, in Virchow's classification, is one to which he gave the name of *lymphoma*, and of which he defined the structure as being identical with that of a lymphatic gland or of a solitary follicle: the leucocytogenic or "adenoid tissue of His." It is made up of cells, which lie in the meshes of a stroma. The cells are leucocytes, and can be more or less readily removed by pencilling a thin section of the growth. The stroma, which then becomes visible, is characterised by being "reticulated;" that is to say, its fibres branch and unite together, many of the points of union being occupied by nucleated cells which are comparable with fixed connective-tissue corpuscles, triangular or stellate in form.

Some pathologists still question whether the enlarged glands of leuchæmia are not the result of the leucocytes of which the blood is full being deposited in the interstices of the tissues. By many French observers, including Cornil and Ranvier, the term "lymphadenoma" is used to include mycosis fungoides, which is by most pathologists regarded as a granuloma, and by some as a sarcoma. In the malady known as anæmia lymphatica or "Hodgkin's disease" growths spring up in immense numbers, and sometimes with extreme rapidity, not only in the lymphatic glands and the spleen, but in almost every organ and tissue; and they spread from one part to another without regard to natural boundary lines. Their histological characters vary so much that we must regard "Hodgkin's disease" as a clinical rather than a pathological name. The difficulty is to distinguish the new growth lymphoma from (1) irritated or inflamed lymph-glands, (2) hypertrophic lymph-glands, (3) tuberculous lymph-glands, and (4) small round-celled sarcoma of the lymph-glands.

The term "lymphosarcoma," like lymphadenoma, has been employed in different senses by different writers. Some seem to apply it to any sarcomatous growth having its starting point in lymphatic glands; while Virchow used lymphosarcoma for lymphoma which does not caseate, and which undergoes rapid enlargement; he would include under this head most instances of Hodgkin's disease, and all the lymphomatous tumours of the mediastinum. But it is certain that many of the cases in question, even where a large number of organs are involved, exhibit no histological characters other than those of a pure lymphoma. The best course seems to be to keep to the anatomical meaning of anatomical terms without reference to clinical events; and to reserve the name of lymphosarcoma for such growths as appear to be transitional between lymphoma and sarcoma. Lymphoma will then denote a tumour composed of normal lymphatic (adenoid or cytogenic) tissue.

7. *Myxoma.*—The true relations of this form of tumour were first recognised by Virchow. Older synonyms for it are "collonema," "sarcoma gelatinosum," and "fibro-cellular tumour" (Paget), and it was probably often confounded with colloid cancer. It consists of a semi-translucent material, sometimes so soft as to quiver like a jelly, of a bluish or yellowish tint, and emitting a sticky fluid when squeezed or scraped. This

from the intercellular stroma, and owes its viscidity to the presence of mucin. The addition of acetic acid to a thin slice of the growth renders it white and opaque from precipitation of the mucin.

The cells are generally scattered at considerable intervals. They may be round, but they are often stellate, with long processes ramifying in the stroma. Virchow showed that this structure is identical with that of the gelatinous tissue which in earlier fœtal life occupies the place of the subcutaneous fat, which at birth is found in the umbilical cord, and which constitutes the vitreous body of the eye. Myxomata are sometimes found in large numbers on the nerve-trunks. Myxo-lipoma and myxo-chondroma are met with. Some polypi of mucous membranes are fibro-myxomatous in structure, and also the so-called "hydatid moles" of the uterus.

8. *Sarcoma.*—Virchow referred to the *connective-tissue* series of growths certain malignant tumours, especially those formerly known as "encephaloid" and "melanotic" cancer. He grouped them with other tumours of innocent character, or which at most exhibit a tendency to return *in loco* after extirpation, and he adopted for them all the name of sarcoma. The word Sarcoma, although it dates back to Galen,[*] and had been employed by subsequent writers down to the time of Abernethy, had fortunately fallen into disuse.

The cells of a sarcoma may be of three kinds:

a. Round-cells.—These vary in character in different cases. Sometimes they are undistinguishable from leucocytes. Sometimes they are much larger, and they are then often exceedingly delicate, so that the addition of water to a microscopical specimen may cause the disappearance of all but the nuclei, which then seem to be free.

b. Spindle-cells.—These occur in the growths which by French writers had been called *fibro-plastic*, and which had in England been recognised by Paget and others as "Recurrent Fibroid." They have an elongated shape, tapering gradually into a point at each end, or having their ends prolonged as delicate sinuous fibres. Their nuclei are oval, fusiform, or oat-shaped.

c. Giant-cells.—Under this name are now described certain bodies, which, however, are rather irregular masses of protoplasm than cells, and which contain round or oval nuclei to the number of ten, twenty, thirty, or even more. From similar elements being found in the medulla of bones, especially during fœtal life, as Kölliker and Robin discovered, they were termed "myeloid cells," or by French writers *myéloplaxes.* The growths in which they occur, and which are comparatively infrequent, were by Paget and Wilks described as a special kind of tumour. Virchow, however, showed that such growths always contain spindle-cells as well, and therefore placed them among sarcomata.

Beside these different kinds of cells, a sarcoma also contains blood-vessels, which are sometimes so large and receive so abundant a supply of arterial blood that it may pulsate; indeed, a pulsating sarcoma has been mistaken for an aneurysm. The walls of the smaller branches of these vessels usually consist of spindle-cells applied to one another in such a way as to enclose a blood-channel between them. It is, therefore, not surprising that they may allow blood to escape into the substance of the growth, so that on section the mass looks like a mere clot. Such tumours increase in size with extraordinary rapidity; and they were formerly known as *fungus hæmatodes*, a name now almost forgotten. In some cases there are so few cells between the vessels that there may be great difficulty in recognising any new growth.*

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[*] *Σάρκωμα ἐστὶ σαρκὸς ἐν τοῖς μυκτῆρσι παρὰ φύσιν αὔξησις, ἐστὶ δὲ σάρκωμά τι καὶ ὁ πολύπους.* (Sarcoma is an unnatural fleshy growth in the nostrils, and a polypus is also a kind of sarcoma.)—'Galeni de Deform. medendis.'

There appear to be few lymphatic vessels in sarcoma, but the cells of a sarcoma are always surrounded by a stroma of connective tissue.

The fact that fibres and blood-vessels run between the sarcoma-cells is characteristic, and distinguishes this group of malignant tumours from those formerly confounded with them under the common name of cancer. Occasionally, however, as Billroth first observed, there is a regular alveolar structure, like that of carcinoma; the cells are, however, smaller, uniform, and not like epithelium, and the stroma is less abundant and firm; moreover the alveoli do not, it would seem, form branching tubular or glandiform passages. Since we have believed that true cancers can arise only from epithelium, *alveolar sarcoma* has acquired importance, from enabling pathologists to account for apparent exceptions to this rule.

Still greater complexity is caused by the combination, in the same tumour, of different structures belonging to the connective-tissue series, such as cartilage, mucous tissue, bone or fat. Perhaps the occurrence of certain rare cases in which a growth, ordinarily innocent, sets up secondary tumours in distant organs may be explained by the presence in it of more or less numerous embryonic cells, the diffusion of which by the blood-stream causes the infection. According to this view all such tumours are "mixed," and should be designated accordingly.†

Another cause which modifies the appearance of sarcoma is the occurrence of degenerative changes. Of these caseation is the most common, but calcification and myxomatous changes also occur.

To the naked eye sarcomatous tumours vary greatly in consistence and in colour; but there is no constant relation between these characters and their histological structure. Still, the softest and whitest among them —those which would be termed "encephaloid" or "medullary"—consist rather of round- than of spindle-cells. True cancers never have so homogeneous, white, glistening an appearance as is shown by many sarcomata; and the only cases in which they are equally soft are those in which they grow into the interior of a free space, as into the enlarged pelvis of a kidney, or into the channel of a vein, where they meet with scarcely any resistance. The giant-celled sarcomata are often recognisable without difficulty at the first glance; they are blotched, or uniformly tinted with a dull pink, contrasting here and there with a greyish hue.

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* In 1877 I made an autopsy in the case of a lad aged fifteen, who had been under Mr Cooper Forster's care for a fracture of the femur, which seemed clearly to have been caused by external violence. Repair did not take place, an incision led merely to the escape of blood; and amputation was followed by the death of the patient. I found the injured bones and muscles soaked in blood, and at first I had no suspicion of there being any tumour elements. But presently I noticed that the blood-stained appearance of the muscles was limited definitely by convex margins; and the microscope showed masses of delicate spindle-cell tissue in them. In the lungs there were five or six scattered secondary nodules, the size of marbles, and reddish in colour, projecting above the level of the pleural surface. These, however, felt quite soft; and when cut into, each of them collapsed, leaving a cavity surrounded only by a very narrow margin of indefinite-looking tissue. For further details of this case see 'Guy's Hospital Reports' for 1880, p. 17.—C. H. F.

† In a case at Guy's Hospital fibromata of the uterus led to the formation in the lungs of similar growths in which the unstriped muscular fibres were as conspicuous as in the uterine tumours themselves. And in another case, that of an infant fifteen months old at the time of death, there were in the liver, secondary to a mixed sarcoma of the neck, a number of dense flat button-like nodules, which consisted almost entirely of well-developed

Distribution.—The primary seat of a sarcoma gives a good presumption of its histological character. A growth which starts in the submucous tissue of the alimentary canal, or in one of the solid viscera, is not likely to be made up of spindle-cells. The fasciculated sarcomata belong to the periosteum and to the fasciæ. Giant-celled sarcomata arise usually in the cancellous substance of bones, and spread out the compact tissue over them. They also often appear in the form of *epulis*, a growth projecting into the mouth from the alveolar processes of the jaws: but some of these tumours are purely fibrous.

Round-celled sarcoma may occur in any connective tissue, but is most common in bone, intermuscular septa, corium, the testis and ovary, the kidney in children, and the central nervous system—the brain, spinal cord, and retina. Spindle-celled sarcoma grows from periosteum and fasciæ, submucous tissues and other fibrous structures, and it also occurs as a mammary tumour. Alveolar sarcoma has been met with in the skin.

Sarcoma is most common in children and young adults, and is sometimes congenital; "cancer" in early life is almost always malignant sarcoma. They rarely occur as primary growths in viscera or on mucous surfaces, but are frequent in bones, in joints, and in lymph-glands. Secondary growths appear to follow the distribution of the veins, not of the lymphatics.

Malignancy of sarcomata.—Among the forms of sarcoma, the "degree of malignancy," that is to say, the likelihood of recurrence after removal and of infection of distant organs, may be thus expressed. The least malignant is the giant-celled or myeloid sarcoma, then the glio-sarcoma of the retina, then cartilaginous, then spindle-celled sarcoma, then the melanotic form, to be presently noticed, and lastly the round-celled sarcoma, which is malignant as the worst cancers. In fact, the sarcomatous growths of this variety, which one meets with in the bones, kidneys, and intermuscular spaces in infants and young children, are more vascular, grow more rapidly, and are more speedily fatal than any carcinoma in an adult.

The recurrence of a sarcoma after removal is probably chiefly due to the absence of any fibrous capsule. They never "shell out," and the tissues around are infected with cells (or "germs" or "virus") far beyond the apparent limits of the growth.

The fact that sarcoma infects by the blood and not the lymph is no doubt due to its vascularity and the absence of lymphatics. Lymph-glands are rarely affected by secondary sarcoma. The lungs are its most common seat.

Melanotic sarcoma.—This constitutes the larger part of what was formerly known as "Melanotic Cancer." Rindfleisch suspected that even when a pigmented growth possesses an alveolar structure it is yet likely to be a sarcoma of Billroth's "alveolar" form. But most pathologists allow that true carcinomata are sometimes melanotic.*

As a rule, melanotic sarcoma starts from some part of the body which normally contains pigment, generally from the skin or from the choroid coat of the eye, and usually in the later period of life.

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* Dr Fagge examined one specimen (from the skin of the neck) in which the cells lining the walls of the alveoli were distinctly columnar in form, and arranged side by side so that their epithelial character could not be doubted.

A curious fact is that the rectum sometimes becomes the seat of a primary melanotic tumour. Grey or white horses, which have a deficiency of cutaneous pigment, are extremely liable to be affected with a pigmented growth situated about the anus or upon the tail.

When the melano-sarcoma starts in the skin, it usually begins in a pigmented mole; and the histological type is most often that of alveolar sarcoma, not true carcinoma. The appearance of numerous pigmented moles is not uncommon in elderly people, and is often a warning that malignant disease may be developing internally. The matrix of the nails is another occasional seat of primary melano-sarcoma.

It is to be observed that the distribution of pigment in melanotic tumours is exceedingly capricious. Whole nodules may be uniformly black as ink, but perhaps others in the same case are white and medullary, and yet others may be of a grey colour, or streaked or marked with black patches and lines. Where there is no alveolar structure, melanotic sarcomata usually belong to the spindle-celled variety. In many instances they are extremely numerous, and they may be thickly scattered in regions and tissues which are seldom affected by new growths. Thus they may be found along the course of the intestine, with ulcerated surfaces projecting into its interior. Others may be found in the spleen, in the thyroid, or in the muscular substance of the heart.

Mixed with the tumours and nodules there are sometimes black spots or patches, which look as though they were mere deposits of pigment without any new growth; for example, in the mucous membrane of the stomach, of the renal pelvis, and of the bladder. Lücke, indeed, says that a careful examination always reveals the presence of cells in spots thus affected; but Thiersch relates a case of melanotic carcinoma of the skin in which at certain points nothing could be discovered but a diffused brown staining of papillæ, the texture of which was perfectly normal.

From a medical point of view it is important that pigment is often excreted in the urine in large quantities, when melanotic growths are present in other organs than the kidneys or bladder.* Urine so affected may have an olive-green hue when voided; it becomes darker on exposure to the air, and the addition of nitric acid turns it a deep black. It may throw down a deposit consisting partly of minute granules, partly of rounded brown translucent bodies resembling nuclei, partly of casts of the uriniferous tubules, brown or black with pigment. The colouring matter, which is known as *melanin*, can be separated as a powder, and when suspended in water it remains unchanged for years. Certain of the tumours may be colourless, even in cases attended with melanuria. It therefore seems clear that the pigment is originally manufactured in the substance of the growths, and in such large quantity that some is reabsorbed and excreted by the kidneys. It is probably derived from hæmatin, but no orange or reddish-brown transition tints have been observed. The clinical recognition of melanuria may sometimes be of help in the diagnosis of an obscure internal tumour; but it must not be confounded with the blackening of the urine which may appear when carbolic acid, creasote, or tar in any form has been applied to the skin. A still more rare symptom of internal melanotic sarcoma is pigmentation of the skin. Dr Wickham Legg published a case of this in 1884 ('Path. Trans.,' vol. xxxv, p. 367).

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* Two instances were brought by Dr Fagge before the Pathological Society in 1876, and it had previously been described by Eiselt and by other foreign writers.

The primary growth was here a spindle-celled melanotic sarcoma of the choroid.

Multiple sarcoma.—Sarcomatous growths in the interior of the body are sometimes accompanied by ambiguous symptoms. In certain instances the chief complaint of the patient is pain, which may either be fixed in certain parts or widely diffused, and may vary in seat from time to time. Advancing emaciation and anæmia complete the clinical features of the disease, the nature of which often remains obscure to the last. The following cases are examples.

In 1880, a man, aged forty-six, was admitted into Guy's Hospital for what was regarded as a gouty affection of the right hand and of the left great toe. He complained of pains in his right arm, in both shoulders, in the loins, and (especially during defæcation) in the lower part of the spine. He also suffered severely from headache. The urine was at one period albuminous, and the case was therefore regarded as one of Bright's disease of gouty origin. Subsequently the urine became normal, and some of those who saw the patient at that time suspected that he was exaggerating his symptoms. However, he grew more and more wasted and bloodless, and his pains became fixed in the iliac fossæ, especially on the right side. The most careful search was made for tumours, which it seemed could hardly escape detection in so emaciated a subject. A few weeks before death hæmaturia occurred, and this of course led to the suspicion of primary malignant disease of the kidney. At last he became unconscious, with rigid flexion of the right arm, and in this state he died. The autopsy showed that sarcomatous growths had occurred to an enormous extent, but that there was nowhere any tumour discoverable by manipulation. In each iliac fossa a layer of sarcomatous tissue was spread out beneath the periosteum, and there was a large quantity diffused over the surface and in the interior of many other bones. One tumour, which was removed, and sawn vertically through, had in its cancellous tissue many rounded masses as large as walnuts. On the under surface of the dura mater there were a number of button-like nodules indenting the brain. One kidney had two small tumours affecting the mucous lining of its pelvis. The growth consisted of large cells of the most irregular form embedded in a fibrous matrix.

Another case is that of a boy, aged sixteen, who came under the writer's observation when clinical clerk to Dr Owen Rees in 1854. He said that he had been strong and robust until eleven weeks before his admission, when he began to suffer from pain, at first in the loins, and afterwards in the shoulders and limbs. There was great tenderness of the whole surface of the body. He had also experienced a sensation of tingling in the area of distribution of the ulnar nerve to each hand. He was a very delicate-looking lad, with a clear complexion, a pink flush on each cheek, a moist skin, a white tongue, and a pulse of 144. The first diagnosis was of subacute rheumatism; subsequently of chronic inflammation of the spinal membranes. He lay for about two months, during which time he became emaciated to the most extreme degree. Towards the last, masses of enlarged glands could be felt through the walls of his contracted abdomen. He continued to suffer excruciating pain on the slightest movement, and there was exquisite tenderness of the skin. One day he became insensible, and had a convulsive attack, after which he lived only three hours. A large mass of medullary sarcoma was found in front of the spine, eroding the vertebræ, and involving the nerves as they emerged from the spinal canal.

Sometimes the growth of multiple sarcomata leads to a still more rapidly fatal illness. In vol. xxv of the 'Guy's Hospital Reports' are recorded certain cases which were attended with purpura and other hæmorrhages from mucous surfaces. One of the most curious is that of a man, aged twenty-five, who was admitted in 1879 under the present writer for a supposed attack of rheumatism. On Whit Monday, June 2nd, he had got wet through; and from that time up to his admission, two months later, he had complained of pains in the left shoulder, in the chest, and in the hips. He had been confined to bed for nine days with profuse sweating; the temperature varied from 102° to 102·3°. The skin had an unpleasant sour smell, and a systolic apex murmur was detected on one occasion when he sat up. He went on well for twelve days, when a purpuric rash came out on the chest and the abdomen, hæmaturia occurred, and his eyelids and his scrotum became greatly swollen. Five days later, on August 14th, he died. At the autopsy the scattered spots of purpura still remained visible. Some of them were flat, but others were slightly raised and indurated, and a few had a central pale elevation with a narrow ring of purple discoloration around it. These proved to consist of small round or irregular cells infiltrating the little lobules of subcutaneous fat. In the kidneys there were several white or pinkish sarcomatous nodules. In the cæcum and adjacent part of the ileum there was what appeared to be the primary growth, a homogeneous-looking yellow mass, of considerable thickness, involving all the intestinal coats.

A second case occurred in a patient, aged thirty-eight, under Dr Moxon in 1877. Up to five weeks before admission he had always, he said, been a strong man. He then "caught cold from being exposed to draughts." After this he suffered from pains in the back, legs, arms, and chest, chiefly in the joints, and flying from one joint to another. Five days before he came into hospital he was attacked with severe pain in the back, hæmaturia set in, and purpuric spots came out on the neck, the groins, and the legs. Afterwards he had epistaxis, his gums became sore and bled. His temperature had been taken before admission, and was found to range from 100° upwards. On admission he was much blanched and extremely weak, with no perceptible pulse. The nose began to bleed almost immediately, and on the following morning he died. The kidneys were found to contain a large number of white tumours, and there was in the right vesicula seminalis a firm growth, to which they were apparently secondary.

A third case was that of a man, aged twenty-eight, admitted under the care of Sir Samuel Wilks in 1872. He said that for some weeks he had suffered from headache and neuralgia, that his teeth had been loose and painful, and that his gums had been spongy and had bled. For ten days he had been suffering with severe pains in the elbows, the knees, and the shoulders, but these seemed to be diminishing in intensity. He was an anæmic man, with a brown tongue, foul breath, and teeth and gums caked with dry blood. His pulse was 128, his temperature 100·2°, his respirations 28. His gums and his nose continued to bleed, and eight days after his admission purpuric spots appeared on the abdomen. On the following day he died. A whitish firm growth was found in the anterior mediastinum, probably affecting the thymus; and there was a large quantity of a similar material in the subserous tissue of the peritoneum and in the mesentery.

A fourth case, somewhat like the others, occurred in February, 1876. The patient, a bank clerk, aged twenty-five, said that he had got very cold about a fortnight before Christmas, 1875; for three days he was chilled through, and he was never well afterwards. His temperature was 101°. His mouth and his tongue were stained with blood, his gums were slightly spongy, and he had purpuric spots on the legs. It appeared that he was not in the habit of eating any vegetables, and the disease was supposed to be scorbutous. But he rapidly grew extremely anæmic and feeble, hæmorrhage from the bowels set in, the lymph-glands in various regions of the body became enormously large, and at the end of a few months he died. There was no autopsy.—C. H. F.

Different views may be taken as to the relation between the symptoms which presented themselves in these various cases, and the underlying malignant disease. It may be that the purpura, the spongy state of the gums, the epistaxis, the fever, and the rheumatoid pains are mere results of a profound alteration of the blood, analogous to that which exists in Idiopathic Anæmia, in splenic Leuchæmia, or in Scorbutus. Another possible explanation of the purpura may be that a minute development of sarcomatous tissue, with vessels made up of embryonic cells, occurs at each spot which is the seat of an effusion of blood; or, perhaps, that sarcomatous cells become adherent to the capillary walls, and produce softening.

In other instances, somewhat less obscure at the bedside, the diffused development of sarcomatous growths is indicated by other symptoms. One is the formation of nodules in and beneath the skin, which can be felt and seen.[*]

In 1876, Dr Moxon had a patient under his care in whom the chief symptom was wasting until sarcomatous nodules appeared in and beneath the skin of the chest and of the limbs, and cleared up the diagnosis.

Another symptom of the formation of multiple sarcomata, especially when they affect the skeleton, is a gradual enlargement of certain bones. In one of the above cases the calvaria was found to be very thick, and its whole substance was homogeneous and opaque; the bodies of the vertebræ, the ribs, and one side of the pelvis were also found to have their cancellous tissue converted into a dense material. Similar changes were observed in two instances recorded by Dr Goodhart in vol. xxix of the 'Pathological Transactions;' and Dr Cayley relates in the same volume the case of a man who came under treatment for chronic enlargement of the lower jaw; his left clavicle was also much thickened, and both tibiæ curved forwards. This patient died of malignant disease of the lung and of the liver. The most remarkable instances are those which were brought by Sir James Paget before the Royal Medical and Chirurgical Society in 1876, under the name of "Osteitis deformans." Here diffused osseous change seemed to precede the development of any local tumour by many years.

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[*] Such nodules, indeed, were present in the last case of the bank clerk, but were supposed to be *molluscum fibrosum*. It would be possible to guard against a similar error in the future by remembering the peculiar discoid shape of molluscous growths, which generally have a flat under and a convex upper surface. Another case was believed to be sarcomata, until at the autopsy the subcutaneous tumours were proved to be cysticerci. Their real nature might, however, have been recognised, if one had thought of it, by their peculiar shape and size; they were firm elliptical bodies, very like a French olive in outline, but somewhat smaller.—C. H. F.

We may compare this non-malignant overgrowth of bones and the ultimate development of malignant tumours, with the white patches of the tongue which terminate in cancer, or with the freckles and vascular changes in the skin which, after a long course, end in malignant growth, as described by Kaposi. Pathologically we must regard the innocent and malignant stages of each disease as distinct. But from a clinical point of view, it is important to keep before one the significance of a thickened tibia or clavicle in an obscure case, that may possibly be one of sarcomatous growth; and it may be important in diagnosis that a patient's hat has become too tight for him, or that his chin is projecting, his arms drooping, and his legs bowed outwards.*

II. New Growths of Epithelial Type.—As already remarked, we owe to Virchow the important doctrine that the "cancer-cell," which was generally believed to be heterologous, *i. e.* unlike any of the elements of the healthy body, is really identical with the cells of epithelium. He explicitly pointed out this fact in a paper in the first volume of his 'Archiv,' published in 1847; and at the same time he indicated the region in which the most perfect representatives of cancer-cells are to be found, namely, upon the mucous surface of the ureters and of the bladder, especially in infants.

Unfortunately, Virchow did not carry out his usual method of nomenclature, and stamp with his authority the practice of applying the term "epithelioma" to all tumours of epithelial structure. The word had been previously and is still often used to denote the variety of cancer called ceratoid (*v. infra*, p. 93). At present "Epithelioma" is very generally employed, in the wider sense which naturally belongs to it.

Epithelial growths, however, are not all cancerous. There are certain innocent forms which bear the names *papilloma* and *adenoma*. These bear the same relation to one another which an intestinal villus does to a Lieberkühn's tubule. The former consists of a solid protrusion outwards of mucous membrane or of skin, upon which epithelial cells are arranged; the latter is a hollow inversion of mucous membrane or of skin, with an epithelial lining.

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* The occurrence of many sarcomata in the same patient does not appear to be infective—they are multiple rather than malignant. Not only is it often impossible to discover any one growth which can be regarded as standing towards the rest in the relation of primary to secondary; but the tumours themselves may fail to present Virchow's first indication of malignancy, that of "local progression" without respect to the natural configuration of the parts affected. For instance, a sarcoma of an undescended testis, weighing eight pounds, had on its outer side a detached piece, bearing the very same relation to it in position and in size which the epididymis has to the healthy organ. In another case one ovary, although of normal shape, was of about twice the length of the opposite ovary, and its substance was opaque, white, and made up of rounded sarcomatous cells. It is perhaps worthy of notice that in young adults who die of sarcomatous growths the thymus is often found to be persistent, if not rejuvenescent.—C. H. F.

1. *Papilloma.*—Of this we have examples in common cutaneous warts, and in the horny growths which are sometimes seen upon the face, neck, or chest. Another common variety occurs as warts on the genital organs, and a fourth as condylomata and mucous patches; these will be mentioned among diseases of the skin. Yet another form is sometimes met with in the larynx, especially at an early period of life. Lastly, there is an affection to which the vesical mucous membrane is liable, and which is known as "villous tumour" of the bladder. It consists in the formation of long, soft, branched filaments, each containing a vascular loop, and covered with epithelial cells. This kind of new growth does not spread to lymph-glands, nor produce secondary nodules in distant parts; but in some cases the resemblance to the structure of a cancer is very close.

2. *Adenoma.*—Of this form of tumour (which resembles true gland tissue, not that of a lymph-gland) we find examples in both the sebaceous and the sudoriparous glands. Upon mucous membranes the most common adenoma appears as "polypi," growing from the intestine, the cervix uteri, or the nasal fossa. In the breast adenoma or "adenocele," the "chronic mammary tumour" of Sir Astley Cooper, very frequently occurs. They are also met with as encapsuled small tumours in the liver.

3. *Carcinoma or malignant epithelioma.*—Carcinoma often combines the anatomy of a papilloma with that of an adenoma. In the skin it may begin in a growth resembling a wart; many cancers of the stomach, of the colon, and of the gall-bladder present abundant villous processes; and in the os uteri, one of the commonest forms of cancer combines a glandular deep structure with a superficial papillomatous or "cauliflower growth."

a. Ordinary carcinoma (alveolar cancer; glandular cancer; cylinder-epithelial cancer; malignant adenoma).—The innocent adenoma consists of tubules or acini or ducts, precisely like those of the gland in which it arises, except that there is no secretion formed, and no channel for its escape if it were. Malignant adenoma consists of a more or less developed and more or less vascular fibrous stroma, provided with lymphatics as well as blood-vessels, and of large nucleated cells of various forms, but all of the epithelial type as distinguished from the cells of connective tissue and the leucocytes of lymph blood and pleuro-peritoneal cavities. These epithelial cells are contained in "alveoli," as seen in transverse sections, which, however, when the tissue is reconstructed by sections in various directions are found to be not closed cavities filled with irregular cells, but tubules and acini lined with them—in fact, glandular tissue. But the cells are not so regular in shape and are not so regularly arranged as in a normal gland or an innocent adenoma.

In soft, rapidly growing cancers, the interalveolar stroma is occasionally found to consist of spindle-cells. Otherwise the softness or hardness of a carcinoma depends chiefly upon the proportion between the amount of stroma and the size of the alveoli. In many instances the substance of the growth is as easy to break down as that of a healthy spleen, or even of a spleen from a case of fever; and then one finds that the alveoli are large and that their walls are made up mainly of blood-vessels, with but little fibrous tissue. This is the "soft," "medullary," or "encephaloid" cancer of the older pathologists. Sometimes it is so hard as to be spoken of as "stony;" the microscope then shows that it consists almost entirely of interlacing dense white bands, the alveoli being very small, and perhaps so few in number that their presence may easily be overlooked. It is to such growths that the old name of "scirrhus" was applied. They are commonly slow in their course; and they contract and shrink so much that their general appearance is often that of a cicatricial relic, rather than of a tumour: and if they occupy the wall of a canal (such as the intestine or the common bile-duct) they are apt to narrow it by forming a dense ring.

There are, however, all gradations between hard and soft cancer, and it is not well to make much of what is, after all, no essential distinction. On the whole, the soft, vascular cancers are more acute in their course. They are most frequent in the stomach as primary and in the liver as secondary growths. As a rule, the hard fibrous cancers grow more slowly, reproduce less abundantly, and are less unlikely to shrink and shrivel. They are more common as primary growths in the breast and as secondary growths in the lymph-glands. The stomach, large intestine, and uterus offer examples of both forms. The hard contracting form of scirrhus (*cancer atrophicus*) sometimes, though unhappily very rarely, crumbles away without ulceration, and at last undergoes spontaneous involution, leaving a dense puckered mass of scar-tissue. The writer once saw this in a woman suffering from cancer of the breast, who for years attended among Mr Bryant's out-patients at Guy's Hospital, and at last was thus cured.

Another form that the hard variety of cancer takes is to spread as an indurating infiltration of the skin, almost like sclerodermia, without any distinct tumour. The writer saw a remarkable example of this disease under Professor Velpeau's care at La Charité, in Paris, in 1864, spreading from the breast. He had previously described it as "squirrhe en cuirasse." (See plate v in Sutton's 'Tumours.')

How far the alveolar walls and stroma of carcinoma are a new formation, there is still some uncertainty; and perhaps different cases differ in this respect. The analogy of keratoid carcinoma, to be presently described, suggests that they may be made up of pre-existing tissue-elements, modified in their arrangement by the pressure of the masses of epithelial cells which are in contact with them on all sides. On the other hand, it is certain that in growing cancers the alveolar walls generally contain numerous leucocytes, from which a new fibrous tissue may be developed. As Waldeyer remarks, when a carcinoma spreads in the interior of a thrombus within the channel of a vein, its alveoli can be nothing else than new formations. It is in cases of this kind, however, that the alveoli are sometimes ill marked.

Externally, cancerous tumours are distinguished by their having no defined margin, by a white, yellowish or pinkish colour, and by the more important character of infectiveness, shown by secondary nodules in the lymph-glands, lungs, or liver. If the cut surface of such a growth is squeezed, or scraped with a knife, the cell-masses, with more or less of an albuminous fluid, escape, forming what has long been known as the "milky" or "cream-like" *cancer-juice*, or appearing as a soft, curdy, solid substance. The cell-masses themselves—the "*cancer bodies*," as Waldeyer called them—may sometimes be drawn out entire from two or more alveoli, so as to have a branching or reticulated appearance.

Special forms.—In 1852 Bidder, of Dorpat, described in 'Müller's Archiv' a case in which the cells of a soft cancer of the pylorus were identical with those of columnar epithelium. Many other instances of the same kind have since been recorded under the title of "cylinder-celled" or "columnar-celled epithelioma." This form of glandular tumour occurs in the stomach and intestines, from the cardia to a short distance from the anus, in the biliary passages and gall-bladder, and (according to Cornil and Ranvier) in the nasal fossæ, the body of the uterus, and the ovaries. Secondary nodules may occur almost anywhere: Dr Fagge recorded them in the liver and the brain, and they have also been found in the lungs and in the bones. A primary columnar-celled cancer forms an irregular shallow ulcer with a soft, raised, slightly projecting border. It may yield an abundant juice, full of the columnar cells, which are often still laterally coherent. A thin section generally shows that they are regularly arranged around the borders of long-branching channels or alveoli. But they may be polymorphous, and there are transitions between this and other forms of cancer. Moreover, primary growths give rise to secondary growths of the common alveolar type.

Duct cancer is also known as columnar carcinoma. It is a malignant epithelial growth in which the cells are columnar or cylindrical in shape, and repeat the structure not of glandular alveoli but of ducts. It is found in the gall-bladder and gall-ducts and in the breast.

*b. Squamous carcinoma.**—This was the form of growth which was formerly known by the inappropriate name of "Epithelioma," originally given to it by Hannover, of Copenhagen, in 1852, and still used by surgeons, and it is also known as epithelial cancer—another unfortunate name, since all cancer is an epithelial growth. Many French writers and the late Dr Hughes Bennett, following Lebert, called it "cancroid." A better term is that used by some German pathologists—epidermoid cancer.

In this form of new growth the cells undergo a chemical change of the protoplasm into keratin, as in the *cornifaction* of the superficial layers of the epidermis. The horny cells of the cancer nodules are pressed into globular masses, more or less laminated in structure, but often having a transparent centre, which looks like a single large cell. These masses were called by Lebert "epidermic globes." In this country they are compared to "birds' nests," by the German pathologists to pearls or onions (Epithelkugeln, Perlkasten, Zwiebeln). These masses in themselves afford no proof of the presence of a malignant new growth. On the contrary, they may be found in lupus, in the hyperæmic borders of cicatrices, and in sebaceous tumours (Cholesteatoma, Margariten-Geschwulst). But when seen in an infective tumour they show it to be not merely a carcinoma, but one which has its origin either in the skin or in some mucous membrane having a squamous, laminated epithelium.

This form of cancer is most frequent about the orifices of the body. The mucous membranes upon which it may occur are those of the mouth, pharynx, larynx, and œsophagus, the lower part of the rectum, the urethra and bladder, the vagina and os uteri, and the glans penis. On the surface of the skin it is most characteristic in the scrotum, where in England it used to form the commonest form of "chimney-sweep's cancer." It occasionally affects the scars of lupus or other ulcers. In many of these situations, however, it is common to meet with growths which, from the presence of horny globes deserve the title of keratoid carcinoma, yet contain the globes in small numbers, and in their general appearance and structure approximate to the ordinary form of cancer. Such transitional varieties are met with in the bladder, and they are common in the cervix uteri. In the œsophagus they become more frequent from the pharynx downwards, until at the cardiac orifice of the stomach cornification in the cancer-bodies ceases altogether, and is replaced by purely glandular carcinoma.*

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* *Synonyms.*—Keratoid, epidermic, epithelial, flat-celled, horny cancer; epithelioma, cancroid.

The keratoid form of cancer infects the lymph-glands which correspond with its primary seat, and is less frequently carried to distant parts by the blood-stream, than glandular cancer or than sarcoma.

In the more characteristic cases of keratoid carcinoma, the material which can be scraped or squeezed from the cut surface of the growth is firm and granular looking, like the secretion of a sebacous gland. The "cancer-bodies" often come out in the form of long worm-like masses.

Histologically, keratoid carcinoma differs from common cancer in some other respects as well as in containing the "globes" or "birds' nests." Not infrequently some of the cells have prickle-edges. Another peculiarity is that the "cancer-bodies" are much more plainly seen to form continuous branching processes which grow more or less vertically downwards from the surface, penetrate the deeper structures and push them aside. Indeed, in its typical forms keratoid carcinoma may be said to present no definite alveoli. The matrix that intervenes between the different cancer-bodies is commonly full of leucocytes.

It has now been clearly proved that the so-called "*Rodent ulcer*," which occurs chiefly on the face of persons advanced in life, and which is characterised clinically by its very slow course, is only a variety of keratoid carcinoma. It is the least malignant of all cancers, and, except from its histology and incurability, would not be called a cancer, for it never affects the lymph-glands or internal organs.

c. Colloid carcinoma.†—The most aberrant form of cancer is that known as *colloid*. At first sight no one would take the soft, trembling, translucent masses which characterise this form of new growth for cancer at all. But histological investigation shows that they are essentially epithelial and alveolar in structure, that they invade surrounding organs, and that they reproduce themselves in distant organs. The epithelial cells are swollen into glistening, structureless globes; and it is only towards the centre of some of the alveoli that a few unaltered cells may still remain visible.

Most pathologists regard these colloid growths as a mere degeneration of carcinoma, either of the ordinary glandular or of the above-described columnar type. But this view is not without difficulty, for it does not explain why colloid degeneration does not affect similar squamous cancers or sarcomata or innocent tumours. The mucoid, gelatinous or myxomatous degeneration of some fibroid growths is a different thing, and even this myxomatous tissue may be a primary formation. Again, the locality of colloid cancer is peculiar, and in some cases the colloid character is apparently present from the beginning. Lastly—and this perhaps is the most important argument,—secondary growths reproduce the colloid structure, whereas the metastases of other forms of cancer repeat the primary structure, but not the caseous or fibrous or hæmorrhagic degenerations.

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* Another proof of the close relation between the two forms of cancer is sometimes afforded by an examination of the structure of the secondary nodules from a case in which the primary growth is keratoid. In some instances a nodule in the kidney or in the lung will present horny laminated globes (birds' nests) as well formed as those in the original seat of disease in the tongue or œsophagus; but in a case of keratoid cancer of the lip in which the cervical glands contained typical globes or pearls, some large secondary masses in the liver had more the structure of a glandular carcinoma, the indications of cornifaction of the cells being slight and imperfect.—C. H. F.

† *Synonyms.*—Carcinoma alveolare, gelatinosum, reticulare.

Colloid material differs from mucus in not being precipitated or rendered opaque by acetic acid, and also in containing sulphur. It closely resembles the mucilaginous contents of thyroid cysts. There can be no doubt that much of this colloid material is a new formation, not degenerated cell-proteid, for the alveoli have so enormously increased in size that they are often visible to the naked eye.

The primary growth is usually found in the stomach or intestine, and huge masses sometimes fill the abdomen. Colloid cancer is very rare elsewhere than in the abdomen, but occasionally affects the breast.

Locality of cancerous growths generally.—Cancer of whatever kind only occurs as a primary growth on epithelial surfaces; never in connective tissues, muscle, or nervous organs. This important generalisation we owe to Waldeyer.

Squamous carcinoma is confined to the epidermis and the similar flat tesselated epithelium of mucous membranes. It may be found on any part of the integument, and is common on the glans penis, the scrotum, the vulva, and the anus. It is still more common on the lower lip and tongue (particularly in men), and is not infrequent in the fauces, the larynx, the pharynx, and the œsophagus. It is also found in the urethra, bladder, and pelvis of the kidney. It often begins as a papilloma.

Ordinary alveolar, glandular, or spheroidal-celled cancer is most common in the stomach and rectum in both sexes, and in the female mammary gland and cervix uteri. It is less frequently met with in the colon, and is extremely rare in the small intestine. It is common in the head of the pancreas, in the gall-bladder and passages, and is met with in the kidney and bladder, in the thyroid and the adrenal bodies. It is not uncommon in the testis, though many "malignant sarcoceles" are now found to be really sarcomata.

Colloid cancer, as above stated, is almost exclusively confined to the stomach, peritoneum, and abdominal organs.

Primary cancer is extremely rare in the liver, the trachea, and the lungs. It never occurs in the heart, the brain and cord, the spleen and lymph-glands, the bones or serous membranes.

Secondary cancer is most common in the lymph-glands, the liver, and the lungs, and in serous membranes; but it also occurs in the kidneys, the heart, and the bones.

On the whole it may be said that the organs which are the favourite seat of primary cancer are very rarely invaded by secondary growths, and that those which are most often infected are themselves very rarely the source of infection.

We shall see in describing cancer as it affects the various organs of the body how very much its local characters differ, so that cancer of each has to be separately described.

Histogenesis of cancer.—The limitation of primary carcinoma to the skin and mucous membranes, *i. e.* to the epiblast and hypoblast, and the glands developed from them, has been now fully established, although there are a few positive exceptions which are hard to explain away and although, as we have seen, some epithelial surfaces are very seldom thus affected.*

That leucocytes are unable of themselves to produce epithelium is evident by the healing of wounds and ulcers from the margins, and by the success of Reverdin's practice of transplanting cuticle.

Klebs, however, supposed there might be an "epithelial infection" or "spermatic influence," by which, for example, in a healing ulcer the rete mucosum of the skin at its margin might convert the cells of the adjacent granulation into epithelial cells.

In 1867 Köster propounded the view that the cells of cancerous growths in the skin are formed from the endothelia of lymph-channels. This view, however, has also met with little support. Virchow's doctrine had much more foundation, namely, that cancer grows in the living connective-tissue cells of organs; but this also must be now abandoned, and the direct and immediate derivation of carcinoma from epithelium which was maintained by Waldeyer is now universally accepted.

Secondary tumours agree in structure not with the organ or tissue in which they are found, but strictly with the primary growth. We must, therefore, derive their cells also, though indirectly, from the epithelium in which the parent tumour arose.

Degeneration of cancer.—Malignant growths do not always kill the organism they invade before they manifest themselves the signs of decay and death.

They frequently undergo "caseation." Whether this is altogether spontaneous—an indication that the cancer-bodies have reached their natural term of life—or whether it is due to interruption of blood-supply is uncertain. It often affects all but the narrowest possible growing margin of a tumour, especially of a secondary nodule; and not uncommonly, when a caseating tumour is seated in a solid organ, like the liver, its centre softens down so as to produce a cavity filled with straw-coloured albuminous liquid. Calcareous degeneration is exceedingly rare, and cancer seldom or never suppurates. Fibrous degeneration with wasting and atrophy as above described (*supra*, p. 92) is not infrequent, even when the progress of the wasting tumour is continuous. Soft vascular cancers are very liable to hæmorrhage.

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* Virchow recorded in 1850 a case in which the tibia is said to have been affected by a primary "cancroid," the skin being healthy; and Otto Weber is quoted as having in 1859 related a case in which there was a similar lesion of the lower jaw. It might be argued that it is possible for a lesion starting from the skin to extend downwards and to spread into the deeper structures, so as to appear to have begun in them. Paget, for instance, speaks of having "seen two examples of primary epithelial cancer in lymphatic glands;" one was in a sweep, whose groin contained a large mass, notwithstanding that the penis and the scrotum appeared to be unaffected. Thiersch records a case in which a nodule seemed for a time to be subcutaneous, but really had had its origin in the sweat glands. Further, it is conceivable, as was suggested by Remak in 1854, that a fragment of germinal epithelium, embedded in other tissues from fœtal life, may sometimes afterwards develop into a cancerous tumour, instead of forming an innocent dermoid cyst.

According to Thiersch it is no uncommon thing for cancer to arise in the wall of such a cyst after it has existed for a number of years; and Waldeyer cites an instance in which a similar change occurred in a congenital sacral tumour.

Ætiology of cancer.—When malignant tumours were first studied they were believed to be parasitical, *i. e.* having a life distinct from the body in which they grow; and there is some truth in this view, for they will be vascular, flourishing, and growing, while the host is pale, emaciated, and dying. They are not, however, like hydatids, mere parasites, but are formed from the tissues in which they lie, and are to a great extent, as we have seen, governed by the locality where they first appear.

Then cancers were compared to oak-galls, and supposed to be monstrous outgrowths due to some imported irritant; but here also the analogy fails, and gives no clue to their infective and destructive virulence.

During the former half of the present century it was generally held, in accordance with the prevalent humoral pathology, that cancer was only a local manifestation of a malignant cachexia of the blood, and this was compared with that of syphilis or of tubercle. The fact that cancer scarcely ever begins in more than one organ, and only spreads from a single primary source, is almost conclusive against this view.

When the microscope was first used to investigate their structure it was hoped that a distinctive "specific" cancer-cell could always be recognised; but, as we have seen, some of the most malignant new growths are not carcinomatous in structure, and innocent adenoma may be almost indistinguishable in microscopic structure from malignant cancer. The essentially malignant character of a new growth is its infectiveness, and this quality is associated with very different kinds of structure.

The great discovery of microbes as the cause of specific fevers, infective granulomata, and of pyæmic infective abscesses, naturally led to search for some similar virus as the cause of cancer, but all such investigations have failed, and at present there is no evidence for the theory. Moreover the pathological course of cancer, and its relation to malignant sarcoma, seem to make the microbial hypothesis improbable. Cancer is not contagious from man to man, and all efforts to transmit it from man to the lower animals have failed, including the most persevering, thorough, and elaborate experiments of Messrs Shattock and Ballance.* It is only infective within the body.

It has been thought that if microphytes fail, microzoa may be invoked; and unicellular organisms, supposed to be akin to psorosperms and coccidia, have been detected, stained, and figured, inhabiting the epithelial cells of cancer ('Journal of Pathology,' October, 1892, *ibid.*, 1893, and Mr Plimmer's more recent paper, 'Proc. Royal Soc.,' 1899). Some of these, as described by Ruffer, appear not to be what others certainly are, altered nuclei of epithelium, but to be true animal parasites; yet that they are specific and related to the infective qualities of cancer is unproved. They exist in other tissues, both morbid and healthy,—as, for instance, in the epithelium of the rabbit's liver, in Paget's disease of the nipple, and in molluscus contagiosum. An ingenious inquiry was carried out by Dr G. T. Brodie in Prof. Halliburton's laboratory to ascertain whether either chitin or cellulose (which are known to form the envelope of encapsuled Protozoa) is present in cancerous tumours. The result was negative.

Traumatic origin: injury and irritation.—If not caused by a particulate vegetable or animal infection, may the origin of cancer be regarded as due to the action of ordinary irritants upon tissues rendered prone to free cell-production, reproducing the local structures, but in a useless, paratypical, imperfect, and exuberant manner?

Here the facts of locality above mentioned are important. The parts most prone to cancer are those most exposed, not to violent injury, but to frequently repeated irritation—the lips and tongue, and the narrows and sharp turns of the alimentary canal, the pharynx, the œsophagus, the cardiac and pyloric orifices of the stomach (*not* the ileo-cæcal valve), the sigmoid flexure, and the rectum.

The skin and the mucous membranes are more exposed to irritants than the mesoblastic organs. We do not see cancer of the arteries and cardiac orifices, because there is no true epithelium there. But when skin or mucosa or their glands are exposed to irritation, they are apt to become cancerous if the person is old or if the organ is worn out. Hence the frequency of cancer in later life, and in the mamma and uterus after their functions are over.

Instances of cancer following long-continued slight irritation, not gross injury, are the following, and it would be difficult to find cases of the occurrence of other new growths under similar conditions:—Cancer of the scrotum from soot, carcinoma mammæ after Paget's disease, cancer of the lip in men who smoke, cancer of the tongue from the irritation of rough teeth or following the chronic thickening called leucoplakia, cancer of the gall-bladder associated with calculi, and cancer of the glans penis in cases of phimosis; lastly, the growth of squamous cancer in the cicatrices of wounds and burns—where there can be no embryonic "rests."

With respect to more severe and single injuries, patients often ascribe the origin of a tumour to a blow, and there are a few striking cases in support of this view.* But these cases are surely very rare compared with those in which no injury can be remembered; and the enormous majority of injuries are not followed by the growth of any tumour.

The embryonic theory of tumours.—Perhaps the most ingenious and original contribution to the ætiology of cancer and new growths in general is the theory of Cohnheim, that their origin in an adult is in some portion of embryonic tissue which was arrested in its development, and has thus remained shut off until its dormant powers are roused into activity.†

Cohnheim maintained that cancers are most apt to arise at the orifices of the great mucous channels, which are the seat of complicated processes in the development of the embryo, so that involutions of the epiblast and hypoblast may be supposed likely to occur there and to leave "rests" behind. The formation of the new growth he attributed to a failure of "physiological resistance" on the part of the normal tissues around.‡

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* The following cases are related by Paget:—A boy was accidentally wounded in one eye, which was sound. Within a few days a medullary tumour grew from the eyeball; it was removed three weeks later, but it quickly recurred, and destroyed life. Another boy fell and struck his knee; swelling followed, which was at first supposed to be inflammatory; but it increased, and proved to be a large medullary growth round the lower end of the femur. A steady man at his work, slipped and strained or broke his fibula; the injury led to pain and swelling, and in the course of eight weeks there was found to be a growth of large size outside and within the shaft of the bone.

† Virchow had previously proposed to account for the remarkable fact that enchondromata arise in bones, but never from permanent cartilage, by supposing that a fragment of the original cartilaginous precursor of the bone might remain unossified, and afterwards form the starting-point of a tumour.

‡ Thiersch attributed the liability of aged persons to squamous carcinoma to the atrophied and inelastic condition of the fibrous texture of the skin at advanced periods of life, whereby the extension downwards of the epidermis may be facilitated.

Cohnheim's theory applies admirably to certain kinds of tumour. It is probably true of dermoid cysts, and accounts for the peculiar liability to neoplasms in the mouth and pharynx, the anus and urogenital tract; also for the presence of striated muscle in tumours of the kidney, and of chondroma of the parotid arising from Meckel's cartilage. It may also, perhaps, explain the frequency of new growths in the uterus or the breast of unmarried women in the later years of life, since in such cases the female organs no doubt contain germs which fail to receive their normal physiological stimulus. In the case of the breast Dr Creighton has worked out a similar idea with extreme care and in great detail. Having studied fully the normal process of evolution or "unfolding," which the mamma undergoes when preparing for its secretory functions, he finds that the growth of tumours in it may be regarded as a modification of that process, under what he terms "spurious stimulation," occurring at a time when the organ is in a quiescent condition.

Cohnheim's hypothesis, however, does not help us to understand the striking difference between innocent and malignant tumours, nor does it explain why a fragment of embryonic tissue, after lying dormant for years, should at last produce a cancer.

Heredity of cancer.—Beside the degeneration of age, may we find another predisposing cause of cancer in the facts of inheritance? That in most cases no such tendency is present is probably true, but we every now and then meet with such striking cases that the popular belief on the subject seems to be justified. The experience of Sir James Paget is very striking: in his hospital days he found that the proportion of cases in which a family tendency could be traced was one in six; subsequently, in private practice, he made it one in four, and latterly one in three. Some of the cases recorded cannot be set down as mere coincidences. Thus a lady, who died with cancer of the stomach, had seven children and about thirty grandchildren who grew up. Some of them were still living; but a daughter had already died with cancer of the stomach, two granddaughters with cancer of the uterus, a granddaughter with cancer of the breast, a grandson with cancer in the bladder, a grandson with cancer in the rectum, and a third grandson with cancer of the axillary lymph-glands (? secondary).

A point of considerable importance is that where the disease is transmitted by inheritance it often appears in the descendants at a much earlier age than in the first patient. Thus a young lady of twenty-four died of "epithelial cancer" of the pharynx; her mother had been attacked by cancer when between forty and fifty years old, her grandfather between sixty and seventy, a great-aunt at about forty, her great-grandmother at eighty or older. A schoolfellow of Dr Fagge died of cancer of the rectum before he was forty years of age; his father and his grandfather were believed to have also suffered from cancer, but at advanced periods of life. In fact, early cancer (like early gout, though perhaps not so constantly) is more often inherited than cancer later in life.

In the 'St Bartholomew's Hospital Reports' for 1866 the late Mr Morrant Baker analysed 103 of Paget's cases, and carefully investigated the question whether the appearance of cancer in successive members of the same family could be attributed to the mere propagation of a *local* defect, or (in other words) of a tendency of some one organ to a special morbid change. But he f[illegible] that it was hardly more common for the [illegible]ase to occur in two members of the same family at the same spot, than for its seat to be different. Hence it would appear that we cannot regard inheritance of cancer as inheritance of a certain structure of particular organs, but as inheritance of a general tendency of the epithelial structures to irregular overgrowth.

But is heredity limited to cancer, or does it apply to all tumours? On this point Cohnheim states that sometimes a mother suffers from adenoma of the breast, and her daughter afterwards from cancer of the same organ; and among Paget's observations there are several in which it seems clear that carcinoma occurred in one member of a family and sarcoma in another. So, again, Sir William Jenner mentions the case of a man who had cancer of the tongue, and whose child, twenty-two years before, had died at the age of two or three years with disseminated malignant growths, which one may presume to have been sarcomata. In the course of the discussion on cancer at the Pathological Society in 1874 Mr Hutchinson remarked that persons who had common warts in large numbers generally knew of relatives affected with cancer. Moreover Paget has insisted on the frequency with which liability to sebaceous cysts, or to certain multiple osseous tumours, is transmitted by inheritance. It may be, therefore, that the hereditary transmission of cancer is only part of a wide-spread proclivity to new growths of all kinds.

This view, however, appears not to be supported by any sufficient evidence. Coincidences must occur with all common diseases; and there is no evidence that sarcoma is more hereditary than lipoma, or that either is more often found in cancerous families than in others.

Locality.—Another suggested explanation of the occurrence of several cases of cancer in a family is that it is favoured by certain *soils* or by certain *houses*. In England Mr Haviland has found that cancer is much more prevalent in inland towns and country districts than in others; that it is as common in proportion to population in the country as in towns, and that it is more frequently met with in damp regions and in clay soils; or, more precisely, "that the cancer-fields in England and Wales are found in the sheltered and low-lying vales, traversed by fully formed and seasonally flooded rivers, and composed of the more recent argillaceous formations; and that the districts having the lowest death-rates from cancer occupy the more elevated areas composed of the oldest rocks, among which the limestone areas are coincident with the very lowest mortality."

Shattock and Ballance, in the first volume of Allbutt's 'System of Medicine,' quote some remarkable instances of numerous cases of cancer of various organs in a country village in Shropshire and in a house at Ashburton. (See also Mr Shattock's Morton Lecture, 'Lancet,' May 19th, 1894.)

Other facts of the natural history of cancer.—Among more general facts of possibly ætiological bearing may be mentioned the popular belief, which Paget did not reject, that the appearance of cancer may be favoured by "deep anxiety, deferred hope, or disappointment." But these conditions, it may be remembered, beset the later periods of life much more than the time of youth.

That true cancer, as distinct from malignant sarcoma, is very rare in children, and comparatively rare in young adults, is a matter of common experience. Cancer of the lip in men and of the cervix uteri in women seems to occur earliest, sometimes at or even under twenty, cancer of the œsophagus and rectum later, and rodent ulcer is almost confined to advanced age.

More women than men die of cancer (more than double in England), but this is probably due to two causes: first, that men have no uterus or functional mamma; and secondly, that men are more liable to premature death from injury, exposure, contagion, and intemperance, so that fewer men than women are living at the time of life most obnoxious to cancer.

In an interesting address on the increased prevalence of cancer generally, Dr Payne gives strong statistical and other evidence that this applies to men as well as to women, though less in degree, and that increased recognition and other supposed explanations are not sufficient ('Lancet,' 1899).

Among races of mankind there does not appear to be any so peculiarly exempt from or so peculiarly subject to cancer as to guide speculations on the ætiology of the disease.

Among the lower animals, Mr Bland Sutton states that such innocent tumours as osteoma and odontoma, papilloma and simple adenoma, are all common; and that most of the malignant growths are sarcoma, which is not infrequent both in wild and domestic animals. True carcinoma is almost unknown, the growths on the penis in stallions being warts, and the glandular tumours of the mammæ being only recurrent and infective, if at all, in the domestic cat.

Provisional results as to nature and course of cancer.—Although the outcome of all the ingenuity, skill, and industry of the workers on this most attractive problem in pathology may seem disappointing, although we are still as ignorant as fifty years ago of the origin, the prevention, and the cure of cancer, yet the labours of Virchow, of Cohnheim, and of Waldeyer have not been without result.

First, we know that local manifestations of the disease are not the result of a precedent "dyscrasia" or "diathesis," and therefore, if we ever succeed in curing the primary growth, we shall cure the whole disease.

Secondly, we know that the origin of a cancer is always in epithelial tissues, in skin or mucous membrane, or their glands, that the infection takes place first by contiguity in the nearest tissues, secondly by conduction from the alveoli by the lymphatic vessels to the nearest lymph-glands, and thirdly from the veins to the right side of the heart and the lungs.

Thirdly, though infective in the individual, cancer is not contagious to others, nor, so far as experiments go, is it capable of artificial conveyance.

In the majority of cases it does not appear to be hereditary, although there are occasional cases in which this condition of its occurrence cannot be denied.

Cancer is observed in both sexes, and in every race, climate, and occupation, but is rarely seen until the later stages of adult life.

Clearly distinct from sarcomata and other non-epithelial tumours both in structure and behaviour, and from innocent epithelial tumours by its infective characters, the group of tumours now included under the term carcinoma is natural and essentially homogeneous. The differences cylindrical celled carcinoma, hard, soft, and colloid, are differences dependent chiefly on locality, and the most satisfactory pathological as well as most useful clinical division of cancer is by their seat—cancer of the lip, of the tongue or the œsophagus, or of the scrotum, of the penis, and of the skin, beginning in warts or in scars; cancer of the breast, pylorus, colon and rectum, kidney, testes, cervix uteri, and so on. Yet, as we have seen, while the locality is all-important for the structure and behaviour of the primary growth, it is without the least effect on the secondary growths; they faithfully copy the original tissue, and are totally unaffected by the organ in which they are found.

The most frequent local cause of cancer—it may be called predisposing or exciting—is long-continued irritation, especially when it causes chronic inflammation.

If we may reason from analogy where definite facts fail us, we should compare cancer with pyæmia, with tuberculosis, or with syphilis. In these conditions, as in cancer, we have a primary source of infection, and similar secondary effects produced by conveyance through either the lymph or the blood. But in all three there is contagion as the cause of the primary lesion, and this is, to the best of our knowledge, absent in cancer. What we observe in the latter disease is that under certain conditions, of which advancing age, local obsolescence of structure and function and hereditary influence are the chief, long-continued irritation of a spot of skin or mucous membrane, which would in other cases produce thickening with hyperplasia of the epithelium, now leads to luxuriant aggressive multiplication of cells which invade the mesoblastic tissues, and mimic more or less imperfectly the structure in which they originated.

The secondary tumours are certainly later in pathogeny as well as in external recognition than the primary, and they must be the result of something conveyed from the original growth, for their likeness betrays their paternity. This something cannot be an influence or a chemical poison, it must be particulate and living. From comparison with contagia of demonstrated vegetable nature, as well as from the negative results of the extended experiments by Shattock and Ballance, D'Arcy Power, and other competent investigators, we may assume that this contagion is not a microphyte. As above said, it produces no suppuration, granulations, or fever, and above all it is not introduced from outside. The last fact, if we may until disproved take it as one, is equally conclusive against the hypothesis of an animal microbe.

What other living contagion remains but the morbid or paratypical epithelial cells of the primary tumour? Probably while young, small, and active, they escape after birth from the parent cells into the lymph-spaces, are conveyed to the nearest lymphatic gland, and there reproduce their kind.

The practical conclusions as to treatment of cancer which result from this view of its pathology are most important. We should jealously watch white patches on the tongue, phimosis, and papillomata in elderly subjects, and remove them on the first suspicious indications; cure should *precede* diagnosis.

When a tumour has already appeared, it should be removed as early, as extensively, and as thoroughly as possible, and not only the tumour, but where possible the entire organ or limb.

No other treatment, rational or irrational, has stood the test of experience as of the slightest value, but early and extensive removal has again and again saved life, and the terrible disease has never returned. This is true not only of such slow-growing and comparatively innocent tumours as rodent ulcer and squamous cancer of the lip and scrotum, but also of cancer of the tongue and of the anus, cancer of the breast and testicle, and even, in exceptional cases, cancer of the rectum and colon, of the stomach and the womb.

SPECIFIC FEVERS

> Nunc, ratio quæ sit morbis, aut unde repente
> Mortiferam possit cladem conflare coorta
> Morbida vis hominum generi pecudumque catervis
> Expediam. LUCRETIUS.

General and local diseases—Specific diseases: infective, general, and febrile—Typical and doubtful members of the group—Table of their clinical course—Table of their distinctive characters—Order followed—List of infectious fevers to be notified.

WE have already seen that no scientific classification of diseases is possible. For they are not comparable things. One is an anatomical change of structure; another is a pathological process; a third the result of the action of a mineral poison, a mechanical injury, or a parasitic animal or plant; while a fourth is only a frequently recurring series of concomitant symptoms, of which we know neither the material conditions nor the probable cause.

We might arrange diseases *anatomically*—either by the *regions* they affect, as those of the head, the breast, the belly, and the limbs; or by the *tissues* involved, as bones, joints, mucous membranes, skin; or by the *organs*, as brain, lungs, heart, liver. We should need a separate class for diseases which affect more than one organ, another for those which appear to affect every organ at once, and still there would remain diseases "of uncertain seat."

A *pathological* arrangement would group together diseases in which inflammation was the most important morbid process; those which consist of fever without local inflammation; contagious and miasmatic diseases; degenerations of tissue; new growths—and so on as far as our knowledge of morbid physiology extends, whatever the region, tissue, or organ which these processes affected.

Again, we might make an *analytical* or clinical arrangement, founded on symptoms; and consider successively diseases which are characterised by a raised temperature, by dropsy, by dyspnœa, by jaundice, by the presence of albumen in the urine. Such a clinical classification is the most useful for diagnosis, and should be borne in mind at the bedside.

Lastly, an *ætiological* classification would put in one group those diseases which depend upon the invasion of animal or vegetable parasites; in another those which are produced by lead, mercury, or other poisons; in a third and fourth dietetic and climatic diseases.

But even if we had completed the task Morgagni set himself, and had discovered the seat and the cause of every malady to which the human race is liable—we should still find that no single principle of classification would give satisfactory results.

The object of medicine is to prevent, to predict, and to cure. These objects are sometimes best served by knowledge of morbid anatomy, sometimes by knowledge of processes, and sometimes by knowledge of causes.

Who is to decide whether so well understood a disease as lead palsy will be best considered with colic and other effects of plumbism, or with other forms of paralysis, or with wasting affections of the muscles, or as a peripheral neuritis? For different purposes each arrangement would be the best.

Every kind of classification which rests on a firm basis of facts is useful so far as it helps us to remember certain common characters of the objects classified, and mischievous if it is allowed to obscure other common characters upon which other classifications may be constructed.

In a systematic treatise, however, it is necessary to choose some one arrangement, and our choice should be decided by reasons of practical convenience. Our divisions need not be upon a uniform plan, but they should be few and plain. They should depend as little as possible on hypothesis as to the nature and causes of morbid processes, and as much as possible on clinical and pathological facts. And they should agree in the main with those traditional names and arrangements which do not contradict ascertained facts, and which are generally accepted by the best authors.

When a clinical group of symptoms is found associated with a definite anatomical change in a given organ, it is best treated along with other "diseases" of that organ. When "it has no anatomy," it is most conveniently grouped with its nearest clinical allies. When it affects several organs, it should be referred to that which is its primary or most important seat. On these principles we are not afraid to place epilepsy near cerebral hæmorrhage, asthma not far from cancer of the lung, and rheumatism with osteo-arthritis.

The general arrangement in the following chapters is local and anatomical, following the organs of the body; while functional diseases are, as a rule, placed with those presenting similar symptoms.

One important exception, however, has been made, in accordance with general practice.

Specific infective fevers.—There is a group of "general diseases" which, though variously defined and more or less extended, has been recognised from the earliest times. The type of this group is the disease called typhus fever, when that name only meant a variety of the genus fever. With it were associated other "continued" fevers, so called to distinguish them from the intermittent fevers common in Greece and Italy.

Closely associated with "continued fevers" is another group of diseases, the eruptive fevers or exanthemata: smallpox, measles, and their allies. These were classed with diseases of the skin, not only by Willan and Bateman, but also by Hebra.*

The condition of fever may, as we have seen (p. 38), be produced by local inflammation, or it may be idiopathic, it may be continued or intermittent, accompanied by a rash or not. Since the discovery of the important part played by microphytes in disease, as described above in the chapter on Infection, it appears probable that all the idiopathic fevers and inflammation itself—or at least purulent inflammation—are alike due to the reaction of the organism when a brood of microphytes invade the blood and tissues.

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* Measles and scarlatina appear in Willan's 'Order Exanthemata' with Nettle-rash and Rose-rash, Smallpox with Scabies, and Chicken-pox with Eczema. Hebra's treatise is entitled 'Acute Exantheme und Hautkrankheiten,' but Measles and Scarlatina appear after the introductory chapters, between Seborrhœa and Erythema. Why dermatologists did not include Typhus is not apparent. It is as much an exanthem as Measles, and Measles as such a continued fever as Typhus.

Leaving the old term "fever" to denote, not a disease or group of diseases, but a physiological state of which pyrexia is a synonym—we may unite the continued fevers and exanthems on the basis of their being contagious, and due exclusively to the entrance of living contagia. In certain cases we can identify the actual microbe; and, without venturing to push the argument from analogy, we may affirm that whether each is directly due to the invasion of specific microphyte or not, typhus and syphilis, measles and scarlet fever, smallpox and chicken-pox, are each caused by a specific contagium.

But not only do they agree in this "specific" (*i. e.* special or peculiar) ætiology, they are all "general diseases." Some, like typhus, are without any local lesion but what is demonstrably the result and not the cause of pyrexia; while in others, like enteric and scarlet fevers, though local lesions are constant, they are clearly parts of a general process, and not its precedent cause.

Again, these contagious and general diseases agree in running a more or less defined specific (or peculiar) course, limited in time, and developing in regular sequence periods of incubation, invasion, ingravescence, culmination, and defervescence (cf. *supra*, p. 35).

Lastly, most of these maladies confer remarkable protection from a fresh attack, while they do not protect from one another.

We have here, therefore, the most natural and scientific group of diseases, each separate and definable, yet agreeing with one another and differing from the rest.

Table of the Clinical Course of Specific Fevers

Disease.	Mode of entrance.	Incubation.	Onset.	Exanthem after onset.	Duration.	Mode of exit.	Patient no longer infectious.
Enterica .	Mouth	2 weeks or less	Gradual	2nd week (7th—12th day)	3—6 weeks	Stools	At once.
Morbilli .	Breath ?	10—14days	Subacute	4th day	10—14days	Epidermis	3 weeks.
Scarlatina .	Tonsils	3 days or less	Acute	1st or 2nd day	7—10 days	Epidermis, urine	6 weeks.
Rubella . .	?	10—17 or more	Subacute	1st day	3 days	Epidermis	2 weeks.
Variola . .	Breath	12 days	Acute	3rd day	3 weeks	Crusts	1 week.
Varicella .	,,	12—14days	Subacute	1st day	5 or 6 days	Crusts	,,
Mumps . .	?	14—21 days	Acute	Absent	5 or 6 days	Saliva ?	3 weeks.
Whooping-cough	Mucus	5—14 days	Subacute	,,	Prolonged	Sputum	Doubtful.
Influenza .	Breath	Very short	Acute	,,	3—7 days	?	A week.
Typhus . .	,,	11—13days	Acute	1st week	2 weeks	Breath ?	At once.
Relapsing fever	Unknown	5—10 days	Acute	Absent	6 or 7 days, relapse	?	?
Plague . .	?	?	Acute	Inconstant	A week	Fomites	1 week.
Cholera . .	Mouth	?	Acute	Absent	3 or 4 days	Stools	At once.
Erysipelas .	Wound	1—4 days	Acute	1st, 2nd, or 3rd day	5 or 6 days	Epidermis	1 week.
Diphtheria.	Tonsils	Short, 2—4 or 5 days	Subacute	Absent	Indefinite	Sputa	Weeks.
Syphilis .	Crack in m. m.	6 weeks	Slight fever	First symptom	Months and years	Lymph or pus	2 years.

Have any other affections than the traditional continued Fevers and Exanthems a right of admission to this class?

Accepting as the best criteria those of exclusive origin by contagion and of "breeding true," we have no difficulty in admitting the typically specific disease Mumps to the list, and, with almost equal claims, Whooping-cough and epidemic Influenza. Diphtheria may certainly be added now that its pathology has been cleared up. Erysipelas is contagious, has a specific micrococcus and exanthem, but it does not in the least protect from recurrence, and in many respects is difficult to separate from infective traumatic inflammation of non-specific type.

Syphilis never arises but by direct contagion; the contagium is particulate; it breeds true; it affects not only the blood, but, as Sir William Gull well put it, body, blood, and bones; it protects against itself with only such rare exceptions, as are not entirely absent even in the case of Typhus and Smallpox; its invasion is accompanied by slight, occasionally by high fever, and by a characteristic rash. It differs from other specific fevers in the length of its course, and in the difficulty of separating this course from what may be called its sequelæ. It also differs in the important fact of being hereditary.

Gonorrhœa is strictly contagious in origin; its microbe, the gonococcus, has been identified, and it produces definite sequelæ in the epididymis, the sclerotic, and the joints. On the other hand, it runs no definite course, does not produce general disturbance, and does not protect from a secondary attack.

Tuberculosis is like syphilis in being hereditary, and in its variable and long-drawn course. Moreover, within certain limits it is contagious. But it is often more local than general; it does not affect every organ, and it certainly does not protect against itself. A definite bacillus occurs in all tuberculous lesions, and in well-marked cases of acute tuberculosis the rapid course and general symptoms much resemble those of a contagious fever. The local and chronic forms of tuberculous disease—as it affects the lungs (phthisis), the lymph-glands ("scrofula"), the joints (caries and white swelling), the skin (lupus), and the adrenals (Morbus Addisonii)—though specific and in one sense infectious diseases, are certainly not "fevers."

Pneumonia, *i. e.* acute lobar pneumonia, has strong claims on clinical grounds to rank as a specific febrile disorder. It has its micrococcus. It invades, runs its course, and subsides like a fever. Its symptoms are scarcely more dependent on the hepatised lung, as to time and severity, than are those of enterica on the typhoid ulcers. But cases of contagion and an epidemic course are both exceptional for pneumonia. It is generally believed to arise from common causes, as exposure to cold; it is often secondary to other diseases; and it is not self-protective.

Tetanus, a terribly painful and usually fatal disease, although its symptoms are those of an affection of the spinal cord, is certainly not organic but toxic in nature, and has now been conclusively traced to the action of a microbe which is probably derived from the earth. Happily it is not contagious from man to man, but it is certainly infective and specific.

Cholera is an exotic endemic and an occasional European epidemic disease, specific in its course and its pathology. To regard it as a mere local enteritis would be less reasonable than Broussais's doctrine that typhoid fever was only symptomatic pyrexia from ulceration of the bowels. Without doubt it is infective, as it certainly is epidemic, and this conclusion is independent of the pathogenic nature of Koch's comma bacillus; for no diseases are more certainly contagious than typhus and smallpox, yet in neither has the microphyte, if it exists, been discovered. Usually there is little or no pyrexia in cholera, but there is reason to believe that this feature is rather masked than absent.

Two epidemic febrile disorders, contagious and once terribly destructive, have happily been banished from this country—the Sweating Sickness since the sixteenth, and the Oriental Plague since the seventeenth century. The latter without question, and probably the former, is, like cholera, endemic and comparatively mild in certain localities; but when epidemic they acquire fearfully active properties, and we have lately learnt that after nearly two centuries' absence the plague may reappear in Europe.

Yellow fever is another specific disease. Its contagion has recently been discovered in South America. It has always been exotic.

Three specific diseases of the brute creation are occasionally transferred to man by inoculation; they are Anthrax or splenic fever, Glanders, and Hydrophobia. In the first the microphyte is known and its whole history investigated; it answers every test of being the true contagium of the disease (*v. supra*, p. 18), so that other maladies may be compared with this as a criterion. Glanders is almost as certainly and completely known. The pathology of Hydrophobia is in many ways obscure, and its microbe has yet to be identified.

Another remarkable malady, as fatal as hydrophobia, is known by the cumbrous name of "acute yellow atrophy of the liver." This has many of the characters of a specific disease, although, like cholera, it is not as a rule a fever, and the search for a specific microbe has hitherto been in vain. At present it is best left with other hepatic diseases.

While we must admit, as undoubted members of this class, some affections in which no microphyte has been found, we must exclude others, though certainly bacterial in origin, because they are not general or not febrile, or run no definite course, or do not protect. Leprosy, for instance, is allied to lupus and other infective granulomata, and will be considered with them under the section of Diseases of the Skin.

Ague and tropical dysentery do not depend on a microphyte, but on a microzoon, and differ greatly from the other specific fevers.

Rheumatism, though a general disease and febrile, is not contagious, does not protect, and has neither an exclusive or specific origin nor a constant or specific course. For purposes of diagnosis it should be considered along with pyæmia, gonorrhœal synovitis, and gout; and at present it is perhaps best to place it with other general diseases, accompanied by multiple synovitis.

The classification and order of these specific fevers is a matter of convenience only. We may see resemblance between enteric fever and cholera in their being what has been called miasmatic-contagious, and conveyed by water; and between others in having a constant exanthem, or a prolonged course, or imperfect protection against return, or in their origin from lower animals; but none of these points help to a single pathological or clinical arrangement.

In the following list are stated, in a brief and, of necessity, dogmatic form, the characters of the several diseases which entitle them more or less decisively to a place in this section.

Pathological Table of Specific Infective Diseases

Disease.	Microphyte. Author.	Local foci.	Exanthem.	Extent.	Course.	Protection.
1. Enterica . .	Bacillus of *Eberth*	Ileum, spleen, &c.	Rose spots	Pandemic	Less definite	Complete.*
2. Morbilli . .	None known	Bronchi	Blotchy	Endemic	Definite	Less.
3. Scarlatina .	Uncertain	Throat, &c.	Punctate	"	"	Less.
4. Rubella . .	None known	Throat	Various	"	"	Marked.
5. Variola . .	Micrococcus ?	Various	Pustular	"	"	Complete
6. Varicella . .	None known	Skin	Vesicular	"	"	"
7. Mumps . .	"	Parotid	Absent	"	"	"
8. Pertussis . .	"	None ?	"	"	Indefinite	"
9. Influenza . .	B. of *Pfeiffer*	Bronchi, &c.	Rare	Epidemic	Definite	Absent.
10. Typhus . .	None known	None	Mulberry	" †	"	Complete
11. Recurrens .	Spirillum of *Obermeier*	None	Absent	" †	"	Imperfect.
12. Plague . .	B. of *Kitasato*	Lymph-glands	Petechial	Both†	"	Doubtful
13. Cholera . .	Vibrio of *Koch*	Intestine	Rare	" †	"	Probable.
14. Yellow fever	B. of *Sanarelli*	—	Absent	Epidemic†	—	—
15. Mediterranean fever	M. of *Bruce*	None	"	Endemic †	Irregular	—
16. Dengue . .	None known	None	Occasional	" †	—	—
17. Beri-beri . .	Doubtful	Nerves, &c.	Absent	" †	—	—
18. Leprosy . .	Bacillus of *Hansen*	Skin, nerves, &c.	Erythema	" †	Long	—
19. Gonorrhœa .	Gonococcus of *Neisser*	Joints, &c.	Absent	Pandemic	Indefinite	Absent.
20. Erysipelas .	Streptococcus of *Fehleisen*	Skin	Marginate	Endemic	Definite	Absent.
21. Diphtheria .	Bacillus of *Loeffler*	Throat and nerves	Absent	"	Less definite	Marked.
22. Syphilis . .	Uncertain	Numerous	Coppery	"	Prolonged	Complete*
23. Tuberculosis .	Bacillus of *Koch*	Various	Absent	Pandemic	Indefinite	Absent.
24. Pneumonia .	Pneumococcus	Lung	Herpes	Sporadic	Definite	Absent
25. Tetanus . .	Bacillus of *Nikolaier*	Cord	Absent	Endemic	Irregular	—
26. Glanders . .	B. of *Loeffler*	Nostrils	"	Epizootic	Definite	—
27. Anthrax . .	B. of *Davaine*	Spleen	"	"	"	Complete*
28. Hydrophobia	None known	Nervous centres	"	"	"	—

* *I. e.* cases of the same person having two attacks, though they certainly exist, are so extremely rare that they do not affect the practical rules as to infection.

† No longer prevalent in this country.

Order and Affinities.—In the first edition of this work I placed Typhus first (as was probably Dr Fagge's intention) on the ground of tradition. But now it has happily become so rare that it seems better to give the first place to the most important of all fevers to physicians in Western Europe and America, that which is unfortunately called typhoid and abdominal typhus. This disease in many respects stands alone, and should be separated as far as possible from Typhus.

It is important to remember that the "continued" fevers are only so called in distinction from the intermittent and remittent fevers better known to Greek physicians, and that they are closely related to the so-called exanthems. I have, therefore, placed the three disorders of childhood with rashes next; and following them, the two with pustul exanthems, which are closely related clinically. They are naturally followe by two specific diseases without rashes—Mumps, which I have transferre from affections of the mouth, and Whooping-cough, which was formerl along with Influenza placed among disorders of the lungs.

Typhus finds its most appropriate place at present with other gra historical, epidemical, and happily in Western Europe obsolete fevers— Recurrens and Plague. With them may go the more modern pest lences known as Cholera and Yellow Fever. These five diseases are a endemic in certain regions, epidemic in others,—terrible in the past, b deprived more or less of their terrors by the progress of scientific medicin Other exotic diseases conveniently follow Yellow Fever.

Erysipelas, Diphtheria, Syphilis, Tubercle, and Pneumonia follow, eac of them with very peculiar characters, which separate them from o another and from the other specific diseases. Though placing them a among specific general diseases I have still thought it best to retain Pne monia among diseases of the Lungs, although Diphtheria is removed fro those of the larynx, Erysipelas from those of the skin, and Syphilis a Tuberculosis from "general pathology."

Notification of infectious diseases.—Under the Act of Parliame (52 and 53 Vict., cap. 72) passed in 1889 for the notification of infectio diseases to vestries or other local authorities, through the Medical Offic of Health for the parish, a registered practitioner is bound to give inform tion of the nature and locality of every case he visits or attends of t following diseases:—Variola, Cholera, Diphtheria (including so-called Me branous Croup), Erysipelas, Scarlatina, Typhus, Enteric, Relapsing, or a other continued fever, and Puerperal Fever.

ENTERIC FEVER

> Ac dum prima lues udo sublapsa veneno
> Pertentat sensus, atque ossibus implicat ignem
> Necdum animus toto percepit pectore flammam.
> VIRGIL.

History of the separation of Enteric from other forms of fever, and particularly from Typhus—Nomenclature—Geographical and zoological distribution.
Clinical course—incubation—onset and early symptoms—the exanthem—later course—Recovery and convalescence—Modes of death—Slight and abortive cases — anomalous cases — Complications — coincident fevers — Sequelæ — Relapses—Protection—Diagnosis, clinical and bacteriological.
Morbid anatomy—intestines—spleen—larynx—heart and muscles, etc.
Ætiology—The bacillus—its conveyance by water, milk, etc.—cases—Contributing conditions—season, drainage—age and habits, etc.—Mortality—Prognosis.
Treatment—food—stimulants—baths and antipyretics—antiseptic drugs—treatment of complications and during convalescence—Prophylaxis.
Febricula—its recognition apart from slight cases of Enteric fever.

Definition.—A specific infective fever of three or more weeks' duration, with a characteristic exanthem and equally distinctive lesions of the intestines.

Synonyms.—Typhoid Fever, Typhus mitior, Febris miliaris (De Haen), Gastric Fever, Infantile remittent Fever, Slow nervous Fever of Huxham (1739), Typhia (Farr, 1859), Pythogenic Fever (Murchison), Fall Fever in America.—*Fr.* Fièvre typhoïde, Dothienentérie.—*Germ.* Typhus abdominalis or Ileo-typhus (Griesinger, 1857), or Typhus without qualification.

History.—Of the fevers which were called "continuous" by the ancients, in contrast with the intermittent fevers common on the shores of the Mediterranean, the first to be separated from the rest were Smallpox and Measles, by the Arabian physicians of the tenth and eleventh centuries. The next was Scarlet Fever, in the seventeenth century; then Chicken-pox, in the eighteenth. The striking appearance of the skin in these four maladies threw their essential pyrexia into the background; they were named exanthems, and described among diseases of the skin. The other "fevers," excluding the Plague, were regarded as forming one "genus," varying in symptoms and course, but essentially the same. So Huxham, Hancock, Pitcairn, Cullen, and Heberden taught during the eighteenth century in England, and so Boorhaave, Sauvages, de Haen, and Hildenbrandt taught on the Continent.

In Great Britain early in the present century it was recognised that fever often occurred in a malignant, highly contagious form, to which the name typhus was given; that this was sometimes mingled with cases of much shorter and less dangerous fever, though equally contagious, which had great tendency to relapse, and was chiefly seen in Ireland; that other forms were less acute in onset, slower in progress, less contagious and less "putrid;" and these were named slow nervous fever, adynamic fever, or, from the frequency of abdominal symptoms, gastric fever (typhus abdominalis). In addition miliary fevers and bilious fevers were described, and lastly, fevers lasting less than a week and running a benign course, which were called febricula.

It was the study of morbid anatomy, so strangely neglected before, which led to the breaking up of this heterogeneous group of diseases. In France, which took the lead in pathology and clinical medicine during the first thirty years of the present century, the first and most important discovery was made. It was found that when the bodies of those who died of fever in Paris were opened, the intestines were ulcerated. The true meaning of this fact was not, however, at first understood. Broussais maintained that a "gastro-entérite" was the essential cause of "continued fever" in general; *i. e.* that the general disorder was symptomatic of a local inflammation. Serres and Petit believed that the intestinal lesions were specific, and resulted from the introduction of a poison into the system, and that they were of an eruptive nature, like the pustules of smallpox. Meckel supposed that the soft white "medullary" *plaques* of the diseased intestine were medullary new growths, primary soft cancer of the ileum. Bretonneau, of Tours, noticed in 1826-9 that the ulcers were specially localised in the solitary and the agminated lymph-follicles of the intestine. He introduced the clumsy term dothienentérie (δοθιήν, a pustule, and ἔντερον, intestine) for the disease; it was used by his pupil Trousseau, but never met with general acceptance. Unfortunately, the name proposed by Louis in 1829, *fièvre typhoïde*, was universally adopted in France and in England. The French physicians assumed that the same lesion would be found in every form of fever, whether known as typhus, putrid fever, bilious fever, gastric fever, or jail fever. But when the anatomy of fever came to be investigated in Scotland and in England the bowels were often found to be healthy. For a time the difference was supposed to be accidental, or of little consequence. But gradually it became apparent that two distinct diseases had been confused together. The credit of indicating the clinical distinctions between them must fairly be divided among many observers, of whom Gerhard of Philadelphia (1836), Lombard of Geneva (1836), Shattuck of Boston (1839), and A. P. Stewart in England (1840) deserve mention.* Louis, in the second edition of his work, published in 1841, expressly admitted that his "fièvre typhoïde" and the typhus of English writers were different diseases.

There were, however, many who maintained the opposite view, that typhus fever and typhoid fever were only two forms or local varieties (as botanists would put it) of the same disease, "continued fever." This opinion was maintained by Graves and Stokes in Ireland, by Alison in Scotland, and in the first edition of Watson's Lectures in England. The issue of the controversy still appeared uncertain until, in 1849-51, Dr

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* Dr Stewart, from his comparison of the fever seen in the wards and deadhouse at Glasgow with that seen in the wards and deadhouses of Paris (1836—1839), concluded that the differences between the two diseases were "so marked as to defy misconception, and to enable the observer to form with the utmost precision the diagnosis of the nature of the disease and the lesions to be revealed by dissection."

(afterwards Sir William) Jenner published a series of researches, based upon observations made at the London Fever Hospital. During three years he investigated with great care every instance in which more than one patient was brought from the same house; and he found that the later cases invariably corresponded with the first one, even when both fevers were prevalent in London. All subsequent writers have adopted this doctrine.[*] In fact, each fever was proved to breed true, and they were therefore "specifically" distinct.

It is interesting to inquire whether enteric fever can be recognised in the description given by medical writers in former centuries; and the answer was decidedly in the affirmative.[†] In John Hunter's Museum at the College of Surgeons there are still two preparations showing the characteristic intestinal lesions; and a well-marked case was dissected by Morgagni. The disease is probably identical with the "slow, nervous fever" which Gilchrist, of Dumfries, described in 1734, and which Huxham, in 1738, distinguished from "putrid malignant" fever. In works of an earlier date it must probably be looked for among the "remittent fevers," attributed to malaria. The mistake of confounding enterica with malarial fevers was common in the marshy districts of England up to a recent period; and in India they are even now separated with difficulty.[‡] Lancisi recorded cases of fever at Rome in 1718, with ulceration of the intestines, and identified them with the "hemitritæus," or semitertian ague of Galen. Spigelius, a century beforehand, described cases as common in Italy, with the symptoms and morbid lesions of enterica, and the same had been done by our countryman Thomas Willis in 1859.

Nomenclature.—The name "typhoid fever" is ill-chosen, since the object in giving a title to a disease distinct from typhus, but liable to be mistaken for it, is to mark the difference between them, not the resemblance. Moreover the same word has long been properly applied to a group of symptoms—stupor with muttering delirium and a dry, brown tongue—which are really *typhoid*, since they are like what is seen in typhus. The resemblance of "typhoid fever" to typhus is no greater than to any other specific toxæmia with pyrexia. The name Enteric Fever was a revival of the old terms Febris intestinalis, mesenterica, abdominalis; it was introduced by Ritchie in 1846 and accepted by Gairdner and Tweedie. It has been adopted in the 'Nomenclature of Diseases,' published by the Royal College of Physicians, and is ordered to be used in the official returns made to the Registrar-General.

Distribution.—Enteric fever is endemic in all parts of Europe and Asia, in the United States, and in Australia. It is common in India, in China, and in the tropics generally, and is far from infrequent in Scotland,

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[*] Dr Wilks ('Guy's Hosp. Rep.,' 1855 and 1856), Dr Peacock ('Med. Times and Gaz.,' xiii, 1856), Sir Thomas Watson ('Lectures,' 4th ed., 1857), and Dr Murchison ('Continued Fevers of Great Britain,' 1862) were among the earliest who adopted Jenner's conclusion. Soon after Jenner's researches were published, Dr Austin Flint, in 1852, showed that the same distinction between typhus and enteric fever was manifest from a comparison of clinical and anatomical facts in the United States, thus confirming the opinion of Gerhard and Shattuck.

[†] An interesting example of the possibility of interpreting historical records by the light of modern science is afforded by an admirable essay in which Dr Norman Moore has satisfactorily proved, from the memoranda of Dr Mayerne, physician to James I, that the disease of which Henry, Prince of Wales, died in 1612 was enteric fever.

[‡] A supposed compound disease, due to both malarial and typhoid poison, is still recognised in America, and by some physicians in India, and has been named typho-malarial fever.

Norway and Iceland. It is well known in Brazil and Buenos Ayres, in Egypt and South Africa. In fact, it may be called pandemic in its distribution.

It is doubtful whether enteric fever occurs in animals. "Pig typhoid" has nothing in common with typhoid fever but the name. Mr Bland Sutton communicated to the Pathological Society ('Trans.,' 1885 and 1889) a paper on the occurrence of a disease in monkeys and in beavers, which on anatomical grounds he identified with the typhoid fever of man. The symptoms observed during life were diarrhœa and hæmorrhage. He quoted Serres, who recorded an epidemic of what he regarded as enteric fever among the monkeys in the Jardin des plantes; the symptoms observed were diarrhœa and fever, ending almost always in death.

Clinical course—incubation.—The source of the disease has now been proved to be never by *de novo* development out of filth or anything else, but always from a previous case. The contagion is conveyed by a peculiar bacillus which is as a rule carried in drinking-water to the intestines of the second host. The period of incubation after entrance of the poison appears to vary, but in most cases is more than a week and less than a fortnight. The date at which a patient receives it can seldom be precisely fixed. At Guildford, in 1867, an epidemic was traced to the fact that contaminated water was supplied on a single day, the 17th of August; a large number of cases came under medical observation on the 3rd and the 4th of September, so that, allowance being made for the gradual development of the symptoms, the incubation probably lasted twelve or thirteen days. Instances are recorded in which the disease has shown itself four or five days after the patient has drunk contaminated milk or arrived in an infected locality. Cases of apparently longer incubation occur, and some observers regard the usual incubation period as varying from one to three weeks. Persons sometimes have not fallen ill until three weeks after leaving a place in which there is reason to believe that they must have taken the fever; but the question is at what date the intestinal lesions began, not when the symptoms first appeared. In some cases accurately observed by Quincke, in Switzerland, the incubation lasted from eight to sixteen days.

Onset.—The first day of enteric fever is, as a rule, difficult to fix, for it is usually slow and insidious. The patient feels depressed and weary, and more so every day; he has headache and giddiness, and pains in his back and limbs; he loses appetite, and sometimes vomits. Diarrhœa comes on of itself; or, if he takes an aperient, his bowels remain relaxed. After five or six days he becomes so ill that he has to give up work and take to his bed. In such a case the duration of his illness must be reckoned from the day on which he first felt ailing. Sometimes malaise and other slight symptoms run on for two or three weeks before a definite illness begins, and its subsequent course is not shorter than usual. It is probable that some of these cases begin as ordinary diarrhœa, and that the disordered state of the bowels renders them more obnoxious to the specific contagion.*

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* When a patient was ailing before he fell seriously ill, one must be prepared in case of death to find some of Peyer's patches in an advanced stage of the disease. The late Dr Irvine supposed that in such cases the definite attack is in reality a relapse, preceded by an almost latent primary fever. The objection to this view is that a relapse is almost always slighter and shorter than the primary attack.—C. H. F.

Sometimes, however, enteric fever begins not gradually, but with a rigor and a rapid rise of temperature, so as to simulate typhus or influenza. The writer has noticed this to be more frequent during the last ten years, especially in 1893.

Course.—The *temperature* during the first three or four days of the fever rises in a zigzag fashion: from morning to evening there is an ascent, from evening to morning a fall, much slighter, except in the unusual cases of abrupt onset; so that each evening the thermometer stands higher than on the evening before. It must, however, be borne in mind that the exceptional cases of enterica in which the onset is marked by definite symptoms are those in which the thermometer is most likely to be used during the first two or three days, and the rules Wunderlich laid down are rarely completely observed by this wayward disease.*

Nevertheless the gradual ascent of the temperature with slight morning remissions during the first week is characteristic; and a normal temperature at any part of the day, or a temperature as high as 104° Fahr., are both unusual before the fourth or fifth day. In mild cases the maximum temperature during the course of the fever may be registered before the end of the first week.

The *pulse* is not very rapid during the first week, generally from 100 to 110 a minute, and sometimes less frequent. The pulse-rate varies at different periods of the day, sometimes in correspondence with the fluctuations of the temperature, sometimes independently, and changes of posture affect it much more than in health. Its peculiarity is its soft, compressible character, with a dicrotism which may be so marked that an unskilled observer may actually count it at twice the frequency of the heart's beats. These features are well shown by the sphygmograph.

The *breathing* is more rapid than in health; but unless bronchitis is present the respirations are not quickened in the same ratio as the pulse until hypostatic congestion begins to set in.

The *aspect* of a patient with enteric fever is that of languor and weariness; the face is not dusky, nor is the aspect so stupid as in typhus. There is usually pallor, with a circumscribed pink flush on one or both cheeks, especially towards evening or when food or stimulant has been recently given. The muscular strength gradually fails, and the mind becomes more listless.

The *tongue* is less thickly coated than in Scarlatina, Rheumatism, or Pneumonia, and the fur is often yellowish instead of white. In the course of the second week it sometimes becomes clean, bright red, and smooth, as though glazed. In mild cases it remains moist, but in severe ones it gradually becomes dry and brown, with deep painful transverse fissures. A yellow streak on each side of the dorsum is very frequent.

Anorexia and thirst are common to this and to every febrile complaint; nor is there much distinction in the nausea and headache which are often present at the commencement of the illness.

The skin may either be dry or moist. Profuse sweats are by no means infrequent, especially at night; and are sometimes accompanied by an abundant eruption of sudamina, especially towards the end of the second and in the third week, which ends in desquamation of the chest and abdomen.

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* In 1879 a patient in Guy's Hospital, convalescent from pleuritic effusion, was attacked with enteric fever. He first felt unwell on April 2nd, and his temperature immediately rose from 98·6° to 102·6°; next day he had rigors; on April 4th there were characteristic stools; on April 8th rose-spots appeared.

In 1878 a girl was admitted who had been carefully watched from the third day of her illness by Dr Ingoldby; the morning and evening temperatures were on that day 103·4° and 105·2° respectively; on the fourth day they were 103·4° and 104·2°; on the fifth day 103·4° and 102·3°; on the sixth 103·5° and 104·2°.

The *spleen* becomes enlarged in the course of the first week. Sometimes its edge can be felt below the ribs, particularly if the patient lies over to the right and the ulnar edge of the hand be gently pressed up under the ribs with expiration and the inspiratory fall watched for. Often its extent can only be mapped out by percussion. With resonant lungs and a full tumid abdomen the spleen may be many ounces heavier than natural, without there being any appreciable percussion-dulness over it, so that the absence of a splenic tumour is far from decisive against enteric fever.

Epistaxis is of frequent occurrence, especially soon after the commencement of the disease. It has been known to be so profuse as to be dangerous. Severe headache is a frequent early symptom, and sore throat another; other cases, again, begin with cough or with vomiting.

Exanthem.—Early in the second week, or sometimes at the end of the first, appears the *rose-rash*, the most characteristic symptom of enteric fever. A diffused blush like scarlatina is occasionally noticed two or three days sooner, in the course of the first week, and when attended with a slight sore throat, this has led to a mistaken diagnosis.* The true rose-rash is much less conspicuous, yet the presence of a few small red spots hardly larger than pins' heads is the conclusive sign of enteric fever. No doubt individual papules must be distinguished from fleabites and from inflamed sebaceous glands. They are not petechial, but disappear under pressure; they are pink in colour, rounded not acuminate in shape, discrete in distribution, and usually few in number. Each papule runs a short course of two or three days, and fresh crops appear as the fever goes on. The skin shows white and clear around them, not mottled and dusky as in typhus. The regions in which they are mostly found are the lower part of the chest and the front and sides of the abdomen; they also occur on the back and loins, but a careful physician will seldom be curious on this point. In an epidemic among the French troops at Montpellier (1870-71) Dr George Turner informed the writer that in several cases the rose-spots could only be found on the lumbar region, and Murchison has seen them limited to the back. In exceptional cases they are scattered thickly over the whole of the trunk. The writer has seen them on the neck, on the arms, wrists and hands, and on the ankles, but never on the face.

The date at which they first appear is usually between the seventh and the twelfth day, occasionally as early as the fifth or sixth day of the fever. They are probably never absent throughout the whole course of the disease; but in the case of children one may fail to observe them, although careful search is made every day. They can not only be seen, but felt, as rounded, smooth pimples; they do not grow after they appear, but become paler and flatter until they fade away; they never become petechial, and after death they are invisible. They appear in successive crops, so that although the duration of the rash as a whole may be from one to three weeks, or longer if the disease is protracted, no individual spot remains more than three, four, or five days, sometimes not longer than twenty-four hours. It has sometimes been noticed that they have come out in large numbers after a warm bath, or that they have first shown themselves on a part that has been reddened by mustard; on the other hand, they become paler and less evident after exposure has cooled the surface. A fresh crop of rose-spots marks a relapse.

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* Roseola scarlatiniformis: see "Two Cases of Enteric Fever accompanied by an Erythematous Eruption resembling that of Scarlatina," by Dr Whipham, 'Clin. Trans.,' 1883, xvi, p. 150.

Besides this characteristic rose-rash and the rare early erythema above mentioned, urticaria has been noticed. Sudamina are not infrequent, and when numerous may obscure the pink papules, and remind one that miliary fever was one of the supposed varieties of continued fever. *Taches bleuâtres*, described by French physicians and by Duckworth, are hæmorrhages due to pediculi.

Diarrhœa.—Ulceration of the bowels is constant in enteric fever, but diarrhœa, though the rule, is sometimes completely absent. The proportion of such cases varies in different epidemics and under different treatment; Murchison, as the result of twelve years' experience, placed it at one fifth. The writer has for several months together seen almost every case constipated, and in another local epidemic has seen constant diarrhœa. Constipation is probably more frequent than formerly, and this may be explained by the strict milk regimen now generally enforced. In many cases the bowels cease to be relaxed when the patient has been ill for a few days, or they are first loose during the third or the fourth week. Louis stated, and subsequent experience has confirmed it, that, as a rule, the severity of enteric fever is proportionate to the urgency and persistence of the diarrhœa. But there is no necessary relation between the number of stools and the extent of the peculiar intestinal lesions. It was taught by Addison that diarrhœa was more constantly present when the colon was ulcerated than when the ulcers were confined to the ileum, and the same fact was observed by the late Mr Busk at the Dreadnought Hospital; but the rule is not without exceptions.

In ordinary cases the number of the evacuations is about four to six in the twenty-four hours. The stools have been compared to "pea soup." Their colour is almost like that of yellow ochre, and they are uniform in consistence,—thick, not watery. This appears to depend upon a considerable amount of mucus being mixed up with the fæces. Addison used to lay stress upon their being almost identical with the normal contents of the small intestine, and he supposed that they were hurried on through the colon and rectum, and discharged without having undergone the usual changes there. Albumen is present in considerable quantity in the stools; their reaction is alkaline, and numerous crystals of ammonio-magnesian phosphate can be seen under the microscope.

Since the "pea-soup" stools are not by any means constant in enteric fever, and are sometimes seen where it is absent, there is serious objection to their being called "typhoid stools." Their presence, like other symptoms, is only a help to diagnosis. Blood may often be recognised microscopically or in the form of small clots, even when the patient has been ill less than a week. Abundant intestinal hæmorrhage will be afterwards described as a serious complication.

In doubtful cases Dr John Harley recommends searching during the third week for fragments of sloughs from Peyer's patches. He advises that the stools should be strained through a net, and that the matters it should be washed, and then examined by a lens; flocculent shreds may thus be obtained, in which the remains of intestinal tubules can be plainly recognised. In 1873 a man died in Guy's Hospital from enteric fever, who two days before his death passed a slough an inch and a half long, in which muscular fibres were discerned; at the autopsy the corresponding ulcer was found to be two inches in length, and the peritoneum was exposed in its floor.

Another sign of intestinal disturbance is, in some cases, the production on gentle pressure (which must be applied with extreme caution) of a gurgling sound in the right iliac fossa. There may also be more or less tenderness in this region.

During the second week of the disease, the bowels—as a rule even when there is little or no diarrhœa—become filled with gas, and sometimes the distension is extreme. This *tympanites* or "meteorism" is a grave symptom.

The *urine* is at first scanty and high-coloured, and may remain so throughout, but more often it gradually becomes pale and copious. Towards the end of the third week it often contains albumen, usually in small, and sometimes in large quantity. It is present perhaps in two thirds of the cases, and is probably febrile in character. Hæmaturia is very rare, but may appear quite early in the disease.

The so-called "diazo-reaction" of Ehrlich (published in 1882) is obtained by making a saturated solution of sulphanilic acid in dilute hydrochloric acid, and adding to 200 c.c. of this about 5 c.c. of ·5 per cent. solution of sodium nitrite. This mixture is poured into an equal bulk of urine, neutralised with Liq. Ammoniæ, and a brownish red colour appears with pink froth. The test applies only after the first week of the fever, when diagnosis is in most cases easy from the appearance of the exanthem and other symptoms, and it is present (as Ehrlich found) in measles and in phthisis. Its absence after the first week is good evidence against the diagnosis of enteric fever; and its presence is a corroboration which may sometimes be of practical service.

There is considerable *anæmia* in and after enteric fever, and, it is generally stated, leucocytosis. But careful observations made by Dr Thayer, in Osler's wards in Baltimore, showed that the white corpuscles were not increased in number. He found the anæmia to begin in the third week, and consist in a remarkable fall both in hæmoglobin and corpuscular richness. The latter sank to 1,300,000. The absence of leucocytosis is confirmed by Dr Cabot from the Massachusetts Hospital.

Cerebral symptoms are almost absent in some mild cases of enteric fever. In the course of the second week the headache subsides, and the patient seems to be convalescent. Nor is there always much muscular prostration. Murchison says that forty-four out of one hundred patients under his care were always able to sit up, and to get out to the night-chair; but this should never be allowed, however apparently mild the attack.

In the third week, however, in most cases, and in many much earlier, the patient becomes altogether helpless, and lies upon his back, unable to turn in bed, and with trembling hands and tongue.* Fibrillary "automuscular" contractions are frequently produced by pressure on the pectoral muscles, and, as Dr Money observed, the knee-jerks are exaggerated. In some rare cases the limbs or the trunk become rigid, or the back of the neck is retracted, or there is strabismus, or spasm of the pharynx, or trismus. Murchison's large experience yielded him only six cases of enterica complicated by epileptiform convulsions.

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* Jenner believed that a disproportionate intensity of *tremor*, as compared with other nervous symptoms, is of significance, pointing to the presence of deep ulceration of the intestine, such as is likely to lead to grave perforation or to dangerous hæmorrhage; and Murchison was of the same opinion.

There may be all degrees of *delirium*, from the slightest rambling or moaning during sleep up to the most violent maniacal excitement, lasting for hours together. Active noisy delirium (which is more common in enteric fever than in typhus) is always a dangerous symptom. Sometimes the patient unexpectedly jumps out of bed, and may throw himself out of the window; or if not properly watched he may cut his throat, as in a well-known case of suicide at Newmarket which occurred during delirium at a late period of enteric fever. As Gairdner observes, the patient may lie for days perfectly still and quiet, apparently understanding everything that is said or done, but unable to articulate intelligible replies (κῶμα ἄγρυπνον, *coma vigil*).

The *pupils* in enteric fever are almost always dilated, as Jenner first remarked; but Murchison observed that when there is complete unconsciousness they may be as narrowly contracted as in any case of typhus.

Later course and resolution.—Deafness is common in the third week, and does not appear to be of grave import. It must, however, be distinguished from deafness in one ear, coming on as a sequel of enterica and due to caries of the temporal bone.

Enteric fever very rarely subsides by a crisis.* The rule is that at some period of the disease, frequently about the end of the third week, the morning temperatures begin to fall, while the evening temperatures rise to the same level as before; so that on the daily chart a series of acute zigzags is formed. After a few days these become exaggerated, so as to make a difference of 4° or 6° between the temperatures of a single day; but the absolute height of the evening rise now begins to decline in its turn, so that each day it is about 1·5° or 2° lower than on the previous day; and thus, towards the end of the fourth week, a temperature is reached which is normal or slightly below normal. This *intermittent* type of pyrexia towards the close of the disease is so characteristic that a correct diagnosis has been based upon it alone.

Many cases, however, particularly in children, undergo lysis several days before the end of the third week, some even during the second; while, on the other hand, there are not a few instances in which the fever runs on for four, five, or even six weeks without any definite change. Sir William Jenner once remarked to the writer, "They call it a three weeks' fever; but I never knew a case (in an adult) over in three weeks." In a woman who was under treatment in Guy's Hospital in 1874 the temperature at the expiration of six weeks was still 103·8°, then it fell suddenly by *crisis*, and within two days became normal. It is in such cases that, if death occurs after several weeks' illness, one finds all stages of the lesions in Peyer's patches and solitary glands from early swelling to healing after the sloughs have been cast off. Patients seldom die before the third week, but Trousseau recorded one case fatal on the fifth day. The latter part of the third and the fourth are the most dangerous. Many die at the end of the fourth or early part of the fifth, and many later still, with or without complications.

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* A medical friend of mine, in whom the temperature from morning to evening had been ranging from 101° to 103° with almost absolute regularity, was found, on what was calculated to be about the twentieth evening, to have a temperature of only 101·1°; next morning it was 100°; in the evening 99·3°; on the twenty-second morning it was 98·3°, and for several days afterwards it remained slightly below normal.—C. H. F.

In Dr Moore's work on fevers is a chart of a case of Dr Bewley's which also ended abruptly.

Convalescence.—When the fever has subsided, the pulse sometimes remains quick, but may drop below sixty, and the temperature may be subnormal for several days. This is often a period of much suffering to the patient, who, as his mind becomes clear, complains more and more of weakness and of prostration, and of not being able to find a comfortable posture for his wasted body. The loss of weight during an attack of enterica is considerable, and may amount to twenty pounds or even more.

Occasionally, as Dr F. Shattuck of Boston has remarked, the temperature may keep up when all other symptoms have disappeared, or may rise again without any other sign of a relapse, and in such cases the best treatment is to let the patient get up and take solid food. Convalescence from enteric fever is slow in comparison with that from pneumonia or typhus, even when its course is uninterrupted by a relapse or by any of the accidents that will presently be described as complications and sequelæ. Many weeks elapse before the patient is fit to resume his active duties.

Mode of death in fatal cases.—The immediate causes of death may for convenience be divided into two classes: the general effects of the fever, and occasional complications. The greater nunmber of deaths are due to failure of the heart's action, particularly in the pulmonary circulation. This leads to congestion of the posterior and lower part of the lungs, the most dependent in the supine posture of fever; and the mere length of enterica, as compared with typhus or scarlatina, makes this result more frequent. The hypostatic congestion, or hypostatic pneumonia, as it is called, is not ordinary hepatisation, but a combination of hyperæmia, œdema, and sometimes hæmorrhage, with more or less lobular catarrh and pulmonary collapse. True pneumonic hepatisation is only an occasional complication.

The weakness of the heart is no doubt aggravated by severe and unchecked diarrhœa or by repeated hæmorrhage from the bowels, in addition to the inability of the patient to take sufficient food, and the direct effects of the febrile process on the muscular tissues. Death by failure of the heart and engorgement of the lungs, somewhat vaguely described as *asthenia*, or exhaustion, or "cardiac failure," *i. e.* weakness of the right ventricle, commonly occurs in the third or fourth week, and occasionally later still. The heart may post mortem be found dilated, or the subject of fatty or hyaline degeneration.

Sometimes a condition which might also be described as asthenia supervenes after the temperature has fallen, and the diarrhœa ceased. There is no evidence of serious obstruction of the lung, either in rapid breathing and cyanosis during life, or in pulmonary congestion after death; but the patient, instead of regaining strength, lies in a state of complete prostration with shallow breathing and fluctuating pulse for hours or even days before death. He may, however, rally from this condition and recover after all, or it may return after apparent improvement.

Death from *syncope*, by sudden failure of the left side of the heart, cutting off the supply of blood to the brain and systemic vessels generally, is rare in enteric fever. Dr Cayley has recorded two such cases, and they would no doubt be frequent if we did not feed our patients with extreme care, and prevent them sitting up in bed until convalescence is established. Death from *hyperpyrexia* is a very rare event in enteric fever.

Occasionally patients are cut short by *coma* within the first, and more often in the course of the second or early in the third week. No morbid appearances are discoverable in the brain.

Slight and abbreviated cases.—The symptoms of enterica are sometimes from first to last so slight that there is the greatest difficulty in persuading the patient that he is really ill, and must not go on with his usual work. Such cases are often seen in hospital out-patient practice in the second and third week, and the clumsy but expressive name of *typhus ambulans* has been applied to them. They sometimes last as long as ordinary cases, but the symptoms are so mild, that perhaps the affected intestinal glands may sometimes subside without breach of surface. That this is not always the case is shown by the occasional occurrence of perforation and acute peritonitis, from the floor of a single ulcer giving way. A servant girl was once walking over London Bridge in apparent health. She fell down in acute pain and severe collapse, from which she never rallied. She was carried into Guy's Hospital in a dying state, and it was afterwards found that latent enteric fever had ended by sudden perforation of an ulcer.

Not a few cases of enteric fever, attended with well-marked symptoms, subside in the course of the third week. Probably the intestinal lesions are comparatively slight. But since it is only by the rarest accident that such cases come to an autopsy we cannot be sure of this, for clinical symptoms and anatomical lesions do not always correspond.

Cases which terminate before the sixteenth day are apt to be set down as examples of "simple febricula," or of a non-specific gastric or intestinal catarrh. Yet it is unquestionable that many such cases depend on the infection of enteric fever, and form part of "house epidemics." Jürgensen, for example, mentions an outbreak near Kiel, in which fourteen out of twenty persons had an abortive form of the disease. Moreover, in addition to malaise, headache, and anorexia, many cases present rose-spots, diarrhœa with characteristic stools, and enlargement of the spleen. The spots are said to appear at an unusually early date, sometimes on the second, generally by the fourth or the fifth day. Moreover this abbreviated variety of enteric fever appears to be particularly definite in its onset. In seventy-four of eighty-seven cases collected by Jürgensen it was sudden, and in forty-two it was attended with chilliness or even with a rigor. The temperature generally rises quickly, and it may be 104° by the second or the third day; indeed, Liebermeister says that he has seen cases in which the thermometer rose to 106° in the axilla, and which yet subsided between the fifth and twelfth days. More frequently in these short cases the temperature does not exceed 102°, 101°, or even less. Its subsidence is generally rapid, by *crisis*, without presenting the marked zigzags seen in the far commoner and more severe form of the disease.

In *children*, from five to fifteen (it is rare in infants), enteric fever is often very mild. Most of the slight and short cases just described occur in patients under puberty. A sharp onset is perhaps more frequent and the fever higher in the first week. Vomiting or rigors are often met with, and absence of the exanthem is not so rare as in adults. Even in the that children often "outgrow their strength" after recovering from typhoid fever. Certain cases, moreover, are as severe and protracted as those in adults, and death from perforation is far from unknown in children.

Enteric fever in persons over sixty is very fatal, though the temperature is usually not high nor the early symptoms severe.

Febris enterica sine febre.—It would seem incredible that this disease should ever run its course without pyrexia; but the fact is asserted by competent observers, whose diagnosis was proved by anatomical evidence—Murchison, Cayley, Struve (during the siege of Paris in 1870), and Fräntzel. The cause of the absence of fever is supposed to have been starvation and exhaustion from fatigue and watching.

Complications.—Many, perhaps a third, of patients with enteric fever die from intercurrent local disorders. These complications are more numerous and varied than in any other specific fever.

The most common complication is *Septicæmia*, and it is one of the most serious. Along with the specific bacillus of Eberth the ulcers of the ileum often harbour numerous streptococci and other suppurative microbes which produce a condition of blood-poisoning, and to this much of the typhoid state of the third and fourth weeks is probably due—the continued fever, the rapid pulse, the sweating, and, above all, the weakness of the heart and consequent congestion of the lungs.

Another grave complication is severe *Hæmorrhage* from the bowels. Its frequency is difficult to estimate, because more or less blood may generally be found in the evacuations if looked for from day to day. Murchison says that in 3·77 per cent. of his cases it occurred to the extent of more than six ounces. Liebermeister, at Basle, found it in 7 per cent., Louis, at Paris, in 6·9. As a direct cause of death it was noted seven times (in four men and three women) among 415 cases at Guy's Hospital, according to an analysis made by our late registrar, Dr E. W. Goodall.

The blood may be either bright red or dark, and it may be fluid or partially clotted; if it has been long retained in the intestinal canal it is dark brown like chocolate. Hæmorrhage during the second week is probably due to a general venous oozing from the swollen mucous membrane of the ileum—unless indeed the true date of the commencement of the disease may have been earlier than was supposed. But at later periods the bleeding is often from an artery exposed during the separation of the sloughs. Dr Fagge more than once recorded one particular ulcer deeply blood-stained or with a clot attached to its floor; and Jenner, in a similar case, injected water into the superior mesenteric artery, and found that it escaped from one of the ulcers. In other instances, however, of death after four or six weeks of illness, numerous ulcers are found all equally reddened, or the whole mucous membrane of the last two feet of the ileum intensely congested, soft, and swollen.

When the amount of blood lost is large, the patient exhibits the usual symptoms of profuse hæmorrhage; he becomes blanched and cold, and the pulse is weak and small. The temperature also is suddenly lowered, a point of importance, as it may afford the earliest indication of what has occurred before any of the blood has been discharged.

The part of the bowel which contains the blood has been found dull on percussion by Zulzer and by Moore. According to Liebermeister hæmorrhage diminishes the rapidity of the pulse, and is often attended with a marked alleviation of the cerebral symptoms. But these effects are transitory.

Hæmaturia may be present in addition to intestinal hæmorrhage, and also epistaxis, particularly at the onset or in the first week of an attack of enteric fever.

With regard to the influence of intestinal hæmorrhage on the course of the disease, Graves, and following him Trousseau, thought that it was not unfavourable. It is true that many patients recover; but the statistics of Murchison and Liebermeister show conclusively that the mortality among cases in which hæmorrhage occurs is far higher than the average death-rate of the disease. It is possible that the cases in question are altogether of greater severity than average cases, independently of their being attended with hæmorrhage. The deep ulceration which opens an artery is very likely to extend still further; and thus it is well established that hæmorrhage, when it subsides, is often followed after a few days by fatal peritonitis from perforation of the bowel.

A considerable loss of blood may impair the patient's power of resisting the disease, and weaken the heart. Murchison says that he has repeatedly seen patients who had been doing well die of syncope a few hours after copious hæmorrhage. A case in point occurred at Guy's Hospital in 1879; the patient, a child aged six, went on favourably for a week after the hæmorrhage and then suddenly expired. Dr Goodhart found considerable dilatation and some degree of fatty degeneration of the left ventricle of the heart. The change was only such as is frequent in uncomplicated cases; but the hæmorrhage probably intensified it.

It is worthy of notice that hæmorrhage seems never to afford the earliest clinical indication of an enteric fever hitherto latent, as is often the case with perforation.

A still more dangerous complication of enteric fever is *Perforation* of the bowel, with resulting peritonitis. Its frequency is very great, especially in England. Thus, whereas Liebermeister gives it as the cause of death in 8 per cent. of fatal cases of the disease at Basle, and Hölscher in 5·7 per cent. at Munich, Murchison found it present in nearly 20 per cent. of fatal cases, and in 3 per cent. of all his cases. All writers are agreed that it more often occurs in males than in females, and in seventy-three instances observed by Murchison the proportion was as fifty-one to twenty-two. In the 415 cases analysed for the third edition of this book by Dr Goodall, 22 (*i. e.* 5·3 per cent of the whole number, and about 30 per cent. of the fatal cases) died from perforation, and of these 12 were male and 10 female.

The aperture is sometimes minute and rounded, but in other cases, as Dr Bristowe stated in volume xi of the 'Pathological Transactions,' it is linear, showing that it was due to laceration. It is sometimes directly traceable to disturbance of the bowel during defæcation, by the administration of an enema, or in the act of vomiting. Perforation may also be due to irritation from solid fæcal matters, or to the presence of pent-up gas. Again, in certain cases the gangrenous process affecting a Peyer's patch extends directly through the whole thickness of the bowel, and then a large slough may drop out, leaving a hole of considerable size.

When gas escapes freely into the peritoneum it allows the liver to fall backwards from the ribs, so that the percussion note in the right hypofæcal matter is extravasated, and thus round-worms have been found free in the peritoneal cavity.

The seat of perforation is almost always in the lower end of the ileum within a foot or two of the valve. It is said to be sometimes in the cæcal appendix or in the colon, but this must be extremely rare.

The time at which this terrible complication is more apt to occur is in the third, fourth, or fifth week of the fever. Cases have been recorded in which it has been as early as the eighth, ninth, eleventh, or twelfth day of the patient's apparent illness, but in all probability the disease was really more advanced than this. Perforation is often the cause of death when there has been profuse diarrhœa, and all the symptoms well marked; but it is sometimes the first sign that anything is amiss with the patient, when the course of the disease has been altogether latent. So, also, it may occur at a very advanced stage, when two, or even three months have elapsed; and this may be the case not only when the fever has been unusually protracted, or when there has been a definite relapse, but even when convalescence has apparently been established, and when perfectly formed and healthy fæces have been passed. In two instances that have occurred at Guy's Hospital, each at the end of the sixth week, perforation was distinctly traced to the ingestion of improper food. One patient was seized with pain very soon after eating two raw apples, another while eating watercresses.

The symptoms of perforation are usually those of a sudden attack of acute peritonitis—severe pain, vomiting, feeble pulse, tympanites, and collapse. But it is important to note that in enteric fever cases of perforative peritonitis may begin insidiously and remain unnoticed. In a case observed in 1864 at Guy's Hospital, the chief sign of an unfavourable turn in the patient's illness was his sudden refusal to take food, after which he died in a few hours. Perforation and consequent peritonitis are often found at a *post-mortem* examination when they had not been suspected. Dr Finney, in a communication to the Johns Hopkins Medical Society of Baltimore (May, 1897), states that sudden and rapid increase of leucocytes is a symptom of perforation. In three cases their number was raised from 8300 to 24,000, 6500 to 10,600, and 3000 to 16,400.

It has been questioned whether recovery after this complication is possible. The doubt lies in the difficulty of determining whether there is an actual perforation in any given case of peritonitis; but several instances have been recorded in which, death having occurred from some other cause, a perforation has been found sealed up by adhesions, or at least closed off from the general peritoneal cavity. In other cases a circumscribed abscess has formed, which has discharged itself either externally or into the bowel.

The following case appears to show that recovery is possible after perforation, but happily the only absolute proof of the diagnosis was wanting.

A boy of ten lay for many weeks in Philip Ward under the writer's care during the winter of 1888–9, suffering from a severe attack of enteric fever. One morning in the fourth week there were all the signs of perforation—sudden pain, meteorismus, and collapse, with thready pulse, sighing respiration, and extreme local tenderness. I saw him within an hour, and feel no doubt that perforation had taken place. A hopeless prognosis was given, but he was treated by full doses of laudanum and frequent small doses of milk and brandy, without any other food, while the temperature, which had fallen very low, was restored and sedulously maintained.

For several days he hovered between life and death. At length the pulse began to improve, the temperature rose again, the tympanites diminished, and very gradually he returned to a state of fever, with great exhaustion and extreme emaciation, but without symptoms of general peritonitis. He slowly but perfectly recovered, and went out after thirteen weeks' stay in the hospital, as rosy and much fatter than I ever saw a boy before.

Acute *peritonitis* has been known to occur in enterica apart from perforation of the gut. Occasionally the mere spreading of a putrid inflammation to the serous surface may light up peritonitis without any actual perforation taking place. It may be set up by sloughing of a swollen mesenteric gland, as in a case recorded by Jenner, in which recovery took place, so that the real nature of the complication would not have been known if the patient had not afterwards died of erysipelas. It has also sometimes been due to extension from an inflamed and ulcerated gallbladder, or from a suppurating embolus in the spleen.*

Embolism of the spleen is not infrequently found after death from enteric fever. It is doubtless the result of plugging of branches of the splenic artery with portions of clot that had formed in the almost stagnant blood in the dilated left cavities of the heart. In the same way *cerebral embolism* causing hemiplegia, with or without aphasia, may arise during the course of the disease, or after convalescence has occurred.

Other complications of enteric fever have their seat in the respiratory organs. A certain degree of *bronchitis* is almost always present, sometimes from the first, and sometimes it comes on so severely in the second or third week as to be the most serious symptom of the disease.

Apart from the hypostatic broncho-pneumonia already mentioned as a frequent occurrence, lobar *pneumonia* with true hepatisation is happily not frequent, though more so than in typhus. It is a very grave complication, and sometimes passes on to gangrene. Pleurisy, too, is often seen, sometimes very early in the fever, and may lead to empyema.

Ulceration of the *larynx* is present in a considerable number of cases, but it seldom or never produces symptoms during life, and therefore need only be described among the anatomical lesions found after death.

Another occasional complication of enteric fever is *parotitis*. It sometimes affects one side, sometimes both. It may either subside after a week or two, or it may lead to suppuration, with extensive brawny induration and purulent infiltration of the side of the neck as far as the sternomastoid muscle.

The occurrence of enteric fever often leads to the reopening of fistulæ which had healed up, with consequent *necrosis* of extensive portions of bone. Apart from previous disease, periostitis is not an uncommon sequel: according to Paget it most frequently affects the tibia, next the femur, and then the ulna and cranium. In patients who have been suffering from soft chancres he has seen wide-spreading gangrene when they were attacked by enteric fever. In two cases at Guy's Hospital an ordinary gonorrhœa led to sloughing of the penis or the scrotum under similar circumstances.

Lastly, we have repeatedly observed renal complications of enteric fever which do not seem to be common, but which have more than once led to a fatal result, and are therefore of practical importance.

Among our 415 cases of the past ten years, two (both in women) were fatal from *suppuration of the kidneys*, and two others from the same cause had been previously recorded. In two other cases there was *cystitis*, sometimes perhaps caused by retention of urine, but this condition, with the attendant dangers of catheterism, is less common in women than in men. In one of these cases (November, 1888) we found the bladder and uterus normal; and in another (Philip Ward, May, 1888) there was no retention of urine, no stricture, and no pyelitis.

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* A patient of the writer's, only eleven years old, died from acute peritonitis with symptoms like those of perforation, but after death none of the above causes were present (October 30th, 1894).

In two women patients acute *tubular nephritis* was found after death, and in one of them, who was under the writer's care in August, 1886, this appeared during life to be the fatal complication. In a third case the patient, a boy of fourteen, happened to be the son of one of the women who died from suppurative nephritis in the hospital a week before (November, 1888). Mother and son had come in with typhoid fever, and both died with uræmic symptoms but from different renal lesions, for in the case of the latter there was anasarca and severe diarrhœa, and acute tubal nephritis with catarrhal colitis were found after death.

Co-existence of other fevers.—Specific fevers do not exclude one another. Patients with enteric fever used to catch typhus, and a case of the two diseases coinciding was recorded in 1888 by Dr C. J. Nixon ('Dublin Journal of Medical Science,' vol. lxxxvii). Murchison saw Enterica with Scarlatina, A. P. Stewart with Variola, and the writer has seen Rheumatic and Enteric Fevers in the same patient.

Sequelæ.—Some of the above described complications of enteric fever may not appear until the patient is already convalescent. It is therefore impossible to draw a fixed line between them and the sequelæ of the disease. What justifies the distinction and makes it useful in practice is that the latter often run on for many weeks, and are the sole obstacle to the restoration of health.

This is the case with *bedsores*, which are apt to form over the sacrum and hips unless great care is taken to prevent them, and which sometimes destroy life by exhaustion, or indirectly by setting up pyæmia.

Another sequel is *thrombosis* of the femoral vein, with the attendant liability to pulmonary embolism.

In some cases enteric fever is followed by *marasmus*. Murchison says that there is not always a repugnance to food; the patient may eat well, but what he takes is not assimilated, and slight errors in diet cause flatulence and diarrhœa. Yet the temperature is normal or below normal, and if death occurs, perhaps after many months, nothing can be discovered except an unusually smooth appearance of the mucous membrane of the ileum, and a shrivelled condition of the mesenteric glands.*

Most writers speak of pulmonary *phthisis* as frequently occurring after enteric fever. But it is remarkable that after searching the records of *post-mortem* examinations at Guy's Hospital, Dr Fagge failed to find a single case in point, nor can the present writer recall a clear instance of this sequence.

Paraplegia has been recorded by Nothnagel and others as an occasional sequel. It is uncertain whether the lesion in these cases is in the cord or in the peripheral nerves.

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* When diarrhœa has persisted after the subsidence of the fever, it has been supposed that the ulcers have remained unhealed. But although Murchison supports this statement with the weight of his authority, I am not sure whether it rests on strict pathological evidence. Dr Wilks long ago taught me to doubt its accuracy, and I have never myself met with any example of it. I remember one case in which a patient was admitted into Guy's Hospital whose sole complaint was pain in the right iliac fossa, which appeared to have been left behind by an attack of enteric fever; after a few weeks this pain subsided. —P. H. P.-S.

Relapses.—Even where there are no sequelæ, the return of the temperature to normal is not always followed by convalescence; enteric fever is apt to relapse. The frequency of relapses seems to differ in different places: at Basle, Liebermeister met with it in 8·6 per cent. of 1743 cases; Murchison says that in the London Fever Hospital it was observed in only 3 per cent. of 2591 cases; Eichhorst, at Zurich, records 4·2 per cent. of 666 cases; but Maclagan, at Dundee, observed it in 13 among 128 cases, and F. C. Shattuck, at Boston, in 21 among 129. The figures given by other writers vary from 11 to 1·4 per cent. In Dublin relapses seem to be very rare. Among our 415 cases from 1879 to 1888 at Guy's Hospital there were 46 relapses, *i. e.* about 11 per cent.

As a rule, there is an interval of some days between the termination of the first and the beginning of the second attack. Murchison stated eleven days as the average; Liebermeister found that among 111 cases of relapse the interval was less than four days in twenty-seven, from five to seven days in seventeen, from eight to fourteen days in thirty-five, and still longer in thirty-two. Dr Moore records one of apparently twenty-four days. The late Dr Irvine, in the 'Medical Times and Gazette' for 1879, maintained that the average interval is not longer than five days. He believed that a relapse sometimes begins before the original illness has come to an end. However this may be, it is certain that a relapse may come on immediately after the subsidence of the primary attack, so that the chart shows no complete twenty-four hours' interval of apyrexia.

It is not very uncommon for the termination of a relapse to be followed after a second interval by a second relapse, and this, again, may be succeeded by a third, and even by a fourth. There is scarcely a more important use of the thermometer in clinical practice than in the detection of these secondary attacks of fever. For the patient may be unaware that anything is amiss, and yet to allow him to go about or to return to ordinary diet is to expose him to fearful risk. Thus at Guy's Hospital, in 1876, a man was apparently going on favourably through convalescence, when on the twenty-ninth day his temperature was found to be 104·2°; he looked ill, though he declared that he did not feel so; and a few days later he died of perforation of the intestine. In other instances the thermometer indicates but a very moderate degree of fever, which ranges from 100° to 101° or 102°, but nevertheless takes a perfectly typical course. The duration of a relapse is almost always shorter than that of the original attack. Murchison found it to be on an average sixteen days. Dr Irvine maintained that it is as a rule twenty or twenty-one days. Cases in which it appears to be longer he explained by the hypothesis of an intercurrent second relapse, interrupting the middle of the first relapse, and subsequently running a regular course. According to this observer the temperature in a relapse generally rises pretty steadily, until on the fifth day it attains its maximum; it remains high until the ninth or the tenth day, when it undergoes a marked fall of from 2° to 6°; directly afterwards, however, it rises again, but it henceforth shows daily remissions, which at length bring it to an end.

It is now well ascertained that relapses of enteric fever are attended with a renewal of the intestinal lesions, although Trousseau maintained the contrary. Dr Fagge recorded nine cases fatal during relapse at Guy's

Hospital during twenty-two years, and in every one of them recent morbid changes were found. As a rule, some Peyer's patches are in a state of early swelling, in others are seen partly formed sloughs, while others again show clean ulcers or cicatrices; but in one instance the floors of the patches were found smooth and bare, while their edges showed the pink tumefaction of recommencing disease.

The symptoms of a relapse do not differ from those of a primary attack, but they are, as a rule, less severe, and run a shorter course. Rose-spots, as Murchison remarked, appear somewhat earlier; in twenty-two out of thirty-eight of his cases they were visible on the third, the fourth, or the fifth day. The patient is often delirious and insensible; and, considering the weakness resulting from his first illness, one is surprised that he does not more often succumb. As a fact, however, relapses are seldom fatal, except by perforation. The only one of forty-six relapses at Guy's Hospital which ended ill during ten years (subsequent to the twenty-two years mentioned above) was one of perforation.

The cause of the liability of enteric fever to relapse is still imperfectly understood. It cannot be due to a fresh infection with the specific poison from without, for the patient is often far removed from the original source of his disease, and little exposed to reinfection from his own excreta.

"Very often the time of the relapse is when he has just begun to take solid food, or when he has once or twice been out of bed. But other instances cannot be thus accounted for; and in any case such an apparent exciting cause can only be regarded as setting up the symptoms of a morbid process which would otherwise have remained latent; since, if the patient happens to die a few days later, the intestinal lesions are found to be so far advanced that they clearly must have begun before the obvious relapse set in. For instance, in 1876 a case ended fatally on the sixth day of relapse, and several of the Peyer's patches had already ochrey yellow centres. Such cases probably afford the key to the whole question. In August, 1861, a man was admitted into Guy's Hospital with bronchitis and emphysema. A month later he was attacked with enteric fever, which ran a regular course and from which he recovered, so as to be able to sit up. But early in October the weather became very cold, whereupon his bronchitic symptoms underwent rapid aggravation, and in a few days they proved fatal. At the autopsy the lower Peyer's patches were found to be roughened and flocculent, as if sloughs had separated from them; but high up in the ileum one or two patches were still somewhat raised, and several solitary follicles showed an early stage of the affection. This case seems to prove that after an attack of enteric fever has to all appearances passed off, intestinal lesions may smoulder on without giving rise to any symptoms."—(C. H. F.)

Dr Maclagan some years ago suggested that sloughs thrown off from the patches first diseased might perhaps infect other patches in their turn. Such an hypothesis is difficult either to prove or to disprove. But with the analogy of spirillum fever to guide us, we may suppose that the relapse of enteric fever is due to a reinfection of the blood with the specific bacillus, derived from patches recently diseased, or possibly, as Dr Hudson thought, from the spleen.

Protection.—Notwithstanding the phenomena of relapse, there is very general immunity from a second attack of the disease among those who have once fully recovered. In our 415 cases there was only one of a second attack, and that happened five months after the first; at the autopsy there was a perforation, and beside the recent ulcers, scars were seen from the previous attack.

Murchison quotes an observation made by Gendron and Piedvache. that after an interval of many years a particular house or locality became for the second time the seat of an outbreak, whereupon it spared those who had previously suffered, but attacked almost every one else. Murchison, however, had himself met with several cases of a second attack of enterica, and he cites others.

Diagnosis.—The diagnosis of enteric fever is often perfectly easy, but it may be of the utmost difficulty. In some cases a single examination of the patient may enable one to speak positively of the nature of his illness, especially in the second or third week; in others the most careful clinical observation from day to day may leave one still in doubt.

At its commencement the disease can never be determined with certainty, although strong suspicion may be excited. The most common mistake is to call the illness a "bilious attack;" and great harm is often done by the aperient dose which follows. The known fact that the onset of enteric fever is generally insidious is apt, when it begins suddenly, to suggest the idea that the case is one of some exanthem, of influenza, or of pneumonia. Headache is almost constant, while rigors and vomiting, nasal catarrh and sneezing are rare, and enterica is very seldom attended with herpes of the lips. Cases have before now been at first set down to mania, to bronchitis, and to gastric catarrh.

The occasional early roseola should never be mistaken for scarlet fever, for it does not appear until the fourth or fifth day. Murchison knew of a copious eruption of rose-spots being attributed to smallpox, in spite of their late appearance and very different distribution.

The mulberry rash of *typhus*, with its petechiæ, its dull diffused mottling, its earlier advent, and its much wider and more abundant distribution, is so different from the scanty crops of rose-spots on the clear skin of the tumid abdomen, that since the distinction between typhus and enteric fever was established mistakes between them are very rare. It must, however, be remembered that typhus is sometimes accompanied by diarrhœa and enterica by constipation.

Even when no rose-spots can be found, one can often feel confident of the nature of the disease from the course of the pyrexia, the presence of an enlarged spleen, and the characteristic appearance of the stools. But in most cases one's opinion is mainly founded upon negative considerations, and, as Liebermeister remarks, he who is most sensible of his liability to errors is least likely to commit them.

About the end of the first week (that is, within two or three days of the time when the case generally comes under medical observation) a positive diagnosis can, as a rule, be given.

The diseases which are liable to be confused with enteric fever without eruption, or before it has appeared, or when it has been overlooked, may be divided into two groups.

First come certain general diseases. Of these the most important in many countries, although not in England, is *ague*. In India and many parts of America the difficulty of distinguishing the remittent forms of malarial poisoning from enteric fever, whether in individual cases or throughout a district, is so great that, unless opportunities should arise for making autopsies, mistakes seem almost inevitable; and probably this has led to the hypothesis of a "mixed" form of "typho-malarial fever." The recognition of Laveran's protozoon in the blood-discs of malarial fever is most important from this point of view, and decides cases which have puzzled experienced physicians.

In this country the most important disease simulating enteric fever is *general miliary tuberculosis*, without marked symptoms indicating localisation in any particular organ. But although it is quite true that at an early period of a case it is often impossible to say whether a patient is entering upon one or other of these diseases, yet Dr Fagge found that the records at Guy's Hospital showed very few, if any, fatal cases in which a correct diagnosis has not been made. It has recently been the writer's fate to supply this deficiency. A boy was ill of enteric fever, which was without diarrhœa or rash, and accompanied by severe broncho-pneumonia. It ran a protracted course, with cyanosis and high irregular temperature, and we regarded it as tuberculosis, not enterica, up to the time of death.[*]

Another general disease that in temperate climates is often mistaken for enteric fever (or enterica for it) is *pyæmia*, when its starting-point is deep-seated, and when it affects, not joints, but internal organs. Murchison saw several cases of pyæmia from caries of the petrous bone, of which the course was very like that of enteric fever. At Guy's Hospital, in two instances at least, this mistake was actually made. In each of them the source of the mischief was latent disease of the lumbar or the dorsal vertebræ, there being secondary abscesses in the lungs, the kidneys, and the heart. The variations of temperature are in pyæmia always much greater, and not infrequently touch or fall below the normal, a very rare event in enterica except from severe hæmorrhage or from perforation.

Ulcerative endocarditis, with the consequent internal arterial pyæmia, has often been mistaken for enteric fever; the discovery of a cardiac murmur should in most cases suffice to prevent this error, but in exceptional cases the heart-sounds remain normal. Moreover a patient, already the subject of valvular disease, might fall ill with pyrexia and enlarged spleen, which might be due either to enteric fever or to septic endocarditis. Again, enteric fever may be followed by endocarditis.

Trichiniasis has often been set down as enteric fever by those who have not seen it before; but with due care this error might probably be avoided.

Before the clinical thermometer was in use, enteric fever was sometimes mistaken for hysteria; in a diabetic patient it has been supposed to be diabetic coma; and in a subject of lead poisoning, uræmia, from granular disease of the kidneys, has been mistaken for enteric fever.

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[*] A remarkable instance of persistent obscurity is recorded by Senator in the 'Berlin. klin. Wochenschrift' for 1881. A man aged forty-two was admitted into hospital on October 25th, 1880. He had not been quite well since the beginning of September, but his illness had been more marked for about ten days before his admission. His pulse was 80 to 84. His temperature was 100·4°, and for some days it ranged from 101° in the morning to 103° in the evening very regularly. On October 29th the spleen was found to be enlarged, and on October 31st distinct rose-spots appeared, and fresh ones again on the following day. On November 1st and 2nd there was repeated epistaxis. The pulse, still 80 to the minute, was now plainly dicrotic. Signs of bronchial catarrh appeared, and increased greatly up to November 11th. The bowels were open without diarrhœa. On November 21st suppuration of the left parotid occurred, with discharge of pus through Steno's duct. The diagnosis, about which up to this time there had been slight doubts, was now finally given in favour of enteric fever. Yet, after the patient's death, which occurred on November 22nd, miliary tubercles were found in the lungs and in other organs, while the appearances characteristic of enteric fever were altogether wanting. The ophthalmoscope had been used with negative results. A correct diagnosis of this case was surely impossible.

Again, many local diseases may be overlooked if a diagnosis of enteric fever be rashly made in the absence of rose-spots.

Among these is *tuberculous meningitis*, which in the case of children is often surprisingly like enteric fever in its symptoms. Vomiting is a frequent symptom in this disease and is rare in enteric fever, while constipation is the rule in the former, the exception in the latter. In cerebral disorders generally the abdomen is flat or hollow, while in typhoid fever it is more or less distended; in certain stages of meningitis the pupils are contracted and the pulse is slow while pyrexia is still present; but in enterica the pupils are dilated even in coma, and the pulse is frequent except during collapse. A point insisted on by Jenner is that in enteric fever headache ceases before delirium begins; the two symptoms are not present simultaneously as in meningitis. Nevertheless the pulse, the pupils, the shape of the abdomen, the state of the bowels, may each and all fail as points of diagnosis. Even optic neuritis may be present, and the disease be fever and not meningitis.

Among thoracic affections, *tuberculosis of the lungs* must especially be borne in mind. Enteric fever is often accompanied with bronchitis; and even when miliary tuberculosis produces marked thoracic symptoms it may be difficult to be sure that they are not due to this complication of fever. Formerly *acute phthisis*, causing a rapidly spreading consolidation of one or both lungs, was sometimes mistaken for typhoid fever; and a similar error was sometimes made with regard to *pneumonia*, when there was no pain in the chest, nor cough, nor expectoration. Even now, although the routine use of the stethoscope keeps us from overlooking the presence of a pulmonary lesion, we may sometimes be in doubt whether it is the primary disease or merely a complication of fever.

Various abdominal affections are liable to be confounded at the bedside with enteric fever; most frequently, perhaps, *tuberculous peritonitis*. In the absence of the positive signs of this affection, its diagnosis from enteric fever may be impossible. We have had more than one case in which the nature has still remained doubtful after several weeks of illness. Again, two or three instances have also occurred at Guy's Hospital in which *acute diffused colitis* has been thought to be enteric fever.

Another disease sometimes mistaken for enterica is *typhlitis*, *i.e.* inflammation of the appendix cæci (or, to use the barbarous modern term, "appendicitis"). The course of pyrexia would soon decide the question if the local symptoms were equivocal, and the treatment would not be different in either case.

Two cases of abscess of the *liver* (one secondary to colitis, the other to suppuration in the broad ligament of the uterus) were once for a few days regarded as probably cases of early enteric fever.

Finally, it is important to remember that some patients will not complain of *affections of the genito-urinary organs* severe enough to cause constitutional disturbance. Sir William Gull used to speak of cases of extravasation of urine to which he had been called as to typhoid fever, on account of the stupor, muttering delirium, and dry brown tongue. Dr Fagge was once asked to see a servant girl who had been suffering from febrile symptoms, with a very quick pulse. It was not until she had been sent home that she mentioned to her mother that she had severe pain in micturition. There was then found intense "diphtheritic" inflammation of the labia, and she narrowly escaped with her life.

In these cases, as in tuberculosis, pyæmia, and septic endocarditis, the condition is "typhoid" and it is "fever," but it is not due to the specific contagion of enterica.

Beside the special points of diagnosis just described, and the observation of the course and progress of the case—particularly the range of temperature, the exanthem, the spleen, and the stools,—recent investigations have added two fresh aids in the diagnosis of enteric fever.

One is the "diazo-reaction" of the urine, first described by Ehrlich in 1882 (*vide supra*, p. 118).

The other is a peculiar reaction of the blood. It was described in the course of the same year, 1896, by Pfeiffer,[*] Knoll, and Grüber[†] in Germany, by Widal in France,[‡] and in England by H. E. Durham[§] and Grünbaum,[‖] both of them pupils of Prof. Max Grüber of Vienna, and following up his and Pfeiffer's observations. The best account of it in English is given by Dr Delépine, of Owens College, Manchester ('Medical Chronicle,' October, 1896, and 'Lancet,' December, 1896, pp. 1587 and 1665). Pfeiffer's plan was to identify typhoid bacilli by means of the serum of immunised animals, and from this arose the converse test of the reaction of the serum of febrile patients to typhoid bacilli. When a neutral peptone broth is inoculated with Eberth's bacillus, it is rendered turbid by the growth of the microphyte in the medium. If, however, the broth has previously been mixed with serum from the blood of an animal rendered immune to enteric fever by repeated injections of the poison in increasing doses, the bacilli will not multiply, but will aggregate in clumps, and then sink to the bottom of the flask, leaving the serum more or less completely clear above. Now, if instead of serum in bulk, obtained by venesection from an immunised animal, we repeat the experiment on a small scale, using a drop of blood from a patient (Durham recommends pricking the ear), in the third week (or later) of enteric fever, the clumping reaction is obtained; if the blood be taken from a healthy person, or a patient suffering from septicæmia, tuberculosis, or typhlitis, the typhoid bacilli multiply, and the serum becomes turbid by their growth.

The practical value of the test is maintained by Widal, and with some reservations by Durham.[¶] It has been carefully examined by Delépine and Sidebotham at Manchester,[**] by Pakes at Guy's Hospital,[††] by G. R. Murray at Newcastle,[‡‡] and by several observers in America,[§§] and the result is decidedly favourable. The writer has found this test more constant than the diazo-reaction, the drawback being that it is not available in the early stage of enteric fever when diagnosis is most difficult.

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[*] 'Deutsche med. Wochenschrift,' March 19th, 1896.

[†] Grüber u. Durham, 'Münchener klin. Wochenschrift,' March 31st, 1896.

[‡] June 26th, 1896, published in 'Presse médicale,' July 29th, 1896; 'Lancet,' Nov. 14th, 1896.

[§] Durham, 'Proc. Royal Soc.,' Jan. 3rd, 1896; 'Journ. of Pathology,' July, 1896; and 'Lancet,' ii, p. 1746 (Dec. 19th, 1896).

[‖] 'Lancet,' Sept. 19th and Dec. 19th, 1896.

[¶] 'Lancet,' Dec. 19th, 1896.

[**] 'Lancet,' Dec. 12th, 1896.

[††] 'Lancet,' May 29th, 1897.

[‡‡] Ibid., p. 1464.

[§§] See papers by Dr. Charles L. Greene in the 'Medical Record,' Nov. 14th and Dec. 5th, 1896, and by Dr Fison in the 'Brit. Med. Journal' for 1897.

Anatomy.—In certain specific fevers the "general" or "constitutional" character, which is one of the notes of the whole group, is unmodified by any predilection of the contagion for fixing itself in one part more than another. Blood and lymph, solids and liquids, bones and viscera, all are alike invaded by the poison, and its effects only differ in accordance with the functions of the several organs.

But in most specific fevers we find, in addition to the universal "intoxication" with the poison, that it fixes itself peculiarly in certain *foci*, as we may call them, where it produces definite local lesions. Thus measles, we shall see, particularly affects the respiratory mucous membranes, and scarlet fever the throat. But nowhere is this localisation of the disease so remarkable as in enteric fever. In fact, so striking and clinically important are the local lesions, that it was possible for Broussais and his school to regard these lesions as the primary disease, and the fever as merely a symptomatic result. The "typhoid deposits," as they used to be called, in the intestine are to be regarded as *infective granulomata*, produced, like the nodules of tubercle and leprosy, by the local action of specific bacilli (cf. p. 60).

These characteristic lesions of enterica are situated in the lymphatic follicles of the *intestine*, both agminated and solitary. Peyer's patches first become injected and swollen, so that they project decidedly above the level of the surrounding mucous membrane. As a rule they rise to the height of 3—5 mm.; but in some exceptional cases, according to Hoffmann, they may be two centimetres thick. Louis distinguished *plaques molles* from *plaques dures;* the difference between them is, however, merely one of degree, the "hard plates" being those in which the change is most rapid and intense. Red and injected follicles and patches are only seen in the rare cases which prove fatal within the first week of the disease, or in the recent extension of the process seen after death during a relapse. We infer that after a few days the redness passes off. The diseased follicles, as usually seen after death, are pale, so white that in the early days of morbid anatomy they were described as encephaloid cancer of the bowel. The microscope shows that there is abundant formation of new cells, which have large nuclei, and often are found undergoing fission. These cells, besides distending the lymph-patches and follicles, infiltrate the intervening fibrous septa, and may spread into the mucous membrane above, and into the interstices of the muscular coat and subserous tissue beneath.* It is not uncommon for adjacent Peyer's patches to become fused together by an extension of the morbid growth, which thus may affect the whole circumference of the lower part of the ileum for some inches above the valve.

The next stage is ulceration. As a rule, the whole of the infiltrated tissue, or at least the greater part, dies in a mass. It then forms a soft, shreddy, flocculent slough, which is of a bright ochre-yellow colour, no doubt from the bilirubin of the fæces. The slough is soon detached, either entire or in fragments, so as to expose the floor of an ulcer, in which the transverse muscular fibres are often plainly visible, while the gut is so thinned that it is translucent when held up to the light. The muscular coat itself may undergo more or less destruction, so as to lay bare the subserous tissue—to the imminent risk of perforation into the peritoneal cavity. The edges of the ulcer are thin and soft, and when examined under water they are seen to be slightly undermined. The shape of each ulcer corresponds with its origin: if it began in a solitary follicle, it is small and rounded; if in a Peyer's patch, it is oval in the direction of the axis of the intestine. In the latter case, too, its position is always remote from the line of attachment of the mesentery. In these respects, as well as in the characters of the floor and edges, the ulcers of enteric fever differ from those of tubercle. In an advanced stage, however, they may be found elongated transversely to the axis of the intestine, owing to secondary ulceration.

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* I have never seen these cells forming grey granules on the peritoneal surface, as described by Hoffmann.—C. H. F.

The suppurating ulcers were compared by Italian writers in the seventeenth century to the effect of burns on the skin; and by Willis (the great anatomist), in the same period, to the pustules and ulcers of small-pox, a comparison often made since.

Gradually the floor of each ulcer becomes covered with a thin grey layer of granulation tissue, and slowly heals by the growth of mucous membrane from the edges towards the centre. The process probably takes about six weeks on the average. At Guy's Hospital five cases are recorded in which death occurred from some other disease within a few months after recovery from enteric fever: in two of them the patches were of a blackish colour, with slaty margins; but in the others the cicatrices seem to have been white, and in one of them it was only on close examination that a slight unevenness of surface could be recognised. There is never any thickening or puckering of the affected tissues after enteric ulceration, so that it can scarcely lead to narrowing of the gut.

All the lymph-follicles of the intestines are by no means equally affected. In many cases the solitary follicles entirely escape; in many others those of the ileum are alone attacked; and when those of the large intestine share in the process, it is often only in the cæcum, or ascending colon; it is but rarely that the morbid change extends to the rectum. The process seems, in fact, to spread from the ileo-cæcal valve as from a starting-point. Precisely the same thing is true of Peyer's patches. As a rule, the lesion is limited to patches within two or three feet of the valve, and sometimes it does not reach beyond a few inches. The jejunum is not infrequently affected, but seldom if ever the duodenum. Cases of typhoid ulcer in the appendix and in an abnormal diverticulum of the ileum are recorded by Wilks and Moxon. In very exceptional instances the agminated patches of Peyer remain unaffected, and the solitary follicles bear the whole brunt of the disease.*

The morbid process is almost always farthest advanced in the follicles close to the valve; and very often every stage is present—clean ulcers below, then ulcers containing scattered shreds of slough, next those in which sloughs have just been formed, and lastly the patches which are only swollen and excoriated. But the progression is not perfectly regular. These differences depend upon the fact that the follicles are attacked, not simultaneously, but in succession.[*]

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* This apparent caprice of distribution in different cases cannot but suggest the inquiry whether there may not be some in which no glands suffer at all, so that, if the nature of the disease were otherwise doubtful, the criterion afforded by morbid anatomy would fail. The nearest approach to this with which I am acquainted is afforded by a specimen which I showed at one of the meetings of the Pathological Society in 1875. The only lesions in the intestine were the following:—"One ill-defined purplish-red patch, of about the size of a shilling, situated a foot above the valve; and a little higher up another patch, presenting similar characters, except that in its centre there was a darker spot the size of a pea, with a breach of surface, visible only when it was examined under water." I think it is by no means unlikely that in mild cases of enteric fever, such as could never prove fatal except by some accident, the intestinal lesions are often very slight, and may possibly in rare cases be altogether absent.—C. H. F.

Sometimes, instead of sloughing or ulcerating, the swollen patches entirely subside, as was long ago ascertained by Chomel and Louis. They supposed that the follicles underwent softening and ruptured, so as to allow the softened morbid material to escape into the bowel. Hoffmann confirms this account, and adds that the follicles become stained in consequence of a little hæmorrhage, so that the Peyer's patches acquire a dotted appearance—the *état pointillé* of French writers, compared to a shaven beard. This condition, however, is not uncommon in those who have died from other causes and at all ages, and is not peculiar to enteric fever. Subsidence of the swollen glands may also take place in a different manner, namely, by the cell growth in the follicles disintegrating, and undergoing absorption like an inflammatory exudation.[†] As a rule absorption is found to have taken place in certain patches only, or even in parts of them, while elsewhere there are sloughs or ulcers. Thus in 1862 Dr Wilks examined a case in which, while the solitary follicles had each a depression in its centre, some patches appeared to be in a state of retrogression, for they were only slightly raised and nodular in parts.

It is a matter of some practical importance to determine as accurately as possible the dates at which, reckoning from the beginning of the fever, one may expect to meet with the several stages of the intestinal lesion. According to Liebermeister, the first week is occupied by swelling and infiltration of the intestinal lymph-glands; during the second week they either slough or begin to subside; in the third week any sloughs that may have formed become detached, so that by the twenty-first day all the ulcers have clean floors; during the fourth week they begin to heal, but the process is often not complete until much later. This statement is easy to remember, and in many cases it may be true, but it certainly is not so universally applicable as to enable us, from the anatomical appearances in a given case, to state positively the duration of the patient's illness. According to Trousseau the swelling of the intestinal glands does not begin to appear until the fourth or fifth day; according to Chomel and Louis not until the seventh or eighth; but Bristowe and Hoffmann found them enlarged in cases fatal on the fifth and fourth days respectively.

Murchison relates one instance, that of a girl who died forty-seven hours after being suddenly attacked with vomiting and fever, in which the solitary follicles were of the size of hemp-seeds or split peas, Peyer's patches being also swollen. Is it not, however, probable that the disease in that case began earlier than its symptoms? In 1871 a man died in Guy's Hospital, who had been definitely attacked twelve days before his death with shivering and aching pains in his limbs. At the autopsy his intestine contained numerous ulcers, all with clean floors except one, to which a few fragments of slough still adhered. Surely the lesion must have been in progress before his illness began. In fact, one is led to the belief that during the period of incubation the invading bacilli produce a local "infective granuloma," and that this remains latent until they leave the intestinal follicles by the lymphatic channels, and overspread the entire organism in swarms.

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[*] Dr Moxon held that the morbid change is of less severity, rather than of later date, in the higher patches. He thought that the greater tendency to destruction of those which lie at the end of the ileum is due to the irritating contact of the intestinal contents, held back upon them by the valve. There can be little doubt, however, that in some of the more protracted cases the glands are affected successively and at considerable intervals of time.—C. H. F.

[†] Among cases which end in recovery, it is perhaps not uncommon for all the patches and all the solitary glands which are affected to take this course, but when death occurs at an advanced stage it very rarely happens that some are not found ulcerated. I have only met with one instance of the kind; the patient died on the twenty-first day, and the patches were found swollen, raised, and reddened, but marked by irregular depressed lines and spaces, so that they had a reticulated appearance. They were not "medullary" in character, as would probably have been the case if the affection had been early; there was no trace of sloughing or ulceration. I preserved the specimen in spirit for the museum, but after a few weeks it showed nothing.—C. H. F.

On the other hand, it is not uncommon, in cases fatal at the end of six or seven weeks of continuous fever, to find sloughs still adhering to several of the patches, while others are merely swollen and medullary-looking. Such lesions are doubtless of different dates, and have been developed in successive outbreaks at intervals of a fortnight or more. In the case of a boy who had been ill for five weeks, and who died in Guy's Hospital on October 26th, 1879, Dr Goodhart describes the glands as fleshy-looking, and as just beginning to ulcerate, so that he would have supposed the disease to be at about the eighth or the ninth day. Probably some other glands had been affected from the first, but had afterwards subsided.

A secondary lesion in enteric fever has its seat in the *mesenteric lymph-glands*. This does not appear to be absolutely constant; at least, in our records of autopsies Dr Fagge found two cases, one of them fatal on the seventeenth day, in which the glands are said not to have been enlarged. It may be present at a very early period of the disease; thus in Hoffmann's case, fatal on the fourth day, the glands were already doubled in size. No doubt it is the result of the lacteals absorbing contagion from the affected parts of the intestines, and in many instances the glands corresponding with these parts are alone attacked; but sometimes the upper mesenteric lymph-glands become also involved, and even (as in a case inspected in 1878) glands in the portal fissure. As a rule, the diseased glands are not larger than hazel-nuts, but they may be the size of horse-chestnuts. Their substance is white, or grey or pinkish, with spots of hæmorrhage. Under the microscope they are found to contain large round cells, some of which have two or more nuclei, like those in the intestinal follicles. Suppuration may occur, or partial sloughing; but caseation, or the deposition of calcareous salts, is not the ordinary process of involution—rather fibrous contractions and puckering.

The *spleen* is found soft, and almost always much swollen; but there are exceptions to the latter rule. Even in young subjects who died at the height of the fever, the spleen has been found at Guy's Hospital to weigh only six, five, or even four ounces. Bacilli have been repeatedly found in this organ with the characters of the specific pathogenic species, *B. typhosus*. Indeed, one enthusiast has proposed to insert a small trocar and cannula into the spleen during life in order to diagnose enteric fever, by finding its bacilli in the drops of splenic blood thus obtained.

The *larynx* is sometimes found ulcerated. Hoffmann observed this in twenty-eight cases out of two hundred and fifty; and it is apparently more common in Paris (Louis) and Vienna (Rokitansky) than in England. As a rule there is a sharply defined ulcer over the base of one or both of the arytænoid cartilages. This lesion very rarely gives rise to symptoms, but it may cause hoarseness or aphonia. In some instances the deeper structures are involved, and necrosis and exfoliation of the cartilage may follow and greatly obstruct the entrance of air. In one of Hoffmann's cases tracheotomy was rendered necessary by the formation of a polypoid blood-clot, which hung down into the interior of the larynx. Again, a laryngeal ulcer may endanger life by eating its way deeply into the connective tissue. In a patient, a boy of twelve, who died many years ago under Dr Wilks, subcutaneous emphysema arose from this cause. A similar instance occurred in a boy of ten under Addison (Guy's Hospital Museum, 2465), and a third, in a girl four years old, is recorded by Ziemssen. In the last case it is specially stated that the seat of the ulcer, which led down to the necrosed cricoid and arytænoid cartilages, was below the cords, and as the child had suffered from bronchitis there can be no doubt that the cough had forced air through the ulcer into the connective tissue. In two cases Dr Fagge noticed sloughs in the larynx of a bright yellow colour, exactly like that which in the intestine is attributed to the imbibition of bile.

Sometimes a thin pellicle of lymph has been found lining the interior of the larynx and the epiglottis, and still more rarely a membranous layer, probably due to a complication of diphtheria, for Murchison relates an instance in which fluids escaped from the nostrils when the patient attempted to swallow them.

Zenker, in 1864, discovered two kinds of degeneration in the fibres of voluntary *muscles*; some become granular with fatty degeneration, others undergo conversion into a glassy-looking substance, in which no striæ can be recognised, and which splits up transversely into discs. As Zenker himself remarked, the same lesion occurs likewise in other febrile diseases if sufficiently severe and protracted. It is usually well marked in the adductors of the thighs and in the recti of the abdomen. Muscles so affected may be so much softened that they are found lacerated after death. Thus, in 1870, a patient died of enteric fever in Guy's Hospital, both of whose recti were torn across, and much blood was extravasated; and in another case, in 1866, the inner part of one rectus was found to be not only ruptured, but in a state of suppuration. Hoffman has shown that similar forms of degeneration occur in the tongue.

The *heart* is often found to be soft and pale, and sometimes the right ventricle is dilated, or occasionally the left. Its muscular fibres are more or less granular and fatty, and may have lost their transverse striation. In one instance Hoffmann detected in them the glassy change just described as occurring in the voluntary muscles. Among 159 cases in which he examined the tissue of the heart it was more or less altered in 103.

The *lungs* are found congested at the bases and behind, *i. e.* in the most dependent parts as the patient lies in bed. This "congestion" consists of much œdema, some collapse, and more or less extensive lobular catarrh. Lobar hepatisation is only an occasional complication.

The *liver* has been described as undergoing a diffused change; it is soft, and on section it looks pale. Under the microscope its cells are seen to be swollen and granular. The condition is that named cloudy swelling by Virchow. It affects the secreting epithelium of most glands, and is the direct consequence of pyrexia.* Hoffmann found great overgrowth of cells in the acini of the salivary *glands* and of the pancreas. The *kidneys* are often of a greyish colour, and their epithelium shows the same changes as that of the liver. Perhaps this causes the albuminuria which we have seen to be frequently present.

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* I doubt whether this can account for jaundice in those very exceptional cases in which it is present. It is worthy of remark that the occurrence in the gall-bladder of a colourless mucus, or of a muco-purulent fluid, after an acute illness, is not to be taken as

Wagner and other German pathologists describe the frequent occurrence of *minute grey nodules*—sometimes visible to the naked eye, sometimes microscopic—in the substance of the liver and of the kidneys. Hoffmann noted their presence in thirty-eight among two hundred and fifty cases. A similar observation was once made at Guy's Hospital by Dr Goodhart, in 1879, in the case of a girl aged seventeen.[*] It is believed that these grey nodules may suppurate and form miliary abscesses in the liver or kidneys.

In most cases which recover, these various lesions are doubtless repaired. The process of regeneration of muscular fibres has been traced, and according to Hoffmann the liver cells are reproduced by proliferation, of which there is evidence in the great excess of nuclei which these cells contain when death happens to occur at advanced stages of the disease.

Ætiology.—It is now universally admitted, after years of controversy, that Enteric Fever is specific in its origin as well as in its course, *i. e.* that it is always the result of infection from a preceding case of the disease, and that it always "breeds true." The phenomena of contagion and incubation, of pyrexia running a definite course, and of subsequent protection—all agree with those seen in other specific febrile diseases, and lead by analogy to the belief that here also we have to deal with the invasion and multiplication of a specific microphyte, and with its chemical products.

The specific bacillus.—The determination of the true pathogenic microbe of Enteric Fever has, however, been remarkably difficult, and even now the identification is one of the highest probability rather than absolute demonstration. The intestines after death from Enterica (as well as after death from any other cause) are found swarming with bacteria of many kinds. Candidates from this crowd were brought forward by Recklinghausen so long ago as 1871, by Klebs, by Klein, and by Friedländer. A rod-shaped microbe, described by Eberth in 'Virchow's Archiv.' vols. lxxxi and lxxxiii, in the year 1883, and since investigated by Gaffky,[†] Koch, Coats and many others, is the one which conforms most closely to the criteria stated above (p. 18). It is short and thick with rounded ends, and often a central vacuole, is 2—3 μ long, apt to congregate in groups and is motile by help of delicate long flagella. It has characteristic reaction to stains, characteristic chemical properties, and characteristic forms when cultivated. It can thus be distinguished by skilled observers from a normal inhabitant of the human intestines, *Bacterium coli commune*, which resembles it most closely.[‡] It is found not only in the intestinal canal,

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showing that there has been a deficient secretion of bile in the liver. Probably it only indicates that for several days before death there was no storage of bile, in consequence of the very short intervals at which food was taken.—C. H. F.

[*] One might suppose that acute tuberculosis was accidentally developing at the same time, just as occurred in a child who died a few weeks later of scarlatinal nephritis; but against such a view is the fact that the lungs in the case of fever were free.—C. H. F.

[†] 'Mitth. aus d. k. Gesundheitsamte,' 1881–2, and Syd. Soc. Collected Papers, 1884.

[‡] The diagnosis is given by Dr Klein in the fourth edition of 'Micro-organisms and Disease,' pp. 241—244, and in a tabular form by Dr Dreschfeld in the first volume of Allbutt's 'System of Medicine,' p. 797. See also two papers in the 'Journal of Pathology,' vol. iv, pp. 429, 439 (1897), by Dr Stoddart and Dr M. H. Gordon, on "The Relation and Diagnosis of the *Bacillus coli communis* and that of Enteric Fever."

which is "outside" the body, but also in the tissues, as shown by Eberth; in the mesenteric glands, liver and spleen, and also the urine, and the stools of enteric patients—but not in the blood. It is demonstrable in every case of the specific and recognisable disease known as typhoid or enteric fever. It is not found in health or in any other disease. It can be also obtained in a pure cultivation. The final and crucial proof is still wanting: for there is no animal at present known which is naturally subject to enteric fever. A pure cultivation of the typhoid bacillus when injected into an animal only produces swelling of the spleen and mesenteric glands, sometimes of Peyer's patches, and the microbe is found in the spleen.

It has long been known by clinical observation that fresh typhoid stools are harmless, or nearly so, and that the contagion is developed after an interval of twelve hours or more. It is not destroyed by freezing, but is by heat below the boiling-point, and by chemical antiseptics; also by exposure to light and air, and by conveyance in running water.

These facts are confirmed by observation of Eberth's bacillus in pure culture. It is destroyed by a temperature of about 150° Fahr. (65° C.), but resists cold. It gradually disappears in water, and is destroyed by prolonged exposure to light, particularly to the short actinic rays of the violet end of the spectrum. The gastric juice does not kill it, but it is readily destroyed by calomel or corrosive sublimate, β-naphthol or phenol.

The bacillus, after gaining the intestine, penetrates the mucous membrane, multiplies in its lymph-follicles, in the mesenteric glands, and in the spleen, and thence infects the lymph and the blood, not probably by further extension of the bacillis itself, but by its products, for it is not found free in the circulation. Brieger has described a ptomaïne (with the constitution of a diamine) to which may provisionally be ascribed the effects of the disease on the heart, lungs, brain, and muscles. Dr Wright, of Netley, and Dr Luff, of St. Mary's Hospital, have isolated the same from the urine; and their results have been confirmed by Dr Horton Smith working in Dr Klein's laboratory, except that he finds it present only during the later stages of the disease.

In protracted and severe cases, it is probable that infection by streptococci from the ulcerated and sloughing ileum materially contributes to the typhoid condition of the patient.

Conveyance of the contagion.—Enteric fever is an endemic disease, but may prevail in certain districts more than in others; yet its diffusion is strictly dependent on the intercourse of human beings, and occasionally it appears in such increased frequency as to be fairly called epidemic.

The contagion leaves the body of a patient in the stools, and is usually conveyed by fæcal contamination to drinking-water, and so gains access to the alimentary canal of a second host. But there are other possible means of conveyance.

Direct contagion from one patient to another is unknown or excessively rare. Dr William Budd believed he had seen such cases,[*] and in 1875 Sir William Jenner, in his Presidential Address to the Clinical Society, stated that he had twice known enteric fever contracted by students who took temperatures before the registering thermometer was in use. In 1871 Dr

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[*] Three persons left the village of North Tawton, Devonshire, during the autumn of 1839, after having taken the fever. Two of them went to Morchard and gave it, one to his two children, the other to a friend, and he again to his two chilldren and to his brother. The third went to Chaffcombe, seven miles off, where ten others were attacked in turn, and so to fresh places.

Collie, of the Homerton Fever Hospital, expressed his belief that certain cases among the attendants there were caused by direct infection, either from the freshly passed evacuations of patients or from their lungs or skin. But, striking as such events naturally are to the observer who watches their progress, there are strong grounds for rejecting the conclusion. Murchison tells us that during nine years, from 1861 to 1870, cases of enteric fever were treated in the same wards of the London Fever Hospital with various non-specific febrile complaints, to the number of 3555 of the former class, and 5144 of the latter. The same night-chairs were used by both sets of patients, and the employment of disinfectants was exceptional. Yet enteric fever was not contracted by one of those who were under treatment for other diseases. In the 'British Medical Journal' for 1879 Dr Shirley Murphy brought down to 1878 the experience of the same hospital as to the occurrence of enteric fever among the nurses and other attendants. During twenty-four years only nineteen persons engaged in the institution were attacked by it. "Of these, ten were in no way connected with the enteric fever patients or the enteric fever wards. Of the other nine, one was a laundrymaid, whose duties would bring her into contact with the soiled linen of the patients, but who was not otherwise in contact with them. Of the remaining eight there were special circumstances connected with drainage which would probably account for the fever, leaving four for whose attacks there was no explanation given." In this time 5569 patients with enteric fever passed through the wards.

Conveyance by excreta.—Dr William Budd maintained as early as 1856 that the stools of enteric fever were incomparably more contagious than the breath or other excreta of the patient, and this is now universally admitted. Dr Cayley, in his 'Croonian Lectures' for 1880, expressed his belief that in the Middlesex Hospital patients have caught the fever from using closets in which pans were placed containing typhoid stools set apart for inspection. Doubtless the poison may be transmitted in fæcal defilement upon linen or sheets, or even on the patient's body; nurses may thus, by carelessness and lack of scrupulous attention to their own hands, contract the disease directly, and washerwomen have taken the disease after washing the clothes and bedding of typhoid patients. Biermer met with several instances of this, and Cayley mentions two cases of enterica in the wards of the Middlesex Hospital which were traced to dried discharges upon the sheets of a typhoid patient in a neighbouring bed. Murchison relates the case of a woman who brought to her house in Warbstowe, on the Cornish moors, the bedding of a sister who had died of enteric fever at Cardiff, in Wales. She remained free, but her sister, who was employed in hanging out the clothes, took the disease; and it spread from her as a centre.

Dr Cayley quotes a case from von Gietl, of Munich, of infection from fæces nine months after they had been buried in a dunghill, and another under his own observation in which, as in Murchison's, a second case occurred two years after the first, in the same house and without any fresh importation of the poison. It is possible that the typhoid bacillus may live and multiply outside the human body, and survive, under favourable conditions, for an indefinite period—perhaps in a dormant state, multiplying itself just enough to escape extinction—and then suddenly undergo immense development. Such a hypothesis would apply to Murchison's statement, that he has seen single cases of enteric fever arise in the same house again and again at intervals of a year or longer. Thus between

1849 and 1857 six cases were admitted from a certain house in the London Fever Hospital; one in June, 1849, one in October, 1851, one in February, 1854, one in November, 1855, one in November, 1856, one in July, 1857.

It is possible that the urine or the sputa of typhoid patients may occasionally be the source of infection.

Conveyance of the fæcal contagion by the air is extremely doubtful, although it was believed in by Fagge, by Gaffky, and by Dr J. W. Moore. Many instances have been recorded in which the disease was apparently caused by the exhalations from drains, or sewers, or water-closets, but probably infection really took place by defilement of water.

Conveyance by drinking-water is the explanation of the epidemics of enterica that frequently occur among the inhabitants of a village, who derive their water-supply directly from one or more surface wells, into which sewage finds its way through a porous soil.

At Wicken Bonhunt, in Essex, the disease prevailed in 1869, and Dr Buchanan investigated its origin for the Privy Council Office. He found that there was a broad division among the people in regard to the sources from which they obtained their water. One hundred and eighteen persons used private wells, and among them there was only one positive case of fever; eighty-eight drank the water of one well called the "parish well," and no fewer than forty of them were attacked. Now this well was situated four or five paces distant from a brook-channel which ran through the place. At the upper end of the village the brook always contained water; but lower down the channel was dry during the greater part of the year, the water being carried beneath the surface in a stratum of gravel, to reappear as a stream at the bottom of the village. That there was a direct communication between it and the parish well was evident from the fact that in times of flood, when the channel was full, the water in the well ran to a corresponding height and became discoloured. On June 24th the first case of fever occurred in the person of a boy, who lived in a cottage about thirty-five yards above the well. He had much diarrhœa, and his stools were thrown, without being disinfected, into a privy which stood almost on the edge of the channel. At this very time the soil water in the village was falling, after abundant rains which had taken place a month before, and pools of water were to be seen here and there in the channel. A month after the boy's illness the persons who made use of the well began to fall ill with the fever. Can it be doubted that the well water had become impregnated with the specific poison? Previously, on May 30th, two cases of fever had been imported from London into a house, of which the sewer opened into the brook two hundred and fifty yards above the well.

At Page Green, in the parish of Tottenham, a great many cases of enteric fever occurred in 1864 and 1865. Dr Seaton investigated the matter, and found that whereas there was to some houses a supply of water from the works of the Local Board of Health, the occupants of many other houses drank water from shallow surface wells. In three instances, in consequence of the families having removed from the place, he could not learn from which source the drinking-water had been taken, but in all other cases, with the single exception of one child, it was ascertained that those who were attacked had used well water. Some of them had had the water the Local Board distributed to their houses, but had been in the habit of borrowing water from their neighbours' wells, because it was bright and pleasant, whereas the other was hard, turbid, and red, from rust in the pipes. The well waters, when analysed by the late W. A. Miller, were found to be quite unfit for drinking.

At Terling, in Essex, between the beginning of December, 1867, and the end of February, 1868, there occurred an epidemic of enteric fever, upon which Dr Thorne reported. It was of extraordinary severity: amongst a population of nine hundred persons, at least two hundred and sixty were attacked during the first two months; there were in all forty-one deaths, and so panic-stricken was the village that it was necessary to discontinue the tolling of the church bell at deaths or funerals. Whether the disease was introduced from elsewhere could not be ascertained, for there had been isolated cases during previous years. But the extension of it was clearly traced to contamination of the drinking-water by sewage. The cottages were supplied, singly or in groups, by shallow surface wells, sunk in a loose and porous gravel. Round about them, but at a higher level, there were numbers of manure-heaps, cesspools, and privies, the ordure from which was often spread out for yards over adjacent fields. During the autumn the water in the wells had been unusually low; doubtless, therefore, the poison had accumulated in the soil, so that it was washed into the wells in large quantities, when, in November, a sudden rise of water took place.

In Caterham, during the fortnight which ended February 2nd, 1879, there occurred forty-seven cases of enteric fever; and, at the same time, no fewer than one hundred and thirty-two cases were observed at Redhill, eight miles distant. In each town the persons attacked were using the water furnished by the Caterham Waterworks Company, but considerable numbers of persons who derived their supply from other sources escaped altogether. On the other hand, cases occurred at the Earlswood Asylum and in other places which also used the Company's water. Now this water is drawn from chalk wells more than 500 feet deep, and it had a deservedly high reputation for wholesomeness. Every point in regard to its sources, storage, and distribution was carefully inquired into, but for a long time in vain. At last, however, the attention of Dr Thorne was drawn to the fact that in January, 1879, the Company had been constructing an adit, at a depth of 455 feet from one of their old wells, to a new bore which was then being sunk. Many workmen had been employed upon this duty, and one of them, it was found, had been ill and had left work in the course of the month. He was sought out, and on inquiring it appeared clear that he had been suffering from a mild attack of enteric fever, which began on January 5th, and which he had probably acquired at Croydon, where he had spent December 25th and 26th. He had much diarrhœa, the bowels acting at least two or three times during each shift of eight or twelve hours, and in accordance with the usual practice under such circumstances he made use of the buckets by which the excavated chalk was being raised to the surface. He denied that he had ever relieved himself in the adit without waiting for a bucket; but it seems to be almost certain that in some way his fæces passed into the water of the well in which he was working, and gave rise to the epidemic. The poison must have been marvellously diluted.

The following remarkable case is taken from Dr. Cayley's Croonian Lectures:

"Lausen is a village situated in the Jura, in the valley of the Ergolz, and consists of one hundred and three houses, with eight hundred and nineteen inhabitants. It was remarkably healthy, and resorted to on that account as a place of summer residence. With the exception of six houses it is supplied with water by a spring with two heads, which rises above the village at the southern foot of a mountain called the Stockhalder, composed of oolite. The water is received into a well-built covered reservoir, and is distributed by wooden pipes to four public fountains, whence it is drawn by the inhabitants. Six houses had an independent supply,—five from wells, one from the mill-dam of a paper factory. On August 7th, 1872, the inhabitants of Lausen, living in different houses, were seized by typhoid fever, and during the next nine days fifty-seven other cases occurred, the only houses escaping being those six which were not supplied by the public fountains. The disease continued to spread, and in all one hundred and thirty persons were attacked, and several children who had been sent to Lausen for the benefit of the fresh air fell ill after their return home. A careful investigation was made into the cause of this epidemic, and a complete explanation was given. Separated from the valley of the Ergolz, in which Lausen lies, by the Stockhalder, the mountain at the foot of which the spring supplying Lausen rises, is a side valley called the Furlenthal, traversed by a stream, the Furlenbach, which joins the Ergolz just below Lausen, the Stockhalder occupying the fork of the valleys. The Furlenthal contained six farmhouses, which were supplied with drinking-water, not from the Furlenbach, but by a spring rising on the opposite side of the valley to the Stockhalder. Now there was reason to believe that, under certain circumstances, water from the Furlenbach found its way under the Stockhalder into one of the heads of the fountain supplying Lausen. It was noticed that when the meadows on one side of the Furlenthal were irrigated, which was done periodically, the flow of water in the Lausen spring was increased, rendering it probable that the irrigation water percolated through the superficial strata, and found its way under the Stockhalder by subterranean channels in the limestone rock. Moreover, some years before, a hole on one occasion formed close to the Furlenbach by the sinking-in of the superficial strata, and the stream became diverted into it and disappeared, while shortly after the spring at Lausen began to flow much more abundantly. The hole was filled up, and the Furlenbach resumed its usual course. The Furlenbach was unquestionably contaminated by the privies of the adjacent farmhouses, the soil-pits of which communicated with it. Thus, from time immemorial, whenever the meadows of the Furlenthal were irrigated, the contaminated water of the Furlenbach, after percolation through the superficial strata and a long underground course, helped to feed one of the two heads of the fountain supplying Lausen. The natural filtration, however, which it underwent rendered it perfectly bright and clear, and chemical examination showed it to be remarkably free from organic impurities; and Lausen was extremely healthy and exempt from fever. On June 10th one of the peasants of the Furlenthal fell ill with typhoid fever, the source of which was not clearly made out, and passed through a severe attack, with relapses, so that he remained ill all the summer; and on July 10th a girl in the same house, and in August a boy, were attacked. Their dejections were certainly, in part, thrown into the Furlenbach; and, moreover, the soil-pit of the privy communicated with the brook. In the middle of July the meadows of the Furlenthal were irrigated as usual for the second crop of hay, and within three weeks this was followed by the outbreak of the epidemic at Lausen. In order to demonstrate the connection between the water-supply of Lausen and the Furlenbach the following experiments were performed:—The hole mentioned above as having on one occasion diverted the Furlenbach into the presumed subterranean channels under the Stockhalder was cleared out, and 18 cwt. of salt was dissolved in water and poured in, and the stream again diverted into it; the next day salt was found in the spring at Lausen. Fifty-six pounds of wheat flour were then poured into the hole, and the Furlenbach again diverted into it; but the spring at Lausen continued quite clear, and no reaction of starch could be obtained, showing that the water must have found its way under the Stockhalder in part by percolation through porous strata, and not by distinct channels. Besides showing the necessity of the introduction of the specific poison in order to render sewage contamination capable of giving rise to typhoid fever, this case is remarkable as an instance of the extreme dilution to which the poison may be subjected without losing its potency, and also the uselessness of irrigation and any ordinary filtration in separating it or rendering it inert. Here the dejections of two cases of typhoid are thrown into a stream; the water of this stream is used to irrigate extensive meadows; a portion of it sinks through the superficial strata, and probably finds its way into subterraneous channels, and passes through a distance of many thousand feet under a mountain, partly, no doubt, by mere percolation; it then takes an insignificant part in feeding one head of a copious spring, which has another head that is not contaminated; and, nevertheless, it gives typhoid fever to one hundred and thirty persons out of a population of eight hundred. The dilution must have been infinitesimal, unless we assume that a multiplication of the poison took place after its discharge from the intestinal canal of the first two cases—possibly in the reservoir at Lausen." This seems to be the explanation.

At Caius College, Cambridge, a local outbreak of the disease occurred in November, 1873, which was traced by Dr Buchanan, with very strong probability, to a precisely similar origin. Twelve out of fifteen cases in students at the college were among the sixty-three residents in Tree Court, a part of the building which had been erected only four years previously with every care as to sewers, drains, and water-pipes. Now Tree Court had an independent water-supply direct from a high-pressure main. This supply was intended to be constant, but there had in fact been a complete intermission of it on two occasions shortly before the outbreak. After such intermissions the water had been noticed to come in with a rush, like "soda-water," evidently in consequence of its having become mixed with air, which had been sucked up into the pipes. Within the Tree Court buildings there were two waterclosets, one in the basement of the porter's lodge, the other on the first floor of one of the staircases. The tap of the lower one, or that over an adjoining sink, if left open during the intermission of water-supply from the main, would have allowed water to drain from the whole pipe system of the court; that of the upper one would under such circumstances have permitted of the free entrance of air. This air, however, would have been mixed with sewer gas from an unventilated sewer in Trinity Street, which, at the very time under consideration, was receiving the excreta of patients ill with fever in other parts of the town. The effect of recharging the pipes with water must necessarily have been to distribute sewer gas in solution to every part of the building. It was, indeed, positively ascertained that not merely air, but water impregnated with fæcal matter, had been sucked up into the supply-pipe of the upper watercloset, for that pipe was lined with a brownish deposit, containing phosphoric acid and a large proportion of intermixed organic matter.

More recent examples, particularly that of the terrible epidemic of enteric fever at Maidstone in 1897, have confirmed the conclusions derived from the earlier cases given above.

Dr George Turner, speaking from large experience as an Officer of Health, remarked: "In the country amongst the cottagers Typhoid Fever appears to spread almost like Measles or Scarlet Fever. A patient returns home ill from the town, and her relations become infected. This is because usually the supply of linen is short; sheets, &c., when stained by excreta, are not immediately changed, and the air of the dwelling, usually a small one, is polluted: but it is more frequently brought about by a scarcity of washing utensils; the same bucket takes the water which has been used for washing the patient's clothes to the privy or cesspit, and then serves for a fresh supply of water, often of drinking-water. I have seen typhoid spread in a village because the family first infected dipped the bucket which had been used for taking away slop-water into the common dipping well, and thus polluted the whole water-supply."

Conveyance by milk.—This mode of infection is clearly established by the following remarkable instances. In some, perhaps most, cases the vehicle is not the milk itself, but the water "used to wash the cans," or possibly added to the milk with intent to cheat, but not to kill.

The first epidemic that was traced to this origin occurred in Islington in 1870. It was investigated by Dr Ballard.

Between July 3rd and September 10th the occupants of sixty-seven houses were attacked, one hundred and sixty-seven individuals, of whom twenty-five died. It was a most remarkable circumstance that the district affected was included in a semicircle, with a radius of a quarter of a mile, drawn immediately on the north side of the line of the North London Railway from a centre almost upon this line. There was no fever in the area contained in the corresponding semicircle south of the railway, which here passes through a cutting. This, of course, at once suggested that human intercourse was in some way concerned in spreading the disease. The right clue was first hit upon by a lady whose family was attacked; and an inquiry convinced Dr Ballard that, far-fetched as the idea appeared, it was probably true. The milk vendor whose milk was suspected had himself fallen a victim to the epidemic, but his father, greatly to his credit, readily consented when applied to to give a list of the customers. It was then found that the dairy supplied one hundred and forty-two families—a very small proportion of those who lived within the semicircle. In no less than seventy among the hundred and forty-two families there had been cases of enteric fever. The way in which the disease picked out the customers of the dairy in particular streets and rows was most striking. In one long road and a street running from it the milkman supplied three families; two of them were affected. In a crescent with twenty-five houses he supplied four families; they were all attacked. In a new neighbourhood, where there were about seventy houses, he supplied four families; three had the disease. In a square with fifty-nine houses he supplied four families; all had it. On the other hand, there were scarcely any cases among those families who had invariably bought their milk from other sources. As might have been expected, women and children were attacked in much larger numbers than men. The source of infection was traced, with much probability, to the water of an underground tank in the cowyard. It was not proved that water from the tank had been used to dilute the milk, but the pails were washed out with it, and some might have been left in by accident.

In the summer of 1873 an outbreak of enteric fever occurred in St. Marylebone, and in certain parts of St. George's (Hanover Square) and of Paddington parishes.

It affected, among others, the family of Dr Murchison, who quickly became convinced that the only probable mode of introduction of the disease into his house was by the milk supply. This was confirmed by a minute investigation made by Mr Netten Radcliffe and Mr Power. It was shown that nine tenths of the two hundred and forty-four cases to which the inquiry extended were in households which consumed milk from a particular service of a particular dairy. Certain ramifications of the same milk supply extended to the east end of Regent's Park, to Belsize Park (Hampstead), and to St. Anne's (Soho), and in these districts also enteric fever occurred among consumers. There was a special incidence of the disease upon women and children, and many striking instances are noted in which those members of a family were attacked who were in the habit of drinking milk, while others escaped who did not do so. The milk which appeared to convey the poison was a special kind, sold as "nursery milk," and taken from three or four cows, set apart for the purpose at Chilton Grove Farm, in Buckinghamshire. Now on the 8th of June the occupier of this farm had died in the fourth week of an attack of enteric fever. His evacuations, instead of being thrown into the common privy, were buried in an ash-heap outside the farm buildings. Subsequently, however, it turned out that this was the worst thing that could have been done with them. For there was a well close by, the water of which was used for dairy purposes, although not for drinking or cooking, as it had been noticed to have a disagreeable taste. Excavations made for the purpose showed that there had been a line of soakage into the well, along the foundations of a wall, of the filth from a pigsty which formed a pool in immediate proximity to the ash-heap above mentioned. If due intervals are allowed for the gradual penetration of the matters containing the poison of enteric fever through the soil, and for the incubation of the disease, the date at which the outbreak in London began—during the last days of June and the first days of July—corresponds exactly with this theory of its origin.

Many cases of enterica due to milk have been recorded in Switzerland, in Australia, in America, and in other countries.

As with milk, so with ices and lemonade, it is the water with which they are made that conveys the poison.

Conveyance by food.—At Kloten, near Zurich, six hundred and sixty-eight persons were attacked in July, 1878, all of whom had partaken of some veal provided for a festival of the choral societies, and partly derived from two diseased calves. One of these was (Dr Huguenin believed) the subject of enteric fever. It is, however, very doubtful whether cattle or any of the lower animals are liable to enteric fever.

Five other outbreaks, also attributed to diseased meat, are quoted by Dr Cayley.

But at Andelfingen, and at Thalweil, the epidemics occurred after eating putrid meat, and only some of those who ate it showed the symptoms of enteric fever during life, or the signs of it after death. The infection at Kloten was believed by Huguenin to be a mixed one.

There can be no doubt from numerous confirmatory cases since Sir William Broadbent drew attention to the subject in 1895, that raw oysters may convey the enteric poison; probably owing to the oyster beds being too near the outfall of a sewer into which enteric bacilli have gained an entrance.* Salads and all other articles of food eaten cold and wet might also convey the infection; and raw potato is an excellent seat for multiplication of the enteric bacillus.

It is possible that flies or other insects may convey infection from typhoid excreta to vegetables or other food.

It is not always, or even generally, possible to trace to their source isolated cases of enteric fever when they occur in large towns like London. There are many chances of infection from impurities in water, which no inquirer, however acute, could trace. The number of cases admitted into the London Fever Hospital varies from year to year much less than might have been expected. Dr Cayley believes that they are not dependent upon the presence of the contagion of the disease in water taken from the Thames; for although there can be no doubt that the poison frequently passes into the upper part of the river from the towns and villages on its banks, he thinks it is always destroyed by exposure to the air and to light, and possibly by the growth of saprophytes.

Predisposing external causes.—Buhl, of Munich, applied the observations of von Pettenkofer on the relation between the *soil-water* and cholera to the case of enteric fever; he showed that when the soil-water in that city (as measured by the depth of water in the surface wells) was falling, the number of cases of enteric fever increased; when it was rising, the number of cases diminished. Liebermeister and Buchanan explained these facts by the effect of the nearness of soil-water to the surface in producing infection of the wells. Similar observations have been recorded in Berlin; but in Buda Pesth enteric fever appears to be more prevalent when the ground-water is rising, and less so when it is falling. In an epidemic at Terling, in 1868, the disease broke out with great severity precisely when the wells were high.

There is no doubt that the *weather* affects the prevalence of enteric fever. In the London Fever Hospital there have been far more admissions during dry and hot summers than in damp and cold ones. Each year there is an increase of the disease during the four autumn months from August to November, while it is less frequent from March to May inclusive. Similar observations have been made in America, where the disease is called "fall-fever," and also at Berlin and at Basle. At Munich the maximum prevalence of enteric fever is in February.

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* See a valuable paper with numerous cases of other edible molluscs being the vehicle of typhoid infection, by Dr Newsholme, 'Journal of the Sanitary Institute,' vol. xvii, part 3, also a case graphically recorded by Dr Cavafy in 'The Clinical Journal' for 1896.

In Australia, as in the United States, enteric fever is most common in the late autumn and after a hot summer. In Victoria (January being midsummer) the disease is most prevalent in March and least so in November.*

All such conditions play but a secondary part in the ætiology of the disease; their effect is merely to favour, or to hinder, the operation of its real cause.

Predisposing internal causes.—The power of human beings to resist the contagion of enteric fever, as of other specific infections, is probably to some extent individual—dependent on the "vulnerability" of the tissues and the activity of the phagocytes. But it also depends on general conditions, of which the most important is *age.* The disease is far more frequent between the ages of five and of thirty than earlier or later. Under two years old it is very uncommon, but in 1864 Murchison showed at the Pathological Society the intestines of an infant six months old who had been attacked at the same time with its mother. In the ten years from 1879 to 1888 we had in Guy's Hospital seven cases of enteric fever in patients between three and five years old, 103 between five and fifteen, 241 between fifteen and thirty, 42 between thirty and forty, and 13 above forty years of age. The youngest patient was three and a half years old, and he recovered; the oldest was fifty-four, and he died.

The statistics of the hospitals of the Metropolitan Asylums Board, between 1871 and 1894 inclusive, are given by Washbourn and Goodall as follows:

Patients under 5	287,	of whom	37	died,	or nearly	13	per cent.
5—10	1209	„	107	„	„	9	„
10—15	2130	„	281	„	„	13	„
15—20	2064	„	365	„	„	17·7	„
20—25	1388	„	279	„	„	20	„
25—30	920	„	209	„	„	22·7	„
30—35	554	„	137	„	„	24·7	„
35—40	321	„	89	„	„	27·7	„
40—45	175	„	44	„	„	30	„
45—50	104	„	36	„			
50—55	43	„	14	„			
55—60	15	„	7	„			
Over 60	13	„	4	„			

During childhood the liability to the disease increases from year to year, probably owing to increased exposure to contagion. After the age of twenty the liability begins to decline, after thirty more rapidly, and beyond forty comparatively few cases occur. Dr Wilks once found enteric ulcers in the ileum of an old woman of seventy. Liebermeister gives a tabular statement of the proportion of cases at varying ages at Basle, corrected according to the numbers of persons at the corresponding ages in the population generally. This correction must not be forgotten, nor yet the fact of protection acquired by having already passed through the disease in youth.

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* From a table given at a meeting of the Intercolonial Medical Congress on January 11th, 1889, by Dr J. G. Carstairs, of Geelong, Victoria, it appears that in Melbourne the mortality is least in November; it rises in December, to attain a maximum in March; falls very slowly in April and May, and has a sudden decline in June. The hottest month in Melbourne is January, when the mean temperature of the air averages 66·2° F.; so that, as with us, the maximum mortality of typhoid obtains about two months after the hottest season of the year is passed. In Melbourne, too, an excess of summer heat is followed by excessive prevalence of the fever.

But it may be that the atrophy of the lymphatic organs in old age, including those of the ileum, is an anatomical condition which is unfavourable to the reception and multiplication of the enteric microbe.

There does not appear to be any constant predominance of one *sex* over the other among patients suffering from enteric fever. Among the 415 patients at Guy's Hospital above mentioned there were 272 male to 143 female, but this is not a constant ratio. There seems to be a certain degree of immunity among women in pregnancy, after labour, and during lactation.

French writers have stated that students, servants, and foreigners are very liable to contract this disease when they first come to live in Paris, and Murchison has shown that more than 6 per cent. of the patients admitted into the London Fever Hospital have arrived in London within three months.

Mortality.—Enteric fever varies in severity at different places and at different times, but perhaps less than might have been expected. It does not appear in malignant outbreaks, as we shall see in the case of measles and of scarlatina.

At the London Fever Hospital the average death-rate from 1848 to 1870 was 18·9 per cent. of all cases admitted, including paupers and moribund patients, the extreme figures in particular years being 28·42 and 12·82. For purposes of comparison between different modes of treatment, it would, however, be necessary to exclude all cases which ended fatally before treatment had fair chance, say within forty-eight hours of admission; and this would reduce the average death-rate to 15·82 per cent. From 1871 to 1882, when paupers were not admitted, the mortality at the same hospital was 17·6 per cent. (Collie), and this includes a considerable number of patients who died within forty-eight hours of admission. At Stockwell Fever Hospital, in 1875-80, the mortality was 22·6, and at Homerton 16·8 per cent. Compare the table given above of mortality in all these Metropolitan Sick Asylums from 1871 to 1894 (p. 146). In the Dublin Fever Hospital, 1871—1890, among 1405 cases the mortality was only 8·6 per cent. (Moore). Statistics from various sources given by Murchison show a range of mortality from 11·37 to 32 per cent. At Basle, the average death-rate from 1843 to 1864 was 27·3 per cent; at Vienna, 22·5 per cent. At Munich, for several years before and after 1880, a very low mortality was observed, and ascribed to treatment by baths; but the patients were soldiers, selected healthy young men, under the most favourable circumstances. At Hamburg, according to Senator, the mortality among 937 patients (1874-7) was only 7·2 per cent., and among 568 patients after treatment by cold baths was 7·3. At Berlin, of nearly 13,000 patients of all ages and under various treatment, the mortality was 14·5 per cent., but it was as high as 20 per cent. among 60,000 cases in France, according to Jaccoud.

According to Goltdammer the mortality from enteric fever in the German army is only 10 per cent., in the Austrian 26·8, in the Italian 28·3, and in the French 36·5 (?).

How difficult it is to judge of the normal mortality, uninfluenced by treatment, even among patients of about the same age, in the same locality. and of the same habits and mode of life, is shown by the following results obtained by Eichhorst in the General Hospital at Zurich in three successive years. In 1884 there was an epidemic of enteric fever, and of 411 patients fifty-six died (13·5 per cent.); in 1885, of 164 patients only seven died (a little over 4 per cent.); and in 1886 there were only 91 cases, of which five were fatal (nearly 5·5 per cent.).

During ten years (1879-88 inclusive) the number of cases of enterica treated in Guy's Hospital was 415; and the number of deaths seventy-two. One of these was from accidental poisoning by morphia, so that the mortality from the disease was 17·1 per cent.—15 per cent. for men, 21 for women. In the preceding ten years the mortality had been 19·3 per cent., while that of other London hospitals varied very widely—from 12 to 20.

Prognosis.—The forecast in enterica depends partly upon the age, habits, and condition of the patient before its invasion, partly upon the symptoms which gradually develop as each case goes on, and partly on early and judicious treatment.

The general prognosis with respect to *age* is, as in typhus and smallpox, most favourable for children above infancy and for young adults; least so for elderly patients. But the mortality does not vary to the same extent; and even in patients over fifty it is not more than twice as high as those between ten and twenty (see the table on p. 146). The disease, however, is comparatively rare above forty, and not common under five. Almost all the very slight cases occur in children. At Guy's Hospital the mortality under five was 1 in 7, too small a number to afford a guide. Between five and fifteen it was rather less than 12 per cent., between fifteen and thirty a little more than 16 per cent., between thirty and forty a little more than 26 per cent., and above forty nearly 50 per cent.

Enteric fever is very dangerous in drunkards, in those who are very fat, and in those who are affected with Bright's disease. Murchison believed that the death-rate is not greater among the poor than in the upper classes. In pregnant women miscarriage almost always occurs; but as a rule the patient recovers.

The prognosis afforded by symptoms is often unexpectedly altered by the supervention of some grave complication. No case, however mild, is altogether free from danger; although the fatal complications are more common in severe and protracted cases. Apart from complications, the death-rate in young adults is found to be greater in proportion to the maximum temperature. Fiedler found that the disease proved fatal to more than half of those patients in whom the morning temperature reached 105·4°, and to every one, with a single exception, in whom it reached 106·25°. The more marked the morning remissions the more favourable the case. A considerable fall is a good sign, but not if it be to subnormal, for this is often due to hæmorrhage or perforation.

The best measures of the extent to which a continued high temperature is injurious are afforded by the state of the pulse and by the degree of mental disturbance. Liebermeister put together the cases that had occurred at the Basle Hospital, and shows that, the average mortality being 16 or 17 per cent., no fewer than 40 per cent. of those patients died when the pulse reached 120, and 80 per cent. of those in whom it exceeded 140. He also arranged his cases according to the severity of the delirium and coma, and corresponding variations were found in the death-rate.

Next to the general severity of the febrile process as shown by the temperature, the pulse, and the nervous system, we have regard to the condition of the *heart* and of the *lungs* in forming a prognosis. A feeble pulse and faint systolic sound, and very rapid breathing with cyanosis, are both grave symptoms.

A source of danger which is not perhaps enough recognised is the supervention of *nephritis*. I have now lost six or seven patients from this complication, and similar cases have occurred to some of my colleagues. Two were cases of suppuration of the kidneys secondary to cystitis and pyelitis, and two from the same cause, apparently directly dependent on the pyæmic element in typhoid fever. Three others were cases of acute tubal nephritis.

Severity of the *diarrhœa* is also an unfavourable symptom, and soon tells upon the strength. But some of the cases which are entirely without this symptom are far from favourable, and Sir William Jenner used to say that he preferred moderate diarrhœa to constipation in enteric fever. Dr Wilks published in the 'Guy's Hospital Reports' for 1855 (third series, vol. i, p. 319) a case in which scybala were found in the intestines with an ulcer under each. A patient of the writer's died from perforation who had been constipated throughout the attack.

The significance of *hæmorrhage* has been already discussed: there is no question that large or repeated bleeding from the bowels is a very grave symptom, not only in itself, but as indicating deep ulceration.

Extreme *tympanites* has long been justly regarded as a very unfavourable sign.

Of all symptoms the signs of *perforation* are the gravest, but the writer has seen one case recover in which this had probably taken place, and others have been recorded (cf. *supra*, p. 124).

The chief dangers both from the gradual failure of the heart and lungs by sepsis, and from the sudden supervention of hæmorrhage or perforation, belong to the third week of the fever.

If, as so often happens, the onset of the fever has been gradual and obscure, the patient will perhaps have been going about for several days after pyrexia began, and this is an unfortunate beginning. Early recognition and consequent confinement to bed and to fluid nourishment is the best way for a patient to start on the dangerous and uncertain course of typhoid fever.

The *sequelæ* of enterica, though numerous and often both tedious and distressing, are comparatively seldom fatal. Necrosis and caries, unhealed bedsores, thrombosis of the femoral, the profunda or the internal saphena vein, suppuration of the parotid,—although they greatly retard convalescence and cause much pain and anxiety, seldom end in death.

Relapses also are very rarely fatal. In fifty cases the writer only met with one death—from perforation; and in spite of the enfeebling effects of the first attack, he has given a good prognosis in apparently severe cases of a first or second relapse, and has been justified by the event.

Treatment.—We may preface this part of our subject with the following quotation from Baglivi, as true now as it was at Rome at the close of the seventeenth century: *In nullo morborum genere tantâ opus est patientiâ, expectatione, cunctationeque ad bene et feliciter medendum, quantâ ad bene curandas febres mesentericas.** The first and one of the most important points in the rational treatment of enteric fever is that the patient from the beginning should be kept at rest in bed. Men are apt to do themselves irreparable injury by struggling on day after day, even if they do not try to "walk off" their illness, or go away "for a change of air" when they ought to be lying in bed. In this respect the members of our profession show no more judgment than others. Liebermeister has known medical men go on seeing patients after having themselves noted their temperature at 104° on the previous evening. Sir William Jenner, speaking at Birmingham in 1879, declared that some of the worst cases of enteric fever he had ever seen were in patients who had travelled, after falling ill, in order to reach home. He added that he scarcely ever allowed a patient to be removed from the place where he was seized with the disease, if his residence was at a distance.

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* Op. omnia, ed. vii, 1710, Praxeos medicæ, lib. i, cap. 9.

The sick room should be large and airy, and provided with a fire or with a door opening into another room, so that it can be well ventilated. The danger of a fever patient catching cold is probably much exaggerated.

Diet.—The patient's nutriment should be entirely liquid. The best food in this, as in other fevers, is milk, of which two, three, or, if nothing else is taken, four pints may be supplied each day; it should be given in regular portions every two or three hours,—oftener if needful, but this should be the exception. As the case goes on it soon becomes necessary to continue the feeding systematically throughout the night. If curdled milk is rejected by the stomach, or if it appears undigested in the evacuations, it should be boiled, or lime-water may be added in the proportion of one part in three, or the milk may be diluted with barley-water; and if it still disagrees, it should be peptonised with *liquor pancreaticus*, which prevents a curd forming. The objection to peptonised milk is that the taste is so unpleasant that the patient often refuses it.

Occasionally, in spite of all such endeavours, milk is not tolerated, and we must then fall back on dilute white of egg, barley-water, water-arrowroot, broth, and jellies.

As additions to a milk diet may be used beef-tea, skimmed mutton broth, blancmange (made with isinglass and milk or cream), custard and calf's-foot jelly. Beef-tea is apt to cause increased diarrhœa. It is, therefore, well to avoid the routine use of broth or extracts made from beef. Beef-juice, veal-tea, and chicken broth are less likely to act on the bowels. When there is no diarrhœa, but constipation, beef-tea is rather indicated than not, and its valuable effect on the heart should not be lost. Jenner advised that some vegetables should be boiled with the broths, and strained off afterwards.

Neither milk nor broth quenches thirst, and a patient in fever may be allowed to drink as much water as he pleases. Nor is there any reason why a patient should not have a cup of tea, if freshly made, not strong and not very hot. Toast and water, or lemonade, or barley-water with lemons may be preferred, or red or black currant jelly in water, or strained tamarind tea; but before many days the tongue becomes furred and dry, the patient can taste nothing, and cold water is the most grateful drink. Iced water quenches the thirst less, and ice to suck often rather aggravates than relieves the parched mouth and throat. Painting the tongue with very weak glycerine is a better remedy, and gum arabic or glycerine lozenges are also useful. The lips should be kept smeared with cold cream or vaseline so as if possible to prevent painful fissures.

Stimulants.—The rules for the administration of alcohol, in which most physicians agree, are as follows. Young and healthy patients may go through enteric fever with perfect safety when no stimulants are administered throughout. When, however, the pulse is irregular or very weak, and the first sound of the heart becomes faint, or when there is great prostration, shown by the body sinking in bed, by sluggish reflex action, by the motions passed unconsciously, the eyes half open, the attention scarcely to be roused—then, whatever the age of the patient, stimulants must not be withheld. Most adults will need alcohol before the disease has run its course, but it is generally better not to begin its administration at once, but to wait for indications in the circulation, the breathing, or the general condition. All cases of fever in patients above fifty are grave, and stimulants are often needful from the first.

When the condition of the circulation or pulmonary congestion or general depression of the patient's powers demands alcohol, it should be given every two hours or every hour, sometimes for a short time more frequently still. Children generally take brandy, diluted and sweetened, better than any other form of stimulant. Young adults often do better with wine, especially red wine, such as burgundy and port. Elderly patients are sometimes wonderfully benefited by champagne. The egg and brandy mixture of the Pharmacopœia is a valuable medicine, and may be taken when other forms of stimulant are refused. Whatever form is found best suited to the case, it should be given as a medicine, *i. e.* in definite doses and at definite intervals.

Drugs.—No method of treatment has yet been discovered by which the course of enteric fever can be shortened, but there is no absurdity in supposing this to be possible. We have good reason to believe that occasionally the disease spontaneously aborts at the end of the second week and even earlier, and the number of such cases may possibly be increased. Liebermeister is disposed to believe that by three or four doses of from eight to ten grains of calomel, given within twenty-four hours at an early period of the fever, he succeeded in certain cases in cutting short the disease, and this treatment is much followed in Germany. Antipyretics, antiseptics, and other remedies are more generally used there than in England, but here also we hear from time to time of new methods of specific treatment, which, as a rule, turn out on trial to be either useless or harmful.

At present the most rational and successful treatment of this, as of most other fevers, is to help the patient through by rest, by suitable food, and by good nursing; to watch carefully and intelligently, and to interfere when needful, but not before.

Treatment of the pyrexia.—The most important and still disputed question is whether it is advantageous to keep down the temperature systematically by *cold baths*. A mass of evidence has been accumulated in support of this practice, which had fallen altogether into neglect, although it had been advocated and successfully practised by James Currie, of Liverpool, a century ago. The revival of the treatment was due first to Brand, of Stettin, in 1861, and secondly to Jürgensen, of Kiel, in 1866. Since that time it has been widely adopted both in Germany and in England; here no one has studied it more carefully than Dr Cayley, of the London Fever Hospital, who recorded his experience in the Croonian Lectures for 1880.

The principle upon which this method of treating enteric fever is based is that pyrexia is harmful, because the tissues, and particularly the heart, are injured by a continuous and protracted high temperature. Those who recommend this practice appeal not merely to the vague impressions derived directly from the observation of cases, but to a numerical analysis of the results which they have attained.*

Notwithstanding, however, the weight which the judgment of those who advocate systematic baths in enteric fever justly carries, many are unconvinced of its advantages. Statistics of results are much open to fallacies. Some epidemics are very slight, others very severe. A case early treated and carefully nursed and fed will do well, when one not seen until the second week will die. The dependence of mortality upon the patient's age is so remarkable that we can only fairly compare those of about the same time of life. The two most formidable complications, hæmorrhage and perforation, may at once change a favourable into a hopeless prognosis. Lastly, there is no acute disease which varies so greatly in symptoms, severity, and reaction to treatment, as enteric fever.

At present, therefore, it seems desirable (in accordance with the experience of Dr Alex. Collie and many other physicians) to employ means of reducing the temperature, not as a routine method of treating the disease, but as a special method of treating the symptom of high fever. It is doubtful whether moderate pyrexia—say under 103° F.—is of itself injurious, and whether its suppression, if possible, is desirable. But there is no doubt that hyperpyrexia is a dangerous condition in itself, and should be dealt with energetically. At what point we should interfere cannot be absolutely laid down. A temperature of 104° in a child may be left alone, when one of 103·5° in an adult must be dealt with. A considerable evening rise which subsided well towards morning may be only watched, while pyrexia which rose high last night and scarcely remitted this morning must be checked this afternoon as soon as it begins its ascent. Moreover the presence of delirium and restlessness, or of a very rapid pulse, shows that *for that patient* the temperature is high and must be treated, although the same or even a higher temperature unaccompanied by grave disturbance of the nervous system and the circulation may be safely left to itself.

When we have decided on bringing down the temperature, there are several ways of accomplishing it. One is to place the patient at once in a cold bath of 60° or 65° F. The shock may sometimes serve as a useful stimulus; but it is almost always better to use a tepid bath of 90° to 85° F., and rapidly cool the water with lumps of ice. The temperature is best watched by means of a thermometer in the mouth, and it must be remembered that it will most likely fall considerably after removal from the bath. Another important precaution is to give brandy immediately after, or even before the bath, so as to stimulate the heart and further the cutaneous circulation. Even with all care and precautions the cold bath is so formidable to patients that, notwithstanding German experience and Dr Cayley's able advocacy, it has not made much progress in the treatment of enteric fever in this country. In America Dr Osler, while admitting its value, finds its application practically limited.

Currie's original plan of cold affusion is well adapted to relieving headache and delirium with pyrexia by directing a douche upon the head and neck; and so are Leiter's tubes, in hospital or wherever they are at hand. The practical difficulties of a bath in the case of adult patients, and the serious disadvantage of lifting and moving them, it may be many times in a few hours, speak strongly for applying cold to the surface as they lie in bed. For this purpose the "wet pack" has been often used with good success; it is usually soothing and sedative as well as antipyretic, but is less effectual than the bath, and sometimes is resented. Another plan is placing bladders of ice in the axilla and over the great vessels of the neck and thighs, or fixing a coil of Leiter's tubes in the same regions and feeding them from a receptacle of iced water placed over the patient's bed. But in many, perhaps in most cases, the easiest and safest is also the most efficient method, namely, sponging the surface with cold water, or rubbing the trunk and limbs with pieces of ice, as the patient lies on a blanket with a waterproof sheet under it. Even when there does not appear any call for active interference, sponging the face, arms, and legs with cold water or spirit lotion is grateful to the patient, and is often followed by tranquil sleep.

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* See, for example, statistics of mortality in enteric fever, before and after the treatment by baths was introduced, at Berlin, at Hamburg, and in the German army by Senator, Goltdammer, and others, 'London Med. Record,' Dec., 1886, p. 557.

A plan which the writer has sometimes adopted with benefit is that of elevating the bedclothes on a large cradle, from which waterproof bags of ice are hung. The cold air bath thus formed is easily applied, grateful and efficient.

An extension of the treatment by bathing is the plan advocated by Dr James Barr, of Liverpool, of keeping the typhoid patient immersed continuously (except the head, which is supported on an air cushion) in a bath of water which is kept at 90° to 93° Fahr. so long as pyrexia continues. In case of collapse it may be quickly raised to 98° or higher. This treatment can only be employed in hospitals or houses with large bath-rooms; but the method is reasonable, and if further experience should confirm Dr Barr's results ('Lancet,' 1890, i, 690) may become frequent.

Many *antipyretic drugs* have been recently used in enteric and other fevers, sometimes in conjunction with, sometimes instead of, what may be called "the cold-water treatment." And certain of these have been credited with antiseptic power in addition, by which the course of the disease apart from the pyrexia is modified. Of these, quinine in large doses was at one time popular, but, except under special circumstances, it cannot be depended on to bring down hyperpyrexia, and is of doubtful value for any other purpose. "Large doses of quinine are as useless and injurious as in typhus" was Murchison's dictum.

It also is believed in this country that salicyl compounds, invaluable as they are in rheumatic fever, are nearly useless in pyrexia without synovitis, and sometimes injurious.

The following is the writer's experience of the more modern antipyretic drugs:

Kairin (a chinolin compound, the hydrochlorate of ox-ethyl-chinolin hydride) was one of the first artificial antipyretics introduced. It proved efficient but very nauseous, the effect was evanescent, and it depressed the cardiac action.

Antipyrin or antipyrrhin, now more modestly named Phenazone (phenyl-dimethyl-isopyrazolone), belongs to the same chemical series. It is more lasting in its effects, but it also has to be frequently repeated, and is dangerously depressing to the heart. Moreover it is apt to produce vomiting, and sometimes rigors, while collapse may follow full doses. It is most useful in relieving the early headache of enteric fever.

Thallin (the sulphate or tartrate of tetra-hydro-para-chinanisol) is probably as efficient, or more so, and safer. But it also sometimes produces rigors, and is not free from danger.

Antifebrin, now called acetanilide (an aniline, not a chinolin compound, phenyl-acetamide), is said to be more useful, more agreeable, and more safe than any. It acts more slowly than kairin or antipyrin, but its effects last longer. It is efficient in small doses; but, when repeated, three grains may prove depressing, and ten grains may cause alarming collapse.

Phenacetin is also effectual in smaller doses than phenazone. Like phenazone, the benefit it gives in neuralgia and certain forms of headache is striking and useful; but on the whole the use of either as a febrifuge is seldom expedient.

One great drawback to all such drugs is the difficulty of knowing when to give them so as to *prevent* the rise of temperature, and another is their accumulative depressing action on the heart. For some years past the writer has abstained from their use in enteric fever, and resorted to the direct application of cold when the pyrexia calls for treatment.

Internal antiseptics.—It is not surprising that various methods have been recommended for checking the putrefactive changes which take place in the intestines during enteric fever. For they are rightly regarded as the chief cause of the abdominal distension and of the septicæmia, which doubtless complicates the action of the toxines produced by the special bacillus of the disease. Calomel and corrosive sublimate have been used for this antiseptic purpose; chlorine water and hydrochloric acid, creosote, phenol, β-naphthol, thymol, salol, and sulphocarbolate of sodium. The writer's experience of this plan of treatment is not encouraging. It seems to be impossible to introduce mercury or any other of these germicide drugs in sufficient quantity to be effectual, and short of this their exhibition is either useless or harmful. The administration of five grains of calomel every or every other night during the first week, or the frequent exhibition of small doses of calomel as an antiseptic during the whole course of the disease was practised by Traube, and afterwards by Wunderlich, Biermer, Liebermeister, and other experienced physicians.

General treatment.—Apart from special treatment of the pyrexia, or of the particular symptoms to be mentioned immediately, it seems to be grateful to most patients to take what is called "a simple febrifuge," such as ten drops of dilute hydrochloric or phosphoric acid in infusion of orange, of calumba, or of serpentary. It is probably a mere placebo, but there is every reason to please as well as to cure our patients.

Treatment of complications.—The most fatal of all accidents, *perforation*, can be only relieved by opium when it has once occurred. The best treatment consists in preventing it, by checking peristalsis, and by scrupulous care in feeding after as well as during the fever. In one case, however, referred to above (p. 124), it appeared likely that a patient recovered from this all but fatal accident under laudanum and brandy.

It might seem that the patient's condition in the third week of enteric fever and the state of the ulcerated and softened bowel would make surgical interference out of the question. Nevertheless laparotomy and suture, or even resection of the perforated gut, have been repeatedly performed; and though the results are usually unsuccessful, they are perhaps not more so than those of treatment by opium.*

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* See a paper by Mr Bowlby read before the Royal Medical and Chirurgical Society, Jan., 1897. He referred to three other successful cases, two in America and one in Russia.

Hæmorrhage should always be regarded as a serious symptom, and arrested, if possible, by gallic acid, by acetate of lead, by ergot, and above all by laudanum, or subcutaneous injection of morphia and ergotin. Turpentine was used by Graves, and is sometimes efficacious, but the chief indication is to stop peristalsis by opiates. According to statistics, the practice of systematic cold baths does not appear to favour intestinal hæmorrhage in enteric fever, but the reverse. An ice-bag may be placed over the right iliac fossa.

In fatal cases which are not cut short by perforation or hæmorrhage the cause of death is usually *failure of the circulation* with hypostatic congestion of the lungs. Beside the free use of stimulants, as described above, great benefit is derived from the common senega and ammonia mixture, or carbonate of ammonia alone in three- to five-grain doses, with its pungency removed by treacle, syrup of tolu, or liquorice, and, if needful, laudanum or paregoric added to prevent laxative effects. With this digitalis may be given, and sometimes its effect on the rapid, feeble, and irregular pulse is striking and most useful; but often it disappoints us, and on the whole is less to be depended on in these cases than when a similar condition of the pulse is produced by valvular disease of the heart.

Diarrhœa should be checked from the first. If there are not more than four loose motions in the twenty-four hours, if the patient is young, and the fever not severe, we may wait; but as soon as this is exceeded, the starch and opium enema of the Pharmacopœia should be administered, and repeated after each movement of the bowels. When pain and restlessness are also present Dover's powder is probably the best form of opiate to give internally, and chalk mixture, catechu, or other astringents are often prescribed with advantage; but in most cases it is better to keep the stomach as much as possible for food and alcohol.

When there is constipation instead of diarrhœa, and especially if there is much flatulence and discomfort, a soap-and-water enema may be administered. Murchison recommends a teaspoonful of castor oil, and repeats it every three or four days if there is constipation throughout the fever. Knowing, however, that in all cases there is some ulceration of the ileum, and that the degree of diarrhœa is no certain guide to its severity, the writer ventures to think it better to err on the side of caution, and to abstain from meddling with the bowels until convalescence is established. He has frequently seen a patient pass through the disease favourably with constipation throughout; he has twice seen such cases die from perforation, and been thankful that even an enema had not been used; and again and again he has seen the bowels act naturally and comfortably after the temperature had fallen, without aid from drugs.

No qualified man would think of giving ordinary laxatives in a case of enteric fever, but we often meet with cases in which harm has been done by saline and other purgatives being taken for a supposed "bilious attack" before the true nature of the disease had been recognised.

Tympanites is not only an unfavourable symptom, but is also harmful in itself. It may be met by turpentine stupes or by enemata containing assafœtida or other carminatives. The distension is chiefly in the colon, and therefore drugs given by the mouth are the less useful; but turpentine in ʒj or ʒij doses in hot spirits and water, or combined with Sp. Chlorof.

Dr Finney, of Baltimore, however, in his paper before the Johns Hopkins Medical Society
147 12 of which ended in recovery.

and almond mixture, is strongly advised by many experienced physicians. Cayley finds the local application of ice to be an efficient treatment of tympanites.

Bedsores ought to be prevented by extreme cleanliness, by careful drying and dusting after each evacuation, and by daily friction with brandy from the first. When the skin is already red and threatens to break, it is better to apply lead lotion frequently. Careful shifting of the patient so as to vary the points of contact as much as possible is part of a skilful nurse's duties. A water-bed is an important preventive.

Treatment during convalescence.—Stimulants should be at once diminished on the subsidence of the fever, and in many cases it is desirable to substitute an ounce or two of wine twice a day for brandy at frequent intervals. With young patients, however, after a favourable attack there is no need for either stimulants or drugs. No solid food should be given for a fortnight after fever and diarrhœa have ceased. The patient will bitterly complain of the restriction, but if the physician has once lost a patient from perforation during convalescence he will be inexorable ever after. Progress to health after enteric fever must be slow if it is to be sure; and it is, as a rule, better to wait until the patient asks to be allowed to take this or that article of food, or to sit up, leave his bed, his room, or his house, rather than to propose these steps in convalescence and find them premature. There is nothing so discouraging as going back on the road to recovery. Leaving the bed, taking meat, going downstairs and out of doors—each stage of recovery must be carefully considered; and there is no disease, not even rheumatic fever, in which it is so important for the patient to have a long period to recover his strength before returning to his ordinary duties.

Prophylaxis.—As we have seen, there is little danger of infection being incurred by doctors or nurses in charge of a case of enterica, if ordinary cleanliness is observed. Either the bacilli in the fæces are not yet become toxic, or they rarely succeed in gaining entrance except when swallowed in water. Nevertheless it is desirable for the patient's stools to be disinfected, so as, if possible, to prevent contagion being conveyed into drains, and thence, by a second want of sanitary supervision, into drinking-water.

For this purpose various methods have been tried. Crude sulphate of iron placed in the bed-pan is cheap and effective; phenol has also been much used, and chlorides of zinc and of lime. Dr Foote, of Yale College, U.S.A., after careful experiments on the sterilising effects of various antiseptics, finds that 5 per cent. solution of carbolic acid (phenol) and 2 per cent. of corrosive sublimate are ineffectual, and that fresh chloride of lime is, on the whole, the most efficient. Corrosive sublimate with excess of hydrochloric acid is very active, but injures leaden pipes.

For the bed and body linen, heat is the only proper disinfectant—boiling, baking, or steaming.

Recently Wright's prophylactic treatment by antityphoid serum has been used on a large scale in India and with satisfactory though not striking results so far. It has also been carried out in the case of many officers and men going out to the present war in Natal (1899).

Prophylaxis of the community, as distinct from the individual, would be sufficient if each case of enteric fever were efficiently guarded from becoming a source of fresh sickness. But this is less practicable than preventing the contagion which is distributed in the fæcal dejections from contaminating drinking-water. This is the business of the sanitary inspector, who insists that drains and sewers shall not leak into the surrounding soil, and so reach the springs, and wells, and streams. By preventing all fæcal contamination we prevent specific bacterial infection. A third measure of defence of the public health from this most preventable disease is to provide an ample supply of pure water for towns, and to stop all other means of obtaining it, at least for dietetic purposes. This has been done for Glasgow, Manchester, and many other great cities with great diminution of mortality from enteric fever; but just as the benefits of Lister's methods were more striking in the hospitals of Paris and Berlin than in those of London, so the effect of supplying pure water from a distance was most remarkably shown in the case of Naples, where this measure alone, with no notable improvement of water-closets or of drainage, has been followed by a great diminution of cases of enteric fever.

Febricula.—When continued fever was no longer regarded as one "disease" with endless varieties, and when the distinction between Typhus and Enteric was established, Relapsing Fever formed a third "species," and a fourth was called Simple Continued Fever, Ephemeral Fever, or Febricula.

This was described by Jenner in his well-known Lectures, and admitted by Murchison among the 'Continued Fevers of Great Britain.' But most physicians now believe that a disease without any morbid anatomy, without any known ætiology, and without any definite or characteristic course or symptoms, cannot be admitted into a useful nosology. This was the opinion of the late Dr Tweedie. In some admirable 'Lectures on Fever' delivered by the late Dr Peacock, of St. Thomas's Hospital, he wrote as follows:—"The cases which have been classed under this head embrace a variety of different affections. Some are probably cases of imperfectly developed typhus, typhoid, or eruptive fever; others may be cases of relapsing fever in which the relapse does not occur; and yet others may depend on common causes—exposure to cold or damp, noxious miasms, or on gastric, intestinal, or hepatic disorder. I should rather regard the so-called ephemeral fevers as abortive attacks of one or other of these forms of disease than as possessing a specific character of their own. You will constantly find that when a series of cases occurs in members of the same family, some are characteristic attacks of typhus or typhoid, while others present only slight febrile symptoms, to which we may apply the term Febricula or Ephemera. Thus in the three cases of typhus which I have before referred to as examples of contagion, and of the varieties in the form of the eruptions on the skin, the first two, those of the patient and the first nurse, were examples of characteristic and severe typhus; the third case, or that of the second nurse affected, was merely a slight febrile attack in which no eruption appeared on the skin. Again, among the typhoid cases is included one which presented the characteristic features of the disease during life, and proved fatal, and after death extensive intestinal disease was found. The brother of this boy was under treatment at the same time, and in his case the fever was only slight; and numerous similar examples might be quoted. We see facts precisely analogous during epidemics of the eruptive fevers, and especially of scarlatina."

This opinion will probably be shared by most scientific physicians, although it was thought needful to retain in the College of Physicians' Nomenclature (1896) "Simple Continued Fever."

It cannot, however, be denied that remarkable cases of fever do occur, which we must at present leave unexplained and without a name.

(1) There are cases of pyrexia and even hyperpyrexia without other signs of fever. Some of them have been referred to already (*supra*, p. 37), which occur in women between the ages of fifteen and forty-five; but others remain. In 1886 there was a patient in Philip Ward, a healthy, decent mechanic, of about forty, who for several weeks suffered (or rather did not apparently suffer) from high temperature, with the normal diurnal variations exaggerated, and sometimes reaching 105·6° F. Yet he ate and slept well, and there was no local lesion to be found. At last the temperature gradually fell, and he went out well. The case was published by Dr Hale White.

(2) Slight cases of enteric fever have often been called febricula (*typhus levissimus* of Hildenbrand). In the absence of the rash, enlargement of the spleen is probably the best diagnostic sign. See an interesting discussion reported in the 'Dublin Journal of Medical Science' for July, October, and November, 1879, by Drs Cameron, Grimshaw, and others; and a valuable paper by Professor Bäumler, of Freiburg-in-Breslau, on the question, "Can the mildest forms of enteric fever be distinguished from acute febrile but non-specific gastro-enteric catarrh?" (*ibid.*, November, 1880). See also Jurgensen's paper in 'Volkmann's Sammlung,' tr. New Syd. Soc.

(3) Influenza without catarrh is probably the true diagnosis of the majority of cases of febricula. They last only one day (*Febris ephemera*), or more often three, five, or seven days (*synocha septimo die soluta*), and often are accompanied by herpes labialis (*f. herpetica*) or by sweating (*f. miliaria*).

(4) Children are apt to be affected by pyrexia, and sometimes by a sharp attack of fever, from what in adults would cause only insignificant disturbance. Slight gastric catarrh from eating indigestible food, diarrhœa from eating raw fruit, from chill, or exposure to heat or fatigue, will be enough to cause febricula, and the diminutive will often apply to the duration only, not to the height of the pyrexia. The "infantile remittent fever" of Evanson and Maunsell is probably almost always enterica without the rash.

In young women (who retain the pathology of children much later than youths of the other sex) similar short and sharp fever is not infrequent from gastro-enteritis due to indigestion or chills, from retention, suppression, or disorder of the menses, and particularly from a degree of sore throat which falls very far short of quinsy.

(5) Again, exposure to the sun, with or without scorching of the face, unaccustomed exertion, or a drinking bout will cause moderate pyrexia, with thirst, anorexia, and malaise. Such are the cases familiar to most of those who notice the effect of a first day's rowing in a hot sun, a first day on snow and glaciers in Switzerland, or a first day's hunting. The same category will probably include cases of febricula which has been described by Irish physicians as fever from fatigue, occurring in haymakers and other field-labourers.

Here may be mentioned the remarkable cases of gastro-enteritis with fever which are sometimes the result of eating bad meat or fish. Some are ptomaine-poisoning, others septicæmia.

It is possible that in some cases the fever is the result of self-infection from products of muscular action or some specific infection from without: but others are sufficiently explained by the presence of a local inflammation.

Murchison, who knew Calcutta and Burma, Sir Ronald Martin and other physicians in the East Indies, describe short and for the most part benign forms of continued fever which are ascribed to heat, and probably correspond to the cases called *Synocha* by the ancients. In India it is known as "ardent continued fever" and "sun fever."

(6) There remain, however, certain epidemic and probably contagious febrile disorders which cannot as yet be brought under any recognised category; they are neither eruptive, malarious, nor symptomatic. The best observed instance with which the writer is acquainted is detailed with great care and judgment by Dr Seaton in the Clinical Society's 'Transactions' for 1886 (vol. xix, p. 26). No less than 157 cases occurred during the four summer months (June to September) in a boys' orphanage. The attack began suddenly with rigor and severe headache, and the temperature rose rapidly to its maximum, which varied from 101° to 105° or even 106°. There was often severe vomiting, with no diarrhœa, but with scanty urine in which the chlorides were remarkably deficient. In most cases the fever ceased in two or three days, in others on the fifth or sixth, and labial herpes usually appeared at the same time. Earache with otorrhœa and pneumonia were the two complications noticed. Death occurred in seven cases. *Post-mortem* examination threw no light on the disease, but it appears to have been only made in a single case.

MEASLES

We bear diseases
Which have their true names only ta'en from beasts,
As the most ulcerous wolf and swinish measle.
WEBSTER.

History and Nomenclature—Ætiology—Incubation—Onset—Course and Eruption—Recovery—Varieties—Complications—Sequelæ — Protection — Diagnosis—Anatomy—Prognosis—Treatment—Prophylaxis.

Synonyms. — Morbilli — Rubeola (obsolete). — *Fr.* Rougeole. — *Germ.* Masern.—*Ital.* Rosolia fersa.—*Arabic* Hasbah.

The name *Morbilli* is Italian, and signifies a little plague, *i. e.* compared to Variola. The plural form, like our Measles, refers to the numerous patches of which the rash is composed. It was constantly used until the middle of the eighteenth century, when Sauvages invented the term *Rubeola* (the red rash, from *ruber*). This name was unfortunately adopted in Great Britain by Cullen and by Willan, and their authority led to its general acceptance. But within the last few years most English writers have reverted to the use of the term *morbilli*. The English word measles, or mesles, means spots, as in the phrase "measly pork."

Definition.—A specific contagious fever with a characteristic exanthem, running a short course, and accompanied by inflammation of the respiratory mucous membrane.

History.—No mention of this common and well-marked disorder is found in the ancient medical writings; but in the ninth century the celebrated Arabian physician Rhazes described Smallpox and Measles in a treatise, which was translated into Syriac, Greek, and Latin, and is still extant.* Measles was long regarded as a minor form of smallpox, from which it was only finally separated by Sydenham in the latter part of the seventeenth century.†

Measles is a strictly specific and contagious disorder, and has been propagated over temperate, warm and cold climates, so that it is now almost pandemic. Its introduction into Iceland, the Faröe Islands, and the

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* Abubekr Mohammed ar-Razi was born at Rai (whence his surname), a town of Irak Ajemi, in Persia, about 850 A.D. His treatise above referred to on the smallpox and measles was translated for the old Sydenham Society from the Arabic by the late Dr Greenhill, 1848.

† Pepys remarks, on the Princess Henrietta falling sick of measles, that this was the third case of the same disease in the Royal family within the year. But he had previously told that the Duke of Glo'ster and the Princess Royal died of smallpox.

Fiji Archipelago took place in recent times. Like smallpox and syphilis, it was much more severe and fatal when thus imported into a virgin soil. Even in England local epidemics may be observed. Sydenham described the prevalence of measles in London in the years 1671 and 1674, and Haslam at Plymouth in 1741. The disease was scarcely recognised on the Continent until the epidemics in Germany at the beginning of the century.

Origin and propagation.—The contagion of measles, though active and sure, is not persistent, and it is readily dissipated by ventilation. It is probably conveyed by the secretion from the affected mucous membranes, by the breath, and perhaps also by the skin after the rash has appeared.

The contagium has not yet been isolated, and it is unknown whether it is a microphyte.[*] Attempts to inoculate it by the blood have failed. It is certainly particulate, and is probably conveyed in the mucus of the nares and by the breath.

Measles is a disease of children. Most persons above puberty have already had it; but cases occur at any age, and unprotected adults take it very readily, and as a rule severely. Infants under six months are not often attacked. Both sexes are equally liable to it. Though now long endemic in England it has epidemic prevalence, regularly about Christmas and midsummer, and irregularly at intervals of several years (Dawson Williams).

If measles occurs among the lower animals, it has not yet been identified.

Incubation.—This has been carefully observed in cases which have occurred on board ship, and in the remarkable epidemic which invaded the Faröe Islands in 1846. Panum (afterwards the eminent professor of physiology at Copenhagen) found that a period of thirteen or fourteen days elapsed between exposure and the appearance of the rash, *i. e.* the incubation period was about ten days.

Further exact observations have shown that although variations occur, they are less than in most other cases of contagion, so that the disease rarely manifests itself before the tenth or after the thirteenth day. When designedly inoculated by mucus from nostril to nostril the period of incubation is shorter, as it is with inoculated variola—eight or nine days.

During this period the child continues apparently in perfect health.

Onset.—Measles begins abruptly, like Scarlatina, Typhus, and Pneumonia, and unlike Enterica. There is anorexia, headache, and malaise, often accompanied by vomiting, by a rigor or (in very young children) by convulsions; sometimes by diarrhœa or epistaxis. On the evening of the first day the temperature reaches 102° or 103°. The course of the fever during the next two or three days is uncertain. On the second or third day the temperature may fall to normal, the appetite may return, and the impending illness is apparently averted. In other cases, however, the temperature remains at about 102°, with only slight oscillations, until the appearance of the exanthem.

There are *catarrhal symptoms* from the first; the patient is troubled with coryza, sneezing, intolerance of light; fluid secretion pours from his eyes and nose, his face and eyelids are swollen, and his conjunctivæ are injected. He is hoarse and coughs, with some wheezing, while sibilant rhonchi are audible on auscultation.

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[*] Messrs Braidwood and Vacher observed minute glistening particles in the mucus of measles which resembled those seen in vaccine lymph ('Path. Trans.,' 1878, vol. xxix, p. 421); and other more [illegible] have described bacilli.

By the second, or at latest by the third day, one finds on looking at the fauces that besides a general injection of the soft palate, there is an eruption of scattered points and spots over its mucous membrane. Ringer attaches some importance to the presence of thin, opaque, white patches on the gums and the inside of the lips. In the pharynx there is a diffused redness, which is seen to reach the larynx by the aid of the laryngoscope. Some writers have regarded this appearance as the indication of an exanthem of the mucous membrane, and have called it an "enanthem." It may be of clinical value in enabling measles to be recognised among the dark races of mankind, in whom no cutaneous exanthem is visible. This slight angina is all but constant.

The tongue is coated with white fur, through which a few red papillæ may perhaps be seen projecting. The pulse is less rapid than in scarlet fever. The respirations are but little increased until the advent of bronchitis or pneumonia. The urine not infrequently shows a trace of albumen, or more than a trace; and this does not appear to add to the gravity of the case. The faucial affection and the early catarrhal symptoms continue into the eruptive stage and then subside. According to Professor Thomas, of Leipzig, indications of the approaching cutaneous rash may sometimes be seen on the face during the prodromal stage, in the form of minute puncta, around which the characteristic papules afterwards develop.

Course.—The *eruptive* stage which succeeds generally begins on the fourth, occasionally on the third day of the illness, or even earlier, *i. e.* from the fourteenth to the seventeenth from infection. In exceptional cases it is postponed until, according to Trousseau, six, seven, or eight days of fever have elapsed; or even, according to Watson, until the tenth day.

The temperature rises again, and in thirty-six hours (as a rule, on the sixth day of the disease) reaches its acme, 104° or 105°. Afterwards it may either begin at once to decline, or may remain near the same point for a day or two. The fall, when it does occur, is rapid, and the normal temperature is reached often before the end of the week, or by the eighth or ninth evening at the latest.

Prof. Thomas studied the course of pyrexia in measles with great care; and his conclusions accord with those of the older physicians, who laid stress on the fact that the fever does not, as in smallpox, abate upon the appearance of the eruption, but sometimes increases. He adds that for the temperature to be low when the rash appears is as unfavourable a sign as for it to keep high when the rash is fading.

Exanthem.—The rash of measles first appears on the face; Ringer says that the earliest traces of it are seen on the forehead close to the scalp. It spreads over the face (not avoiding the parts about the mouth) and then over the whole trunk; on the limbs, especially the lower limbs, it generally comes out rather less freely, but it shows no decided predilection for the flexor surfaces, and it may be well marked upon the palms and the soles. The rash occasionally, according to Dr Goodall, begins on the trunk or nates. The eruption commonly takes three days for its complete development, but sometimes not more than a few hours; the later it is in beginning, the more quickly it diffuses itself over the body. Thomas says that before it has existed at any one spot for twenty-four hours it always begins to decline, so that when it comes out slowly it has faded upon the face and neck before it appears on the more distant parts; but according to Watson it may remain three days at least on the face before its subsidence. The old doctrine that a rapid retrocession of the rash was often followed by some dangerous complication is discredited by most modern writers. But there is sometimes a brief recurrence of the exanthem when from any accidental cause the fever increases.

The colour of the rash is a more or less deep rose or crimson, inclining to purple rather than to scarlet, *i. e.* it is red, with a slight admixture of blue. It consists of spots of irregular form and of varied size. They are at first isolated, but afterwards coalesce into patches, the margins of which are sharply defined, and are here and there "crescentic" in outline. They are slightly raised; even the earliest papules can be distinctly felt with the finger. They have been supposed to be enlarged normal papillæ, or to be the hyperæmic mouths of sebaceous follicles; but Gustav Simon, having excised a portion of the skin from a patient affected with measles, examined it histologically without discovering either of these supposed facts. The latter is certainly not the case. When there is much sweating, a few vesicles of miliaria are sometimes to be seen.

There is usually œdema of the face, and this, with the watery eyes, running nose, and blotched swollen features, makes a picture easily recognised in cases of ordinary severity. Before it subsides, the eruption acquires a yellowish tint, which is particularly well marked when the blood-vessels are temporarily emptied by the pressure of one's finger. This is no doubt the result of the diffusion of altered blood-pigment in small quantity into the substance of the cutis; for even in cases which are doing well it is no uncommon thing for actual hæmorrhage to occur; so that, after the rash has faded, purple stains remain, which afterwards become brown and yellow, and do not finally disappear for two or three weeks.

Slight desquamation takes place, especially from the skin of the face. No large scales are detached, but only a fine mealy powder, which (as Trousseau remarks) is often best seen when one brushes the skin of the patient with one's coat-sleeve, or (according to Ringer) when the surface is stretched and viewed sideways. The process begins on the sixth or seventh day of the fever, and continues for a week or ten days.

Convalescence begins about the end of the week, unless it is delayed by complications or sequelæ. The patient continues contagious until the desquamation is completed.

Varieties.—The course of measles is not always that just described. As with other exanthemata, some cases are slighter than usual, while others are unusually severe, so as to deserve the name of malignant or pernicious.

Measles without a rash, *morbilli sine morbillis*, is a problematical variety, but the eruption is undoubtedly very slight and fleeting in some cases in a school or large family, where others are well marked. The only case in which question might arise would be if during an epidemic an unprotected person should suffer from catarrhal symptoms without rash.*

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* Some writers have asserted that, as in the case of latent scarlet fever, doubt may ultimately be removed by the occurrence of desquamation; but this is so slight, even when the affection of the skin is intense, that one would hardly expect to see it when there has been no eruption at all.—C. H. F.

In some of the mildest and most benign cases there is a rash, but little or no catarrh. Of *morbilli sine catarrho*, Thomas remarks that it is most apt to occur in very young infants, and that it is almost unattended with fever. There is reason to believe that many cases of rubeola, or "German measles," were formerly supposed to be slight measles without catarrh, and perhaps this explains Sir Thomas Watson's remark that the incomplete form of the disease confers no immunity against recurrence. Many will agree with Dr Eustace Smith in doubting whether *morbilli sine catarrho* is measles at all.

The most malignant, and happily the rarest variety of measles is attended with hæmorrhage from the mucous surfaces, and with a *purpuric* form of eruption. At the present day this is occasionally observed in young and sickly children, but is very infrequent, so that when one finds the older writers describing such cases of "black measles," one is almost inclined to suspect them of having mistaken cases of hæmorrhagic smallpox for this disease. It is interesting to notice that Sydenham speaks of an unusually bad kind of measles as prevailing in London in 1670 and 1674, at a time when variola also was remarkably malignant and fatal. The hæmorrhages are said sometimes to begin before the ordinary morbillous rash comes out, sometimes afterwards, and then the exanthem quickly fades or turns of a livid purple colour. Petechiæ and vibices cover the skin, while blood oozes from the mucous membrane of the nose, kidneys, and intestines, as well as into the substance of the deeper tissues and internal organs. Death generally takes place within two or three days.

In other cases measles proves fatal by the severity and prolongation of its usual symptoms. The fever is from the first intense and persistent; the rash, although it may come out early, is of a livid colour, and often develops imperfectly; instead of the temperature falling on the seventh or eighth day, it remains high throughout the second week; the pulse is very rapid and feeble, the patient becomes delirious and drowsy, and passes into a "typhoid" condition with a dry brown tongue and sordes on the lips. Death is preceded by prostration and collapse. After death the lungs are usually found gravely affected, or some other secondary inflammation explains the result.

Complications.—In all but the most severe hæmorrhagic cases of measles, it is not the fever itself but one of its complications that is really the cause of death. Even when the symptoms are not sufficiently marked to lead to its recognition during life, a local lesion is sure to be discovered at the autopsy; and in cases that recover similar affections are very frequent.

Bronchitis with tracheitis and coryza, *i. e.* more or less severe catarrh of the respiratory mucous membrane, is part of the disease, and may be compared to the enteritis of typhoid fever.

Broncho-pneumonia, with hepatisation of scattered lobules throughout the lungs, is the most frequent complication, particularly in certain epidemics; indeed, it occurs so often that some writers have included it in the regular course of the disease. But it is not quite constant. There is nothing specific in the anatomy or effects of this catarrhal inflammation of the lungs. More or less extensive pulmonary collapse often follows in young children. The symptoms of pulmonary inflammation of a dangerous kind are cyanosis, rapid breathing, pallor, and movement of the alæ nasi, with sucking in of the epigastrium and base of the chest.

In some instances true fibrinous lobar pneumonia is found, but this is quite exceptional.

A dangerous but happily rare complication of measles is the formation of a layer of plastic exudation upon the fauces or within the larynx—a secondary membranous laryngitis, which is now admitted to be true specific *diphtheria.*

In other cases the catarrh extends from the fauces along the Eustachian tube, causing *deafness* and earache. As a rule, it seems merely to lead to an accumulation of mucus in the tympanic cavity; but now and then suppuration takes place, and even necrosis of part of the temporal bone. The writer has seen permanent deafness, without suppuration of the tympanum or rupture of its membrane, result from this cause. This inflammation of the middle ear is, however, far less frequent and far less destructive than when it follows Scarlatina.

In many cases *diarrhœa* sets in so severely as to bring about a fatal issue, especially when the evacuations contain blood and mucus or pus. Notwithstanding its severe character, little more than redness of the mucous membrane of the colon may be found after death.

Epistaxis is frequent in measles, and occasionally serious. Apart from catarrh of the conjunctiva, purulent *ophthalmia* is common, and often runs on long after recovery. Dr Fagge once saw a diphtheritic membrane form again and again on the conjunctiva. Sometimes iritis occurs, and sometimes destructive corneitis.

Endocarditis has been described as the result of measles ('Proc. Roy. Med. and Chir. Soc.,' February 24th, 1891), but it was probably caused by preceding rheumatism.

Sequelæ.—Almost any of the complications of measles may be so prolonged as to become a sequela; but there are some other affections which have a better right to that title, since they do not begin until after the patient has recovered from the primary disease. Among these is a form of gangrene, attacking the mouth (*cancrum oris*) or the female genitalia (*noma*). According to Thomas it does not arise spontaneously, but is preceded by some slighter lesion of the same parts, such as a decayed tooth, or infantile leucorrhœa. It is generally but not always fatal. Such cases are among the most frightful that can be witnessed. Even when the first spot of mortification is detected and destroyed by the use of energetic caustics, the child, if it recovers, is more or less deformed for life.

In other cases measles is followed by *necrosis* of a portion of the upper or lower jaw, or by abscesses in the neck.

Children often remain in a state of ill-health for many months after recovery from this malady; and during this time they are apt to be seized with bronchitis or pneumonia, especially in the cold early spring.

The swollen bronchial lymph-glands may become caseous, and thus *tuberculosis* is not unfrequently set up. Indeed, this is one of the commonest and most fatal evil results of measles.

An intercurrent attack of measles is said greatly to accelerate the downward course of a pre-existing phthisis.

On the other hand, eczema, and dermatitis generally, often seem benefited by the sup[illegible] of the exanthem, at [illegible] according to Rilliet and Barthez, the same thing happens in some cases of epilepsy, chorea, and incontinence of urine.

In 1877 there was a case in Guy's Hospital of paralysis probably due to peripheral neuritis which had followed an attack of measles.

Whooping-cough is often a sequel of measles,—more often, according to Dr Goodhart, than its predisposing cause.

Protection.—Measles often fails in preventing a second attack. Even relapses have been recorded, and second or third invasions in a lifetime are not uncommon.

Diagnosis.—This depends on the coryza, and the late appearance, the colour, distribution, and form of the rash. Measles has been mistaken for smallpox; but the symptoms are far less severe, the papules look and feel different, and the non-development of pustules decides the point. It is more often mistaken for scarlatina, but the eruption of measles comes later; it is purple not scarlet, blotched not punctate, and appears first and is most marked on the face. It is often confounded with Rubeola, or German measles, to be described in a following chapter. The student must beware of mistaking the measly rashes caused by drugs for the true exanthem.

Anatomy.—The post-mortem appearances of measles are few, and chiefly due to its complications. The exanthem completely disappears, except perhaps for a little branny desquamation. A little muco-pus may be found in the trachea, and in most cases the immediate cause of death is apparent in the lobular hepatisation and lobular collapse found in both lungs. The pneumococcus may be present in either this common lobular or the less common lobar hepatisation, but it is not constant.

The bronchial lymph-glands are found swollen and soft. The spleen is also somewhat larger than natural.

Occasionally ulceration of the colon is present; but this is often absent, even when there have been dysenteric symptoms during life.

In the purpuric cases ecchymoses are found on the mucous and serous membranes.

Prognosis.—The mortality of measles varies widely in different epidemics. It has sometimes been so low as 2 to 3 per cent. of those who have been attacked, sometimes so high as 50 per cent. Malignant cases, killing within a few days by prostration, with or without a hæmorrhagic rash, are rare—much rarer than the corresponding malignant form of scarlatina; and petechiæ are often seen in ordinary benign cases. Trousseau relates that in 1845 and 1846 he lost from broncho-pneumonia twenty-two out of twenty-four children with measles who were under his care in the Necker Hospital. According to Thomas the few cases which occur during the first six months of life are generally mild; but the disease is more severe in infants during dentition than in older children. Convulsions, if they occur after the rash has appeared, are of ill omen.

Among adults measles is more severe than in children. It is most dangerous to women who are pregnant, or who have recently been confined, and to the very few old people who are susceptible of the disease.

The most dangerous form of measles is when it invades a virgin population as an epidemic. It is then as destructive as Smallpox or the Plague.

In countries where some tolerance is probably inherited, even an epidemic like those of 1834–7, which spread over Europe and the United States, and of 1859-63 in England, are comparatively harmless. But when first introduced into Iceland and the Faröe Isles in 1846, and into Hawaii in 1848, the disease was much more formidable.

The most severe epidemic recorded is that which devastated the Fiji Islands in 1875. It was imported from Sydney in H.M.S. "Dido," and in about four months destroyed 40,000 out of 150,000 inhabitants. Pneumonia and dysentery were the most fatal complications. Since 1875 Measles has been endemic in Fiji, and as innocent as it is in England.

A Peruvian corvette came to London in 1862, and nearly the whole crew were attacked with measles. They were taken into Mark Ward in Guy's Hospital, and more than half died with pneumonia.

Treatment.—The general plan of treatment is that suitable for febrile diseases in general, for we have no specific method. The patient should be confined to the house, but only kept in bed if the temperature is high or the catarrh severe. He should be lightly clothed and allowed to drink as much water as he likes. As Dr Eustace Smith pertinently remarks, if the cup or glass be a small one, and the child allowed to drink it to the bottom, he will be satisfied.

Poultices may be applied to the chest, but with little children a cotton-wool jacket is better, since it interferes less with breathing and does not get cold or slip off.

In ordinary cases of measles no drugs are required. The child should be kept in bed if the temperature is high and given plenty to drink—cold water, lemonade, raspberry vinegar, or any other cooling beverage. If the appetite continues, as it sometimes does, light solid meals may be allowed. When the cough is troublesome, black-currant jelly or sweetened barley water slowly swallowed relieves the irritation of the fauces and thereby the cough. Two or three drops of ipecacuanha wine with ten or fifteen of paregoric, sweetened with syrup of tolu, and given every two hours, promote secretion, and thus may relieve cough and procure sleep.

If the temperature is not above 104°, aconite, thallin, and other antipyretics probably do more harm than good, and cold baths are undesirable; but sponging is of comfort and use. When delirium and high temperature are present cold water should be poured on the child's head while he sits in a tepid bath. Bronchial complications may call for ammonia; and pneumonia or collapse, or great depression with persistent high temperature and rapid pulse, must be met by the free exhibition of brandy. Diarrhœa is not a good symptom, and should be checked by chalk or bismuth, by withdrawing beef-tea and substituting arrowroot, with milk and lime-water. Purgatives at any stage of the malady are undesirable. In severe dyspnœa from bronchitis, laryngitis, or pneumonia, Dr West records recovery after the application of leeches or venesection. Dr Eustace Smith recommends dry cupping, which he believes may save a child from dyspnœa.

During convalescence, the chief dangers are fresh bronchitis, whooping-cough, or the supervention of caseous disease of the bronchial lymph-glands, and consequent phthisis. To guard against these the child should be well fed, and if needful the appetite helped by quinine. Anæmia should be combated by steel, and want of nutrition by cod-liver oil. The body should be warmly clothed in flannel and the child be taken out as much as possible in the open air.

It must be remembered that measles is infectious from the very beginning of the disease, so that isolation should be enforced as soon as its presence is recognised.

SCARLATINA

"This distemper is sometimes so slight as to require no remedies, and sometimes so violent as to admit of no relief."—HEBERDEN.

History and Nomenclature—Distribution—Contagion and transference—incidence on age—surgical and puerperal scarlatina—Incubation—Onset and Course—The throat—The rash—Abortive and malignant varieties; scarlatina anginosa—Complications and sequelæ—Diagnosis—Anatomy—Prognosis—Protection—Prophylaxis—Treatment.

Synonyms.—Scarlet Fever—Morbilli confluentes (Morton, 1700)—Febris rubra (Heberden).—*Fr.* Scarlatine.—*Germ.* Scharlach.—*Ital.* Febbre Scarlatina.

Definition.—A specific contagious fever with a characteristic exanthem and local lesions in the fauces, and often in the kidneys.

History.—Scarlet Fever was recognised and discriminated as a distinct disease by Sydenham in 1675, and the separation from measles was completed by Withering a century later (1778). It had, indeed, been described as far back as 1556 by Ingrassias under the name of Rossalia, at Naples, and again by Döring, at Breslau, in 1627; but even Morton, who was a contemporary of Sydenham, maintained that it was only a variety of measles. The distinctive name *febris scarlatina* (Ital. *scarlatto* = *scarlet*) was given by Sydenham. It is remarkable that he did not mention sore throat as one of its symptoms. Probably the bad cases of *Sc. anginosa* were then called *Cynanche maligna*, a term used as late as the time of Cullen and Heberden.

The name used by the latter writer for ordinary scarlatina, *Febris rubra*, was adopted by the Royal College of Physicians in their 'Nomenclature of Diseases' for 1884 as a synonym of Scarlatina, which is not a classical term. But as neither the name nor the thing was known to Cicero, any distinct term must be in one sense barbarous, and Febris rubra will never supplant the universally adopted and distinctive term imposed on it by Sydenham.

From the persistence and virulence of its contagion and from the severity of its effects, this disease is justly dreaded. Since the introduction of vaccination it has taken the place of smallpox as at once the most common and fatal of all the specific fevers. Its incidence, however, falls chiefly on children, in marked contrast to Enteric Fever.

Its *geographical distribution* is now very wide, but like smallpox and measles, relapsing fever and typhus, it has been introduced from Europe into other parts of the globe within historical times. Thus it appears to have been unknown in the colonies of North America until the year 1737, and in South America for nearly a century later (1829). It was brought to Iceland in 1827, to Greenland in 1847, and about the same time the first cases were recorded in the Australian colonies. In Australia it appears to be as a rule milder than in England. In India it is a rare disease. As with measles, in places which scarlatina rarely visits its character is unusually severe. Thus Darwin ('Voyage of the Beagle,' p. 434) speaks of the terror with which the advance of the disease was regarded at St. Helena.

Ætiology.—The channels and mode of entrance of the contagium are not always the same. The dry epidermic dust of desquamation is probably most often taken in with the air in breathing, or with food and drink, particularly milk, in swallowing. It is often conveyed by clothing or other fomites.

The *contagium vivum* is chiefly present in the epidermis which is shed. It is particulate, but although several forms of micrococcus and bacillus are present in the secretions of the fauces during the disease, and have been described by McKendrick in 1872, and by Pincus in 1883, none have been certainly identified as pathogenic or even as characteristic.

Attempts to reproduce scarlatina in the lower animals by transference of blood or epidermis have repeatedly failed; but investigation into a remarkable epidemic at Hendon in 1885, by Dr Power and Dr Klein, appears to render it probable that Scarlatina may not only be conveyed from one person to another by means of milk, as before ascertained, but that the disease may originate in cattle, and be conveyed from them by their milk to children.

Dr Klein has since, he believes, succeeded in reproducing the disease in cattle ('Proc. R. S.,' March 3rd, 1887) by inoculating them with streptococci obtained by cultivation from the blood of patients suffering from scarlatina, and also by feeding calves with the same microphytes suspended in milk.*

The doubt is not as to the reality of this micrococcus, but as to its specific character. Many of the complications of scarlatina are septic or pyæmic, and it is probable that, as in enterica, there are two infections in scarlatina,—the one constant and specific, the other occasional and pyogenic.

The contagion of scarlatina, though active and very persistent, is not easily diffusible. Thus it rarely crosses a street, and epidemics do not spread so quickly as those of typhus, measles, variola, or diphtheria; but they linger long and die slowly out.

Although scarlet fever never arises but by contagion, yet, as in other cases, there are conditions which predispose to receiving and developing it.

First of these is the *age* of the recipient. It is rare in infants, most common in childhood, and rare again after puberty. Murchison found 64 per cent. of cases to occur between the ages of one and five, but Washbourn and Goodall give the numbers at the hospitals of the Metropolitan Asylums Board to be as follows:—Out of over 80,000 cases from 1871 to 1894 the highest number occurred between five and ten (33,600), next under five (23,000), and next between ten and fifteen (14,300). Between fifteen and thirty the incidence of scarlatina rapidly decreases, and is rare after thirty; but it may occur even in advanced age. When measles and scarlatina are both rife in a town, it has often been remarked that very few unprotected persons escape the former and many the latter.

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* See also a paper by Drs Jamieson and Eddington, with figures of the bacillus which they selected as probably pathogenic ('Brit. Med. Journ.,' June 11th, 1887).

Secondly, puerperal women are peculiarly liable to take the contagion; and, with somewhat masked features, it forms a considerable proportion of the cases formerly known as puerperal fever. Dr Braxton Hicks found that in eighty-nine cases of this terrible disorder no fewer than thirty-seven either showed a scarlatinal rash or had been subjected to infection from patients with scarlatina.

Thirdly, persons who are suffering from wounds are extremely prone to infection. The sore throat and fever are frequently present without the rash, or the rash is slight and quickly over. But that the affection is genuine scarlatina is proved by its "breeding true" and by its protecting against future attacks. Moreover, although most of these cases are mild, like inoculated smallpox, occasionally a severe one occurs along with the rest and exhibits the sequelæ as well as the symptoms of the disease. Prof. Trélat in France and Sir James Paget, Mr Howse, Dr Gee, Dr Eustace Smith, Mr E. C. Stirling, and Dr Goodhart have placed the true nature of this "surgical scarlatina" beyond doubt. It is remarkable that, according to the observation of the last-named author, antiseptic precautions do not prevent the infection; and this throws doubt on the otherwise probable supposition that the contagion gains a direct entrance through the open wound.

The *season* when Scarlatina is usually more prevalent in England is in the autumn months, from the middle of September to the end of November. Dr Moore finds the same rule in Dublin. This was observed by Sydenham, who wrote, "Scarlatina febris licet nullo non tempore possit incidere, ut plurimum tamen exeunte æstivo se prodit" ('Obs. Med.,' sectio vi, cap. 1).

Incubation.—This period is short compared with that of enterica, typhus, measles, or smallpox. It most commonly lasts between two and six or seven days. Occasionally the interval between infection and the first symptoms may be still shorter, twenty-four hours or possibly even less. Among the cases with the shortest incubation are those of puerperal and surgical scarlatina just mentioned. Rare instances occur in which more than a week has elapsed between contact with a case of scarlatina and appearance of its first symptoms, and still more rarely the incubation may be as long as that of smallpox or of measles.*

Course.—The onset of scarlatina is generally sudden. In children the first symptom is often vomiting or convulsions. In adults it is usually sore throat, and there may be chilliness or even rigor. The patient complains of headache, malaise, and prostration.

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* In one case, under my own observation, a child left his father's house, where three of the family had died from scarlatina, on November 19th, and continued well until December 2nd, when he developed the disease, just a fortnight after the last exposure to contagion. It is, however, possible either that the infection was from a different and more recent source, or that it was derived from fomites which, notwithstanding every care, may have been carried away with him from the house. Surgeon-Captain Holl has informed me of a similar [illegible] incubation observed in his own family

The face quickly becomes flushed and the eyes red. The *pulse* is remarkably rapid, and the skin hot. For a child's pulse to be at 140 or 160 within a few hours is not uncommon, nor of unfavourable augury; and it may remain high for several days. This characteristic acceleration of pulse without corresponding quickening of the breath is a good sign of scarlatina before the rash appears.

The *temperature* may rise to 104° or 105° in the course of the first day, or it may attain the same point or a still higher one more slowly afterwards, while the rash is coming out. It then as a rule rests stationary, or nearly so, until the rash begins to fade. The extreme dryness of the surface is apt to give one an impression that it is hotter than is really the case; Addison spoke of the pungent heat of the skin in scarlet fever as comparable only to that of acute pneumonia.

Dr Gee describes the fever as frequently ending in a complete crisis; this occurred in two of his cases on the fourth day, in four on the fifth, and in three on the seventh. But its fall is more often gradual, taking from three to eight days for its completion. After fever is over the temperature is often subnormal for a day or two.

The *urine* is scanty and high-coloured as in other fevers; an abundant precipitate of lithates forms as it cools. Urea is abundant, and the proportion of potash salts is increased. There is often, probably most often, albumen present at this stage, but this febrile albuminuria must be distinguished from that of acute nephritis, which may occur later.

The *tongue* is at first coated with a thick creamy layer, as in other febrile diseases. But before long enlarged fungiform papillæ are seen projecting as shining scarlet points, and after two or three days the white fur clears away from before backwards, leaving a bright red surface; this, with the prominent papillæ that are thickly scattered over it, is what has been called the strawberry tongue. Sometimes, however, no such enlargement of the fungiform papillæ occurs. German writers speak of a miliary vesicular eruption as occasionally present, especially on the dorsal surface. The tongue does not regain its normal aspect until convalescence has set in.

On looking at the *fauces* one finds the arches of the palate, the uvula, and the tonsils more or less reddened or purple. Usually they are swollen; and the redness may extend to the roof of the mouth and to the pharynx. Swelling of the mucous glands often gives a granular appearance to the affected parts; and their secretion may accumulate upon the surface so as to simulate the presence of ulcers. The tonsils may project inward until, with the club-shaped uvula, they block up the passage. After a few days one or both of them may suppurate; and one tonsil is often more affected than the other. There is also more or less swelling of the neighbouring cervical lymph-glands.

Exanthem.—The rash generally makes its appearance in from twelve to thirty hours after the commencement of the disease. Sometimes it is later; but, as Dr Gee remarks, the proof of this is difficult, not only because the first signs of it are easily overlooked, but because it may recede for a time, and then come out again. In some of the more severe cases it is ill-developed, and (it is said) may be delayed until the third or the fourth day. In the great majority of cases the rash begins to appear towards the end of the first day, and is fully out on the second.

As a rule, the eruption is first to be discovered upon the sides of the neck and the upper part of the chest. It generally takes twelve or twenty-four hours, or even two or three days, to reach its full development; but sometimes it comes out almost at once over a very large surface. The face often remains free; and, when present, the rash is in most cases limited to the forehead and temples, the cheeks showing only the ordinary flush of fever, while the parts round the mouth, nearly to the chin, are pale. This "circumoral ring" is perhaps only due to contrast. The palms and soles do not show the rash owing to the thickness of the cuticle. The upper arms are often covered with the eruption, and it is exceedingly well marked on the abdomen and the inner surface of the thighs.

The colour is usually a bright scarlet, so that Watson compared it with that of a boiled lobster; but sometimes it is of a lighter pink, and sometimes duller. The redness appears to be uniformly diffused, but on careful examination one can see that it is made up of very minute red points, which are at first isolated, and, even when they have coalesced, often leave an islet of healthy skin here and there. This minutely punctated appearance is even more distinct from the "blotchy" aspect of the rash of measles than the bright crimson of the one from the rose tint of the other; but now and then we see the scarlatinal eruption, though confluent on the trunk, as blotchy as measles on the limbs. The rash disappears on pressure for a moment, except where there has been some degree of hæmorrhage or perhaps capillary stasis, in which case isolated red spots remain, with a more or less general yellow discoloration. As Watson long ago remarked, on the forearms and the legs, as well as on the backs of the hands and of the feet, the rash of scarlet fever consists of larger and more prominent papules than elsewhere. The skin of the affected parts is slightly swollen; the eyelids and the cheeks look a little puffy; and Trousseau has seen the fingers so swollen as to prevent the patient from closing his hand.

Löschner discovered exudation-cells in the rete Malpighii, and they were seen by Dr Fenwick also, who further observed that the basement membrane of the sweat-glands was thickened, and their channels were obstructed by an overgrowth of epithelium, or by extravasated blood. That the scarlet fever eruption bears no definite relation to these glands, nor to the hair-follicles, seems to follow from a case (cited by Thomas on the authority of Landenberger) in which it did not fail to develop itself over an immense cicatrix, the result of a burn which was said to have destroyed the whole thickness of the skin.

Certain modifications of the eruption of scarlet fever are sometimes observed. Instead of being punctiform, it may in rare cases consist of large, irregular, slightly raised maculæ, more or less like those of measles, or it may be marked papular. Again, it may be associated with immense numbers of miliary vesicles, especially upon the neck and chest, when there has been much perspiration.

At a variable period after the subsidence of the rash—which has usually faded by the end of the first week, often on the fifth day and sometimes earlier—the superficial layers of the cuticle begin to peel off, or, in technical language, to *desquamate*. This is sometimes observed within a few days, sometimes not until two or three weeks have elapsed. Its amount is not always proportioned to the intensity of the cutaneous affection; it may be well marked where the rash was so slight as to have been overlooked, and so may be of service in clinching a doubtful diagnosis. In some cases it assumes a furfuraceous form; a dusty powder or a fine scurf over the whole surface, especially the face. But more generally it is membranaceous;" distinct flakes come away, and may be very large. The first step towards the throwing off the epidermis at a particular spot is often the formation of a little opaque raised pseudo-vesicle, very like those of eczema, but dry; this breaks at the summit, leaving a free edge in the shape of a ring, which gradually becomes larger and larger. The cuticle of the hands is now and then shed altogether like a glove; and the same may be the case with the feet; in some cases not even the nails are left behind. So complete a desquamation as this takes several weeks for its completion.

The effect of the exanthem on the nutrition of the nails is usually shown, not by their exfoliation, but by the formation of a transverse groove, to which Sir Samuel Wilks long ago drew attention. It is seen on several of the nails at equal distances from their roots, and of course it ultimately becomes lost at their distal extremities. Such grooves on the nails are not peculiar to scarlet fever, being sometimes seen after other acute diseases; but even with this qualification their presence sometimes throws valuable light on the origin of sequelæ, the real nature of which might otherwise have been doubtful. The hair comes off with the cuticle, but perhaps not more than after any other fever of equal severity.

Desquamation follows the same course as the rash, beginning on the chest, shoulders, and neck, then spreading over the arms and back, and then over the lower extremities. It is often delayed in the hands and feet until long after the rest of the skin is clear.

Secondary rash.—Occasionally, long after the characteristic exanthem has disappeared and while desquamation is completed or in progress, in the second or third week after the onset of the fever, or even later, a second eruption appears. Dr Caiger calls it a morbilliform eruption of septic origin. Washbourn and Goodall describe it as beginning on the extensor surface of the arms and legs and on the nates, papular and discrete at first, but soon running into blotches; it lasts a week or more. Another form is that of a bright rose-rash, and a third is urticarial. These secondary rashes usually occur in anginal or nephritic cases.

Subsequent course.—In the majority of cases after about a week of high fever the temperature rapidly falls (often on the sixth or fifth, occasionally not till the eighth or ninth day), and the pulse with it. While the rash is fading the tongue cleans, the throat recovers, the swollen cervical lymph-glands subside, and the appetite returns.

When the sore throat is severe, or when scarlatina is complicated by diphtheria (*Sc. anginosa*), there is a second rise of temperature, and the disease may last ten days, a fortnight, or longer before convalescence is established.

Other complications and sequelæ, also described below, may prolong the sickness of the patient. Or its duration may be less than a week, and that from two causes—the extreme mildness or the extreme and fatal rapidity of the disease.

As remarked by Heberden in the words which stand at the head of this chapter, a striking feature of scarlet fever is the great variability of its severity and of its course. In this, as in some other characters, it resembles enteric fever: like it, it may be very mild and may be most severe; it is always treacherous and uncertain, and when the fever has subsided the mischief is far from passed, for sequelæ are frequent and sometimes dangerous.

Slight forms.—In some cases the disease is rudimentary or abortive. Thus, during epidemics, it often happens that adults become more or less feverish, complain of a slight sore throat with redness of the mucous membrane, and have a little pain and swelling of the cervical glands, but are at no time really ill. This modified scarlatina occurs in those who have already had an attack in childhood. Some doctors and nurses are again and again subject to it when attending patients suffering under scarlet fever.*

Again, the chief symptom may be feverishness, so that the complaint passes for "a mere febricula," there being little or no affection of the throat, and no redness of the skin beyond what might escape notice; or the only symptom may be an eruption, and this perhaps limited in extent. The real nature of this affection is often first shown by the occurrence of desquamation, or by the unforeseen supervention of dropsy or of otorrhœa.

Malignant scarlatina.—In a very different class of cases the symptoms are incomplete because the end comes too quickly for their full development. In several recorded instances the patients have died in from eight to fifteen hours, the brief and terrible course of the disease being marked by convulsions, by tonic spasms with trismus, by delirium or coma, or by incessant vomiting and diarrhœa, with extreme rapidity of pulse and of breathing. Sometimes there is hyperpyrexia, but this is not constant.

The writer saw a little boy with Dr Andrews in 1883, who was taken ill the same morning with severe headache, vomiting, and prostration. When seen he was already comatose, but there was no exanthem. The temperature was 105·6°, the skin was pungently hot, and the pulse too rapid to be counted. Such a condition could be due only to smallpox or scarlatina, and the presence of good vaccination marks, together with the characters of the skin and pulse, justified the latter diagnosis. He died the same afternoon, and the nature of the case was confirmed by the child's nurse afterwards sickening of scarlet fever.

The cases just referred to are the most extreme examples of malignant scarlet fever. But there are other varieties almost as certainly though less rapidly fatal. Sometimes, after early symptoms of a "typhoid" character, the rash comes out late and imperfectly, and its hue is not bright scarlet but a livid violet. In other cases the disease assumes a *hæmorrhagic* form, and this is a most dangerous symptom.

Sometimes, again, the eruption develops itself in the regular way; but the febrile disturbance, which from the first is high, runs on for two or three weeks instead of subsiding after a few days. The patient then falls into a typhoid state, with muttering delirium, a dry brown tongue, and sordes upon the lips. Many of these cases end fatally.

Scarlatina anginosa.—In certain cases the affection of the throat is unusually severe. Sometimes the fauces become gangrenous and slough away, leaving a horribly offensive cavity. The inflammation often spreads to the lining membrane of the nose, and an acrid fluid flows from the nostrils, which excoriates the upper lip. The nasal bones even may become necrotic. Still more frequent is extension to the ear along the Eustach tube; suppurative tympanitis is set up, with intense earache, until pus is discharged by perforation of the membrane. Here, again, there often great destruction of the tissues: Sir Thomas Watson mentions a c in which, every time that the child swallowed, some of the liquid ran at one of its ears. It is no uncommon thing for fatal hæmorrhage to en from perforation of the carotid artery itself or of its ascending pharyng branch.

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* I once saw an instance in which a father so affected gave to his children a disease of such severity that one of them died.—C. H. F.

Faucial diphtheria is a very dangerous complication, and sometim occurs in almost every case of a local epidemic.

In other cases the chief local mischief is outside the throat, in the c vical lymph-glands and the adjacent structures. A large swelling may for on each side of the neck near the angle of the jaw; or the whole spa from the chin to the sternum may be occupied by a brawny shining ma which has been termed a "collar." The salivary glands, parotid and su maxillary, do not appear to take any part in the process. Induration of t connective tissue may spread backwards between the pharynx and t spine, or downwards into the mediastinum. If suppuration occurs, and t abscess is not early opened, the pus may be discharged by fistulous op ings, which burrow and undermine the skin in all directions; or a po pharyngeal abscess may be formed and cause suffocation by pressu Extensive sloughing sometimes takes place, so that the muscles are l bare, as in a clean dissection; and fatal hæmorrhage may ensue from p foration of an artery or vein in the neck.

Complications and sequelæ.—The affection just described might reckoned as a complication of scarlet fever, but it is an exaggeration c constant feature, the faucial inflammation. Another complication is c paratively rare, and is unrepresented in the normal course of the dise This is *synovitis*, a painful swelling of some of the joints, and it is by means confined to cases of great severity. It generally sets in while skin is peeling, and affects sometimes the smaller, sometimes the lar articulations; it generally subsides quickly, and it is even more fugit than the synovitis of ordinary rheumatism. In some cases, however settles into one particular joint, leading to chronic effusion, or even suppuration. Probably most cases of synovitis following scarlatina true rheumatism, as shown by the previous or subsequent occurrence rheumatic fever, and by its affecting the peri- and endocardium. suppurative synovitis of one joint is probably not rheumatic but pyæmic

Dr Ashby, of Manchester, says that synovitis *in the course of the feve* very rare and unaccompanied by evidence of cardiac inflammation, and t endocarditis (rheumatic or nephritic) is less common as a sequel than d tation of the heart.[*]

A more frequent complication of scarlet fever is acute *nephritis*, acc panied with albuminuria. Whether any affection of the kidney is pres in ordinary cases of scarlet fever, which end in recovery, is not prove but it is certain that nephritis has been found after death in some exc tional cases of scarlatinal dropsy in which the urine, although scanty, c tained neither albumen nor casts during life. This fact makes it probable that renal changes may be present in cases which recover, without albuminuria or dropsy or hæmaturia. It is also certain that the more assiduously the urine is tested throughout the whole course of the disease, the more numerous are the cases in which a trace of albumen is detected at one time or another. Thus some German writers look upon a renal catarrh as bearing to scarlet fever the same relation which bronchial catarrh bears to measles. But albumen and occasionally hyaline casts are found in the urine in other fevers, so that their presence does not prove the action of a specific poison upon the kidneys.

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[*] 'Lancet,' May 22nd, 1886. See also Dr Goodhart's paper on "Sudden Death f Dilatation of the Heart in Scarlet Fever" ('Guy's Hosp. Reports,' 3rd series, xxiv), Ashley and Wright's 'Diseases of Children.' Compare the report from Glasgow ('Clin Trans.,' 1885–6).

Dr R. S. Thomson ('Med.-Chir. Trans.,' lxix) found in 180 consecutive cases in the Glasgow Fever Hospital (1882-3) albumen or hæmoglobin in 112, besides 2 in which there was dropsy without albumen.

Dr Sweeting found albuminuria in from 14 to 35 per cent. during 1882-8 among pauper patients with scarlatina in the Western Hospital of the Metropolitan Asylums Board.

Dr D. A. Gresswell found albuminuria in the epidemic of scarlatina in London of 1887-8 in half the patients, observed while still in bed, during the months of December and January, and in every case during October and November (600 in all).

Dr E. W. Goodall ('Guy's Hospital Reports,' vol. xlvi, 1889) found in patients with scarlatina at the London Fever Hospital that slight early (febrile) albuminuria, without other sign of nephritis, was present in the great majority of cases, perhaps in nearly all; but that albuminuria beginning in the third week, or later, accompanied by blood or casts or dropsy, only occurred in 8·7 per cent. He observed that it was most frequent between five and nine and most fatal in male children under five.

On the whole, we are probably right in separating the slight febrile albuminuria pathologically as well as clinically from the hæmaturia or albuminuria with dropsy which follows the fever in a minority of cases. The one corresponds anatomically to "cloudy swelling," the other to the "large red kidney."

Scarlatinal *dropsy* and *uræmia* will be discussed with the anasarca that results from other acute forms of tubal nephritis. Anasarca without albuminuria is occasionally met with after scarlet fever.

Serous inflammation.—Pleurisy and pericarditis are of not infrequent occurrence, usually in connection with the affection of the joints, and this confirms its rheumatic character. The same is true of endocarditis, which often produces chronic changes in the valves of the heart; whether this ever occurs without synovitis is very doubtful.*

Pleurisy and pericarditis, or pneumonia, may also accompany scarlatinal nephritis; they are obviously renal, not rheumatic in origin.

Chronic enlargement of the *tonsils* is very common in children who have passed through scarlatina. Stomatitis and eczema or impetigo are less frequent sequelæ.

Otitis.—Far more important sequelæ affect the *ear*. Indeed, scarlatina is by far the more frequent origin of deafness acquired in early life. Not only during but after an attack, suppurative tympanitis may spread from the Eustachian tube, and lead to long subsequent pyæmia; or it may years after cause fatal meningitis or abscess of the brain. Hence the importance of otorrhœa in insurance practice.

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* In making *post-mortem* examinations of children who have recently had scarlet fever without synovitis, I have always found the valves healthy.—C. H. F.

Necrosis of other bones is not very uncommon, and glandular abscesses sometimes follow during convalescence.

An important complication, sometimes occurring in the first week, sometimes not until convalescence, is *diarrhœa*. It occasionally leads to fatal exhaustion.

Icterus is an occasional complication of the most septic cases, but as a sequela the writer has seen good recovery from jaundice after scarlatina.

Diagnosis.—The sudden onset and early high temperature, the rapid pulse and burning skin, and the appearance of an angina usually decide the nature of the fever on the first day. The development of the rash on the following day, its appearance, course, and distribution, are in most cases decisive.

Difficulties occur chiefly with the very mild cases, and here we must sometimes hesitate until desquamation or albumen throws light on the case.

Measles are not really like scarlatina, in either the exanthem, or the chronology, or the symptoms; but rubella may closely resemble it, as will be described in the following chapter.

The sore throat alone may be exactly like that of diphtheria, but the rash is distinctive, and scarlatinal angina is not followed by paralysis.

The exanthem may be closely simulated by the rashes caused by belladonna, by copaiba, quinine, or other drugs; but the only danger of mistaking these toxic rashes for scarlatina is forgetting their existence.

Anatomy.—Nothing distinctive is found on examining the body after death from scarlatina, beyond the appearances due to fever as such, and those produced by any complications which may have been present during life. Internal hæmorrhages are common in septic cases. No trace of the rash remains, although the presence of desquamating cuticle may give a hint of the nature of the disease. The inflammation of the fauces, even with ulceration, leaves surprisingly slight effects. Occasionally the heart is found in a state of acute dilatation, as after some cases of diphtheria. There are not infrequently signs of intestinal catarrh, with more or less swelling of Peyer's patches. The kidneys may merely show a greyish pale aspect on section, as if boiled, with granular swelling of the epithelium on microscopic examination, or they may show the characters, to be described under acute Bright's disease, of either glomerular nephritis or ordinary tubal inflammation.

Prognosis.—The prognosis of scarlet fever is never free from anxiety, for in the mildest case some dangerous complication may arise. Sporadic cases are sometimes as severe as those which form part of an epidemic; and epidemics differ widely in their gravity. One prevailed for many months in a country town in Kent without there being a single death or a single case of dropsy; and Dr George Turner observed an epidemic of 120 cases at Portsmouth without a death. Graves recorded the fact that whenever scarlet fever appeared in Dublin between 1805 and 1833 it was always mild, so that on one occasion eighty children were attacked in the same public institution, and all recovered; but during 1834 and 1835 the city was the seat of a very malignant and fatal epidemic.

Thomas says that in Saxony a mortality of from 13 to 18 per cent. is very common, but that it not infrequently rises to 25, and sometimes even to 30 or 40 per cent.

In the London Fever Hospital Dr Goodall found among 55,443 cases of scarlet fever (1879—1888) that 297 was the total number of deaths, *i. e.* 5·45 per cent., made up of 6·1 for male and 4·8 for female cases.

At the same hospital during an earlier period Dr Collie (1871—1878) found the total mortality 9·5, while at Stockwell Fever Hospital it was 13·2, and at Homerton 13·4.

These last figures are higher than usual, but the mortality of scarlet fever varies greatly in different epidemics. At Tours, in the time of Bretonneau, great contrasts were observed in this respect, and in 1874 an epidemic at Melbourne was of most unusual severity for Australia. Still, in England there has certainly been a gradual decrease in the total case-mortality.

Scarlatina is most fatal under five years of age, and in adults is usually less severe than in children. In puerperal women, however, it is notoriously dangerous, though far from being always fatal. It is, in fact, one of the many pathological conditions formerly confounded together as puerperal fever.

Apart from the individual danger of a case of scarlatina, the just apprehension with which it is viewed is also due to its powerful contagion. Now that plague and typhus are happily obsolete, and smallpox checked by vaccination, there is no disease but scarlatina and diphtheria which enters a household and destroys a majority, or sometimes the whole, of a young family. The following is an instance of the terrible virulence of the disease.

The writer was sent for into the country to see a child with a febrile exanthem, which, unfortunately, had been called measles by the medical attendant. On arriving at the house he was informed that the child (an infant) had already died, but that another was very ill. This second case was undoubted scarlatina. A third child had already showed febrile symptoms, and died in the course of that night. A fourth afterwards had a very severe attack, but recovered. The eldest boy alone, who was happily away from home, escaped the disease; but of the other four, three died within forty-eight hours.

Most of the inferences to be drawn from particular symptoms have ready been indicated. But it may be added that, according to Trousseau, e occurrence of convulsions during the first or second day is always a sign danger, whereas in other exanthemata it is not of evil omen. Another ognostic made by Trousseau is that in scarlet fever, as in smallpox, e more intense the rash the more severe is the disease. Here, however, differs from most other observers; and the statement perhaps only eant that those cases in which a bright red eruption covers the whole of e body, and lasts for an unusually long time, are generally grave ones, hereas when the eruption is slight and quickly over the fever also is oderate and of brief duration.

Excluding malignant cases, the greatest immediate dangers are from vere faucial inflammation, and, above all, from diphtherial inflammation, om acute nephritis, and from dilatation of the left ventricle. The sub-quent dangers are from chronic Bright's disease and f

Protection and relapses.—This is less complete than in the case of enterica, smallpox, typhus, or perhaps measles. Second attacks are, however, very uncommon. Relapses, though not unrecorded, are certainly rare.*

Treatment.—We have no means of *prophylaxis* but isolation. Belladonna was supposed to be indicated by the fact that poisoning by belladonna produces dryness of the throat and a roseolous eruption, just as in former times red curtains were hung round a patient's bed because of the colour of his skin. It has, however, been fully tried, and conclusively proved to be useless. Arsenic has been credited with a similar power, but, unfortunately, without good ground. Decoction of cinnamon is the last prophylactic recommended.

When the contagion has been once taken in, we must be content to treat scarlatina on the same general principles as those indicated in the chapter on Enteric Fever.

In mild cases little is needed beyond careful isolation and free diluents. Tepid baths and sponging the surface are grateful in almost every case.

When the temperature is very high, 104° or upwards, we must check it by cold sponging, wet packs, rubbing with ice, or cold baths, as described above (pp. 152-3).

The angina causes much pain and difficulty in swallowing. Sucking ice gives relief in many cases; and, if the patient will allow it without too exhausting a struggle, it is useful to clear the throat with a large camel-hair brush or with cotton wool firmly tied on to a penholder; or a spray medicated with thymol, carbolic acid, or some other disinfectant, may be employed. Older patients may use Condy's fluid or chlorate of potash as a gargle, or borax and honey as a linctus, with advantage. In severe cases nutrient enemata or suppositories may be tried; and if they fail, as they unfortunately often do, the patient must be fed by the nose; not with a catheter—a child's nasal passages are too small, and the process would be too long,—but by inserting a small glass funnel, of the kind used for filtration in laboratories, into the opening of one nostril, closing the other, and pouring milk and egg, beef-tea, or other fluid nourishment into the pharynx. An aural speculum answers the purpose well.

Brandy must be given if the pulse requires it, and must be given freely in anginal and septic cases. Quinine in doses of five grains three times a day for a child of five years old is recommended by Dr Eustace Smith. Chlorate of potash is also of undoubted benefit in many cases of scarlatina anginosa.

As soon as the eruption is fully out, the whole surface should be anointed with carbolic oil (1 in 30); and when the fever has subsided and desquamation has appeared the hair should be cut short, and the whole body well washed with hot soap and water, followed by inunction. A warm bath with soap may then be given daily. The oil is not only pleasant to the patient, but prevents the desquamating scales from flying about.

Earache should at once lead to examination of the meatus, and attempts may be made by hot water injections or a leech on the mastoid ocess to relieve pain and inflammation. If the membrane is already per- rated the cavity should be washed out with warm boracic, permanganate, r dilute carbolic lotion. If suppuration has already taken place the membrana tympani should be incised with a sharp knife; tension and pain are thus relieved, and in favourable cases the cut heals perfectly.

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* The general experience is that scarlatina is very little liable to relapse. Cases, however, are cited by Rilliet and Barthez, by Jaccoud in France, by Dr Brodie of Edinburgh and Dr Hopwood of the London Fever Hospital, by Henoch at Berlin, and by Thomas and Körner at Leipzig. Goodall and Washbourn relate a case, but say that they are uncommon; Dr Caiger, however, thinks that they not infrequently occur.

Great care should be taken during the stage of peeling to keep the patient from draughts. The body should be clothed in flannel, and any chill carefully guarded against. The urine should be daily tested, and while it contains albumen the patient should not be allowed to leave his bed or to take meat.

If, in spite of precautions, nephritis and dropsy follow, the case is one of acute Bright's disease, and must be treated by purges and other means, which will be detailed in the second volume.

Rheumatism as a sequela is amenable to salicyl compounds—another proof of its true nature.

During convalescence tincture of steel is the most useful drug, and port wine the best form of stimulant, when it is required. Even in mild cases the child should keep his bed for three weeks and his room for a month at least from the beginning of his illness (Eustace Smith).

The rules for disinfection given on pp. 28, 29 must be thoroughly carried out. The patient must not associate with other children until the desquamation has ceased, except perhaps that on the soles of the feet, which sometimes lasts for weeks after the rest of the surface is clear.

The infection of scarlatina begins later and lasts longer than that of measles or of smallpox, probably for six weeks, or even more.

RUBELLA

Facies non omnibus una,
Nec diversa tamen, qualem decet esse sororum.
OVID.

Recognition and nomenclature—Its characteristics—Incubation—Onset and course—Diagnosis from morbilli, scarlatina, and roseola—Protection—Prognosis and treatment.

Synonyms.—Rubeola, Rubeola notha, bastard measles (Babington); German measles.—*Fr.* Roséole épidémique.—*Germ.* Rötheln.

Definition.—A specific contagious fever of short and usually benign course, with an exanthem.

History.—After scarlet fever had been recognised as distinct from measles, German physicians in the latter half of the eighteenth century began to describe a third member of the same group of diseases, for which they adopted the term "Rubeola." The relation of this Rubeola or Rötheln*—German or "bastard" measles—to Morbilli or Masern—"true measles"—has been the subject of much controversy.

In this country, Dr Paterson, of Leith, described the supposed third exanthem in the 'Edinburgh Medical Journal' for 1840, and among English writers it has since been recognised by Murchison, Squire, Bristowe, and most other writers.

The term "epidemic roseola," adopted by Squire and by Bristowe from Trousseau, conveys the wrong impression that the disease bears some relation to other eruptions known as roseola. Moreover Trousseau believed that his "roséole épidémique" did not correspond with Rötheln or *Rubeola notha*, and there are epidemics of Roseola in England which are certainly not Rubella.

It would perhaps be best to use the word Rubeola, first invented by Sauvage as a synonym of morbilli, and afterwards appropriated to "German measles" by Copland. Measles in English, and *morbilli* in Latin, are satisfactory names, distinctive and unambiguous. It is a pity to lose a word which has already been applied to a disease in want of a name, and which is a name in want of a disease. Moreover Rubeola is the accepted equivalent of Rötheln with German authors, and is adopted by Aitken, Liveing, and other good authors.

Nevertheless Rubella is the term finally adopted by the Royal College of Physicians, with Rubeola as a synonym, and this form is accepted by Dr Osler and by Dr Dawson Williams. It is therefore used in the present edition of this chapter.

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* According to Seitz, *Rötheln* is a local or dialectic name for ordinary measles (*Masern*) in many parts of Germany.

Rubella, then, is a specific and infectious febrile disease, occurring in epidemics, arising from contagion and breeding true, accompanied by an exanthem, and protecting against itself.

Its most essential features are that, with catarrhal symptoms like those of measles, and a variable but distinct rash, it resembles scarlet fever in having a very short prodromal stage, and in being attended with marked sore throat.

Schönlein and other former writers thought that it was a "hybrid" between these two diseases; Niemeyer wrote of the symptoms alternating in different cases, so that sometimes the rash of measles would be associated with the local lesion of scarlet fever, and sometimes the reverse would be the case. But this was purely fanciful. There is no doubt that two specific exanthems may exist together in the same patient, but they do not combine so as to produce a modified affection; still less could such an affection be epidemic, or protect against itself while affording no immunity against either of the constituent diseases.

In the first edition of this work Dr Fagge contrasted the account given by Paterson, and accepted by Aitken and other systematic writers, with that of German authors, and especially with Professor Thomas's article in Ziemssen's 'Cyclopædia,' based on two epidemics at Leipzig in the years 1868 and 1872. In the former account we read of marked disturbance at the invasion, high fever, severe catarrh, with œdema of the glottis, bronchitis, severe angina with dysphagia, and a raised blotchy eruption appearing on the third or fourth day. Death, we are told, was not infrequent from suffocation or from convulsions.

The discrepancy of this account from that given of Rubeola by Thomas and other German writers, and now recognised in this country, must be admitted. Possibly Paterson's epidemic was not rubeola at all, but measles, which, as we have seen, is sometimes unusually malignant (p. 164).

However this may be, there can be no doubt that the disease described by Thomas, Eichhorst, and other German writers, by Bristowe, Liveing, Cheadle, Eustace Smith, Goodhart, and Dawson Williams,* is one and the same, different both from measles and from scarlet fever. There is nothing surprising in the severity of the symptoms varying widely. For we have seen that both Measles and Scarlatina, the nearest allies of Rubella, are sometimes almost trivial in their symptoms, and only formidable from their contagion, while at others they are as fatal as smallpox or typhus. The rule is undoubtedly for Rubella to run a mild and benignant course, but a case which will presently be narrated shows that this rule is liable to exceptions.

There was an epidemic among the nurses at Guy's Hospital in 1885, which satisfied all who saw the patients of the reality of the disease. The cases differed considerably in severity and in the character of the rash. Sore throat was always present, and catarrh was much less frequent, so that it was distinct from measles; while the absence of the characteristic signs of scarlatina and of any severe cases prevented confusion in that direction. Some of the mildest cases, when the patients were about, and therefore the rash not obvious, might have been set down as "epidemic sore throat;" others, if the throat had not been looked at, might have been called Roseola.

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* The reader may also refer to a succinct but full account of an epidemic of fifty cases by Dr Douglas, of Newbury ('Brit. Med. Journ.,' May 26th, 1877); to a short paper by Dr Ryle (*ibid.*, July 24th, 1886); and to an account of an outbreak in the City Hospital of Birmingham in 1890, by Dr N. S. Manning ('London Medical Recorder,' April 20th, 1891, p. 121).

Rubella occurs in the British Isles and on the Continent, in Egypt, in India,* and in America.

Origin and incubation.—All writers agree that rubeola arises strictly by contagion from other cases. The most important predisposing cause is age; it is most frequent about puberty and soon after, is rare in young children, and not common in adults. Boys in a family are said to escape oftener than girls.

The incubation stage is variable, but probably about as long as that of measles. In an epidemic at Guy's Hospital in 1888 several of our students were attacked, and among them the shortest incubation period observed was eight days, while one was as long as seventeen days. Of 86 cases observed with respect to incubation, it lasted from a week to ten days in 32, from eleven to fourteen days in 45, and two or three days longer in 9 only. Bristowe puts it at about a week, or rather less; Squire about ten days; Liveing ten to fourteen days; Manning two weeks, more or less; Douglas fourteen or fifteen days, seldom more, and only three less than a fortnight; while Thomas of Leipzig gives a fortnight, or even three weeks. Dr Clement Dukes, whose opportunities as medical officer of Rugby School were exceptional and exceptionally used, states that out of twenty-four carefully observed cases the incubation lasted twelve days in two, and from fourteen to twenty-two in the rest.

Dr Haig-Brown recorded an epidemic of rubeola at the Charterhou School in the 'British Medical Journal' for April 16th, 1887. He four the infection is apparently less active than that of measles and less persi tent than that of scarlet fever, for more inmates of a house or school escap during an epidemic of rubeola than during one of measles, and cases seldo occur after an interval of cessation.

Nothing is exactly known of the nature or the mode of conveyance o the contagion.

Onset and course.—As a rule the first symptoms are not severe. Ther is usually little or no catarrh before the rash, and the fever is not high.

The *exanthem* appears early; often it is the first symptom noticed, an it is scarcely ever delayed beyond twenty-four hours after the temperatur rises. Among 159 cases it was out on the first day in 119, and on th second in 39. It is more like the rash of measles than that of scarl tina, but it is of a brighter rose tint; it is patchy, but less mottled an more diffuse than measles, while it is less vivid red, less diffused, and les punctuate than scarlet fever. It appears earliest on the forehead an cheeks, and afterwards the trunk, particularly the back. It is not markedly developed on the face as measles. The upper arms are affecte the nates and the thighs; but seldom the hands and feet. There is ofte troublesome itching. It spreads more rapidly and less regularly than th other two exanthems.

Sometimes the eruption lasts three days or more, longer than measle but never so long as scarlatina. Desquamation is usually present, b needs careful looking for. It never resembles that of scarlet fever, and is often altogether absent.

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* Surgeon-Major McLeod read a paper before the Epidemiological Society (Feb. 11t 1885) on an epidemic rose-rash which visited Calcutta in 1881, and which he right decided to be rubeola (*i.e.* rubella).

With the rash there is, in probably every case, some amount of angina; the fauces are injected and swollen; but there is never sloughing, and the lymph-glands at the angles of the jaw are usually not enlarged. But the glands which run along the hinder border of the sterno-mastoid muscle can be felt to be swollen; and sometimes the same applies to those of the axilla and the groins. Dr Dawson Williams states that this enlargement of the glands often precedes the exanthem.

There is lachrymation with some photophobia, and usually slight nasal catarrh, accompanying but not preceding the exanthem. In exceptional cases this is followed by bronchitis. But catarrhal symptoms may be absent, and they are rarely so marked as in even mild cases of measles.

Pyrexia on the second day is sometimes rather high, with quick pulse, restlessness, and occasionally delirium; 100°—103° is the usual height reached, but 105° has been more than once recorded. The temperature subsides with the rash on the fourth day, and the pulse falls with it.

Traces of albumen are often to be found in the urine, and Dr Liveing has in exceptional cases seen it persist and anasarca follow. The tongue is moderately furred, and does not resemble that of scarlatina. Dr Douglas, of Newbury, informs the writer that, in an epidemic in 1883-4, he twice saw "rheumatoid" synovitis of the wrists lasting three or four days.

The whole attack occupies four to six days, about as long as a slight one of measles. There are, however, longer and more severe cases than usual. The following case, which the writer saw with Dr Sidney Turner, is a fair example of the less frequent and graver forms.

A healthy youth of 18 had a rigor on May 10th, 1895, followed by repeated vomiting and high fever with an exanthem. The temperature ran up to 103° that day, and once reached 104°. The rash appeared first on the cheeks and front of the chest, and then spread over the trunk and limbs. The face and the legs were least affected; the wrists and insteps, chest and back the most. There was no coryza, but sore throat quite unlike that of scarlatina. The lymph-glands at the angle of the jaw were not swollen, but those behind the sterno-mastoid were. The lungs were free; the urine febrile. There was no lues, and no drug had been taken. The headache was very slight, and muscular pains absent. There were no children in the house, and no other case occurred; but there was an epidemic of rubeola (Rötheln) in the neighbourhood. Convalescence was rapid and complete after pyrexia of nine or ten days.

Sequelæ appear not to exist. Dr Goodhart affirms that if after an attack of German measles a child remains thin and feeble, or has a discharge from the ears, the disease was either scarlatina or measles.

Diagnosis.—The first cases of an epidemic may often be doubtful; but after seeing a few, the eye recognises the peculiar aspect of the rash. Between slight cases of scarlatina and rubella the distinction is often difficult: the tonsillitis and the desquamation in the former case are probably the most helpful points to look to. The early appearance of the exanthem (usually along with the fever) is the best distinction from measles, and the catarrhal symptoms are absent or slight. The face also is least, not most affected. The swollen cervical and mastoid lymph-glands are very characteristic. They do not depend on severe angina, as in scarlatina, and appear at the very beginning of the pyrexia.

It is often difficult to distinguish rubeola from "ordinary rose-rash," whether set up by gastric disturbance or definite poisons, or caused by local irritation. Dr Dukes says that the roseola produced in some persons by handling the hairy larvæ of certain moths (the "woolly bears" which schoolboys collect) is sometimes exactly like rubeola.

There is no doubt that cases of a diffused bright red eruption, which is not contagious, appear in groups during hot weather. The name Roseola, or "rose-rash," is suitable for this affection, which is quite distinct from true Erythema multiforme (to be described under Diseases of the Skin) as well as from Rubella, from Morbilli, and from Scarlatina. It is clear from the absence of catarrh and sore throat, from the severe itching, and from the proneness to relapse, beside many other characters, that this affection, graphically described as Roséole by Trousseau ('Clinique médicale,' tome I, p. 161) and as Erythème scarlatiniforme by Hardy ('Leçons sur les Maladies de la Peau,' 2me partie, p. 35), is not—as the late Dr Tilbury Fox ('Skin Diseases,' p. 93) and other writers have supposed—identical with the rubeola of German authors. Probably the papules described by Vogel and Borsieri as *ardentes et prurientes*, and named Essera Vogelii, were of the same nature, or they may have belonged to true Erythema. Essera is defined by Blanchard ('Lex. Med.,' ed. 1702) as *tubercula parva, ad rubrum vergentia, duruscula, cum insigno pruritu subito universum corpus occupantia*, characters agreeing better perhaps with Erythema papulosum or "prickly heat" than with Roseola, but widely differing from Rubella.

Protection.—Second attacks are rare, probably rarer than in the case of true measles; statements to the opposite effect no doubt refer to epidemic roseola, not to rubeola. It does not protect from measles or from scarlatina. Among sixty-three cases of rubeola seen by Dr Dukes, thirty-nine had previously had measles. Among thirty cases observed by Dr Cheadle, twenty-two had had measles, and ten of these cases had been under his own care. There are several instances of an epidemic of rubeola following one of measles, but there seems no reason to suppose that measles predisposes to rubeola. Measles attacks, as a rule, younger, and rubeola older children.

Prognosis.—Most authors agree that in epidemics isolated cases, much more severe than usual, may occur, and that death may occasionally happen. If, however, we exclude the description by Paterson, these cases must be extremely rare. A favourable prognosis may therefore be given.

No special *treatment* is necessary. The patient need not in many cases be confined to bed after the nature of the complaint is clear, but he should be secluded from others for a fortnight if infection is to be prevented.

SMALLPOX

> "That day that the Friends on both sides met to conclude the Marriage, she fell sick of the Smallpox, which was in many ways a great trial upon him: first her Life was allmost in desperate hazard, and then the Disease (for the present) made her the most deform'd person that could be seene, for a great while after she recover'd; yett he was nothing troubled at it, but married her as soon as she was able to quitt her chamber, when the Priest and all that saw her were affrighted to looke on her. But God recompenc'd his justice and constancy by restoring her as well as before."—*Life of Col. Hutchinson, by his Wife.*

Name and history—Contagion and spread—Incubation—Onset—Early rashes—Specific eruption—Varieties of the Exanthem—Course and symptoms—Discrete, confluent, and modified Smallpox—Complications and sequelæ—Protection—Diagnosis—Prognosis and treatment.

Vaccinia—Inoculation—Introduction of vaccination by Edward Jenner—Course of the eruption—Its protective power—Its drawbacks—Its relation to Variola that of an attenuated virus—Past and recent legislation.

Synonyms.—Variola, or frequently in early authors Variolæ.—*Scotticè* the Pocks.—*Fr.* La petite Vérole.—*Germ.* Blattern, Menschenpocken.—*Ital.* Vajuolo.—Mod. Gr. Εὐλογία, a euphemistic term.—*Arab.* Jadari.

Definition.—A specific contagious fever with a characteristic pustular exanthem, leaving scars.

History.—This terrible and repulsive disease, after the disappearance of the plague and before the introduction of vaccination, was the most fatal of all epidemics. It appears to have been unknown to the ancients, and was first described by the Arabian physician Rhazes about 900 A.D., under the name Jadari, translated into λοιμική, *i. e.* the pestilential eruption, by the Greeks. Smallpox was long regarded as a more severe kind of measles.

It is probable that some of the pestilences of the later Roman Empire and of mediæval Europe were really epidemic Variola, particularly one which broke out during the siege of Mecca in 570 A.D. As the knowledge derived from the Arabian writers by Greek and Jewish physicians became diffused, the more severe and dangerous disorder was discriminated from measles; but even when, in the sixteenth century, smallpox was generally recognised, the two were often confounded together, and so late as 1660 Pepys speaks of measles as "the same disease" as the smallpox ('Diary,' January 11th, 1660-6).*

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* The English term Smallpox refers to the pockets or little pocks, pokes, or bags in

Variola appeared in America soon after its discovery, and was terribly destructive among the natives. It is almost as much so in Central Africa now. In China, and also in India, long before the Christian era, Variola was well known, and is described in books still extant.

Origin.—The infectious character of smallpox has never been doubted; its virus is as active as that of typhus, and almost as persistent as that of scarlatina. It resides in the serum and pus of the exanthem, and can be preserved in the powdered crusts of the pustules. It was long ago proved to be particulate by Chauveau and Burdon Sanderson.

Streptococci and staphylococci (three species of the latter were identified by Copeman) have been described as present in the pustules of smallpox, but they are probably those of common suppuration, and certainly have no claim to the specific epithet of S. variolæ. In the epithelium surrounding variolous or vaccine pustules, and in that of the cornea of rabbits inoculated with vaccine lymph, organisms which are believed to be parasitic sporozoa, coccidia, or amœbæ have been discovered by Loeff and L. Pfeiffer ('Centralbl. f. Bakt. u. Parasitenkunde,' 1887, and 'Die Protozoen als Krankheitserreger,' Jena, 1890), by Guarnieri, by Ruffer and Plimmer, Jackson Clarke, von Sichner in Buchner's laboratory, and by Ernst Pfeiffer. It is doubtful whether these are independent organisms ('Münchener med. Woch.,' August, 1895). Klein discovered a short bacillus in the lymph of variola and vaccine ('Report to the Local Government Board,' 1892-3), and this has been independently confirmed by Dr Copeman, who regards it as possibly the specific microbe of the disease (see his paper in the 'Journ. of Path. and Bact.,' vol. ii, p. 407, 1894, and a comprehensive summary of the pathology of Vaccinia and Variola in the second volume of Allbutt's 'System of Medicine,' pp. 636—656).

The disease may be spread without actual contact with a patient either by dried-up secretion, or by fomites which have been contaminated with the pus,* or probably by the air within a certain limited distance. The blood does not appear to be contagious, nor the excretions.

There are no decided predisposing causes of smallpox. It attacks persons of every age and race, and is equally destructive in every climate. It has, however, been remarked that children and pregnant and puerperal women are particularly liable to take it severely. In England cases are most numerous in the spring and least so in the autumn.

Variola can be transmitted to many of the lower animals by inoculation with the pus, but is a much less fatal disease than in man, and does not spread. Many pathologists, however, believe that vaccinia in

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which the matter is contained, and denotes the pustular form of the eruption. It also marks the distinction between this and the still more dreaded Great Pox, *i. e.* Syphilis, although the term Smallpox occurs in a MS. of the thirteenth century, 'De Variolis et Morbillis,' as I am informed by my learned friend Dr Norman Moore, who has seen it at Pembroke College, Oxford. The low Latin word *variola* is a diminutive formed from *varus* (with a short *a*, although this did not prevent a Ciceronian pun between it and *varius*), a pustule or pimple, used as a translation of the Greek term ἴονθος. The word variola first occurs in this sense in the sixth century. In older medical literature the plural form *variolæ* is used, referring to the individual little *vari* or "pushes" of the eruption. So in English, pocks, measles, shingles, hives, gripes, some of which forms must be explained as referring to pains or attacks or fits of the disease. All bear witness to the fact that ancient and vernacular names of diseases respect not the condition but the symptoms.

* The *fomes* may be a person who conveys dried-up pus to another, or a corpse with crusts of variolous pustules upon it.

cows and one kind of the affection known as "grease" in horses is modified variola.

The incubation of smallpox is, as a rule, twelve days. On the thirteenth day from that on which the contagion entered the patient's body he is seized with symptoms of fever. The interval may sometimes be a little longer; Bristowe extends it in exceptional cases to sixteen days; but Marson never found the eruption fail to appear in fourteen days after infection. He admits, however, that the incubation is sometimes shortened, and Zülzer believes that in the hæmorrhagic form of variola it is constantly shorter than usual. When variola was intentionally transmitted by inoculation the time of incubation was only nine or ten days.

During this period the patient generally feels well; but occasionally he complains from the first of a vague malaise, with gastric disturbance, headache, and giddiness; and towards the end there may be a little pharyngeal catarrh, with reddening of the uvula and tonsils.

Prodromal stage.—This usually sets in with a severe rigor or with a succession of slight chills. The temperature at once begins to rise, and may reach 102° to 104° within twenty-four hours, and 105° or even 107° by the second day (*fièvre d'invasion*). The strength fails as the fever increases, and the patient totters if he attempts to stand. When carried to the hospital in cold weather he has cold limbs, a pale shrunken countenance, and a small pulse; so that he is hardly to be recognised a few hours later, when he has become warm in bed and when his face is red and swollen. The pulse is much accelerated, varying from 100 to 120 in men, while in women it may reach 130 or 140, and in children 160. The breathing is quick, short, and laboured. The skin is generally dry, but sometimes moist or sweating. Some experienced observers, among whom was the late Mr Stocker, believe that it already emits a peculiar odour; and it was said that Dr Guy Babington could recognise by the smell any cases of variola in the taking-in room. The breath is fœtid, and the tongue is very foul. The patient complains of anorexia and thirst, of pain across the forehead, and sometimes of intense general headache. Children are not infrequently attacked by eclampsia; or they may become delirious or comatose. They often have diarrhœa; but in adults constipation is more usual. In the more severe cases enlargement of the spleen may be detected during the first three days of the fever.

So far there is little to distinguish the early period of variola from that of other fevers. A more characteristic symptom is *vomiting*, attended with violent retching and pain at the epigastrium. This is sometimes so severe and persistent that a case has been sent up to hospital as one of ileus. Another characteristic symptom is *pain in the sacrum and loins*. This, in a severe form, is not quite constant; but it is seldom absent in unmodified cases. Women are apt to suppose that it indicates the approach of menstruation; or, if they are pregnant, that labour is about to set in. As a matter of fact, the catamenia do frequently appear during this stage; sometimes prematurely, but often by a mere coincidence in their regular course. In men the pain is commonly called lumbago.

Another set of symptoms, which are of importance because they may cause the disease to be mistaken for measles, are those of common *catarrh*—sneezing, epistaxis, intolerance of light, lachrymation, sore throat, and hoarseness. The tonsils and the palate may also be reddened.

Lastly, the prodromal stage of smallpox is occasionally attended with one or other of two distinct forms of cutaneous rash, which differ in their characters and still more in their significance.

Early roseola.—One of them may be described as *roseola variolosa*, the name employed long ago by Rayer. Morton, writing in 1718, records it in one of his cases (No. xxxviii), and calls it an early rash resembling scarlatina. Until recently most observers regarded the presence of this remarkable rash as a proof that measles or some other exanthem was present in addition to variola. Reinhold, in 1840, seems to have first taught that it is a preliminary symptom of smallpox alone. In this country Dr Wilks drew attention to it in the 'Guy's Hospital Reports' for 1857 and for 1861. Watson also mentioned it in 1857. But Simon, of Hamburg, is the writer who has most fully described it, and has recorded the largest number of cases.

This prodromal roseola is more common in some epidemics than in others. As a rule it comes out on the second or the third day; but sometimes it immediately follows the initial rigor, and occasionally is the earliest sign that anything is amiss with the patient. It may last a day or two, but sometimes is over in a few hours.

There are several varieties of early roseola. One is in blotches, and sometimes closely resembles measles; it may come out all over the body, perhaps appearing first upon the face. Another is an evenly diffused red blush, resembling scarlet fever, but (according to Simon) darker and of a more purplish tint. This has often a characteristic distribution, which was pointed out by Hebra: it is limited to a triangle having for the base a horizontal line drawn across the abdomen about the level of the umbilicus between the two iliac spines, while the apex is formed by the contact of the two thighs. It may also be visible in the axillæ, and on the adjacent parts of the upper arms and of the chest. On the limbs it affects the extensor surfaces of the elbows and knees, the backs of the hands and fingers, and each foot in the course of the tendon of the extensor hallucis. Its distribution is so characteristic that it once enabled Dr Fagge to diagnose a case in which there was no other reason to suspect smallpox. Simon has recorded an instance of *roseola variolosa* in which the disease aborted, although its nature was proved by the patient's sister suffering from an attack of variola at the same time. Sometimes the seat is irregular. We have in our museum at Guy's Hospital models of a case in which it affected only the flexor surfaces of the forearms (Nos. 8 and 9); and in women it may be limited to the skin about the nipples. A third variety resembles urticaria, while in many cases there are petechiæ which leave brownish stains after the rash has faded. Hebra and Trousseau independently remarked that the parts affected by *roseola variolosa* afterwards remain free from the proper smallpox rash; and subsequent observers have confirmed this statement.

This early, prodromal, or initial roseola is not a common symptom of variola. In an epidemic at Bradford in 1893 Dr W. A. Evans observed 42 cases among 1055 patients, and this was a larger proportion than usual. Among these forty-two cases, seven were like scarlatina, four exactly like measles, and the rest were local, not general, most of them affecting the lower part of the abdomen and the thighs (see also Dr Sharkey's paper in 'St. Thomas's Hospital Reports' for 1880).

Early purpura.—Very different is another form of initial eruption, which marks the most fatal variety of smallpox, the *Variolæ nigræ* of older writers, also known as *Purpura variolosa*, and *Malignant* or *Hæmorrhagic smallpox*.

After the usual early symptoms have been present for eighteen to thirty-six hours there appears on the trunk and the limbs a diffused scarlet redness, which at first can be made to disappear by pressure with the finger. This soon becomes the seat of extensive effusions of blood, in spots and patches of all sizes and shapes. Large black rings form round the eyes, and the conjunctivæ are ecchymosed: this, according to Marson, may be seen very early in the case, and is always a most dangerous symptom. The breath has a horrible fœtor, from a sloughing inflammation of the fauces. Hæmorrhage may occur from any or all of the mucous surfaces. Bloody liquids are coughed up or vomited, or discharged from the uterus or the bladder; or there may be epistaxis, or a sanguineous flow from the eyes or the ears. The urine is extremely fœtid, and it is albuminous even when it contains no blood. The patient generally complains of severe pain at the præcordia, and suffers severely from vomiting. Yet, according to Curschmann, the temperature is seldom over 104° until just before the fatal termination. Both he and Marson remark that consciousness is retained almost to the last; "few patients are so fortunate as to become quickly delirious or comatose." Anæsthesia or hyperæsthesia of certain parts of the surface, and paralytic affections of the limbs, are said to have been observed by Zülzer. Before death the whole body becomes black or of a leaden grey colour. According to Marson, the smallpox eruption is nearly always confluent in cases of this kind; but they often end too rapidly for this stage to be reached. Dr Fagge, however, recorded two or three instances in which, although the disease was prolonged for several days, not even papules could be discovered. The first of these cases occurred in the clinical ward of Guy's Hospital; the patient lived four or five days, and as no sign of a proper smallpox eruption could be detected, it was supposed not to be an example of *purpura variolosa;* but a few days afterwards the clinical clerk who watched the patient fell ill with a mild attack of smallpox.

Purpura preceding the exanthem of smallpox is particularly apt to attack drunkards, women recently confined, and those who are pregnant.

The early stage in slight and abortive cases.—Occasionally the symptoms during the prodromal stage are very slightly marked, or even absent; the proper exanthem apparently succeeds a few hours' malaise, or is itself the first sign that the patient is ill. In such cases the disease is always very mild. They are not common even in vaccinated persons, and are exceedingly rare in unprotected patients.

Unlike a slight prodromal stage, a severe one is no guide to the future course of the case. In women and in children alarming symptoms are often present during the first day or two, and yet the subsequent course of the disease is mild. The most extreme instance of this is a form of smallpox which was long ago described by Sydenham as *Febris variolosa*, and by de Haen as *Variolæ sine variolis*. After a well-marked initial stage the disease aborts, and the patient is well in three or four days, or in six at the latest.

The exanthem.—As a rule, the *third* day is that on which the characteristic eruption of variola appears; but in children it is often the second day. Exceptionally nothing is to be seen until the fourth day, and this delay is of favourable prognosis.*

The specific and constant exanthem of smallpox is first papular, then vesicular, and finally pustular. The papule is round and smooth; at first it may be scarcely redder than the rest of the skin; and as it is hard, like a small shot, it can often be better felt than seen. After twenty-four hours it is always decidedly reddened and has increased in size. It depends upon a definite change in the superficial and middle cells of the rete mucosum, which are swollen and opaque from the first. Liquid exudation quickly takes place, so that by the end of two days the horny layer of the epidermis is raised to form a minute conical vesicle. By the fourth or fifth day of the eruption (seventh or eighth of the disease) the vesicle is as large as a split pea, hemispherical in form, and opaline in appearance. As a rule, the *pock*, or pocket, has a central depression—the umbilicus. The origin of this has been much discussed; it generally seems to depend upon the fact that the original papule was developed round the mouth of a hair-sac, or else round that of a sweat-gland, either of which structures may afterwards form a *retinaculum*, tying down the roof of the vesicle in the middle. Rindfleisch gives a drawing of a preparation in which a sudoriparous duct is plainly seen in this relation to the pock, and he says that he has many such specimens in his possession. However, it would seem that this explanation is not always applicable, since the pock does not bear a constant relation to any of the canals which traverse the cuticle. In all probability a similar function may be discharged by one of the numerous bands which cross the upper part of every vesicle in a direction more or less vertical, dividing it into a number of separate chambers. This *loculated* character of the pock attracted notice long before its structure was understood; it explains why only a small part of the fluid is evacuated when a needle is introduced into the roof at a single spot. As Auspitz and Basch showed, the septa are formed out of the original cells of the rete mucosum, small bundles of which cohere together, and become stretched out into filaments and bands as the exudation accumulates around them. In this fluid leucocytes are present in small numbers from the first; they go on increasing, and thus transparent serum gradually passes into opaque pus; the change is complete in about six or seven days from the first appearance of the papule,—that is by the ninth or tenth day of the disease. The pustule, when fully formed, is often hemispherical, the umbilicus having disappeared in consequence of the rupture of the retinaculum which formed it.

While the roof and cavities of the pock are thus being developed, changes are going on in its floor, which consists of the papillary layer of the cutis, with at least the lowest cylindrical stratum of the rete Malpighii. Whether a swollen state of the papillæ has any share in the formation of the original papule appears to be doubtful, but there is no doubt that its redness is due to their hyperæmia; and (according to Bärensprung) this extends down through the whole thickness of the skin. The exudation which fills the vesicle, and afterwards the pustule, is derived from these vascular tissues. But Curschmann says that, so far from the papillæ being always enlarged at this stage, they are often rather flattened by the pressure to which they are subjected. In some cases, however, they become the seat of an infiltration of leucocytes, which is so intense that it obliterates their blood-vessels, and, indeed, destroys their structure completely, converting them into a white or ash-grey substance. German histologists, in accordance with their usual terminology, describe this form of pock as *diphtheritic* (*vide supra*, p. 53). The contrast is the greater because the surrounding skin is of a bright red colour for a considerable distance, making what is termed the *halo* or *areola*. The infiltration of leucocytes into the floor of the diphtheritic pock may extend to a varying depth in the derma, or even through it into the looser tissue beneath. Rindfleisch gives a drawing from an injected preparation, in which the affected area had failed to receive any of the colouring matter, showing that its vessels were no longer permeable.

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* Among thirty-eight cases of Variola recorded by Dr Richard Morton in the city of London two hundred years ago (1670–94) the eruption appeared on the third day in twenty-six, on the fourth in seven, and on the second in five.

Many of the pustules of smallpox undergo destruction almost as soon as they are fully formed. They break, or are ruptured, giving exit to a honey-like matter, which collects in drops upon the face or other exposed parts of the patient's skin, and saturates his shirt, his pillow, and his sheets. Others, however, remain uninjured, and ultimately dry up without discharging their contents. First a yellow-brown spot appears in the roof of the pock; this sinks in, so as somewhat to resemble the earlier umbilicus; gradually it enlarges and extends to the periphery. The process of desiccation, both in ruptured pustules and in those which are entire, begins about eight or nine days after their first appearance in a papular form. The crusts vary in colour from yellow to brown, or even black, as more or less blood is mixed with the pus of which they are formed. It is now that the extent to which the papillary layer of the true skin has been involved in the inflammation becomes important. If it has escaped, the crusts fall off in four or five days (about the fifteenth or sixteenth day of the disease), leaving purple-red stains, but little or no permanent cicatrisation. But if pus-cells have infiltrated the papillæ, or the whole thickness of the skin, these structures, to whatever depth they may have been affected, slough away and become detached as shreds, adhering to the under surface of the crusts. Their separation takes longer, and may not begin until the eighteenth or twentieth day. Moreover, when it occurs, granulating surfaces are exposed which may take a considerable time to heal. The resulting cicatrices are for a time of a brown colour, but ultimately they become whiter than the skin around them. They may either be so faint as to be scarcely perceptible, or more or less deep and pitted; and it may be only months after recovery that they become depressed to the full extent. (See models in the Guy's Hospital Museum, Nos. 12—18.)

Distribution.—The exanthem of variola appears first upon the face and scalp, especially on the forehead and about the eyes, the nose, and the upper lip. A few hours afterwards it is to be seen upon the trunk and the arms. Marson mentions the wrists as among the earliest parts affected, and says that on the legs and the feet it is generally two days later than elsewhere. He also lays stress on the fact that the papules come out "in threes and fives, forming crescents," or even a complete circle if two crescents happen to coalesce. During the first day or two fresh spots keep appearing, even on those parts which are already more or less thickly covered; but by the end of this time the eruption is complete, for even if a few more should afterwards show themselves they soon abort and die away. A curious circumstance is that smallpox specially affects any parts of the skin which may happen to have been recently irritated: the red patch from a mustard plaster applied during the incubation or shortly before will present many more pocks than the regions adjacent.

Certain *mucous membranes* take part in the eruption of smallpox, but not in quite the same way as the skin. There first appear raised spots, whitish or grey in colour, contrasting with the reddened state of the surface around them; and they quickly pass into excoriations. They are sometimes present in large numbers on the inner side of the lips and of the cheeks; sometimes they affect the tonsils and the palate, which may become greatly swollen and the seat of deep phlegmonous inflammation, ending in abscess. The tongue very seldom presents any traces of vesicles, but is sometimes much swollen, protruding from the mouth, and rendering the patient unable to close his jaws. The air-passages are said to show more or less distinct pustules as low as the bifurcation of the trachea, or even (according to Wagner) down to bronchia of the second or third order; there may also be deep ulcers in the larynx, with perichondritis and necrosis of cartilages, and œdema of the epiglottidean folds. In the alimentary canal nothing resembling a pock can be seen below the upper part of the œsophagus, except perhaps in the rectum close to the anus, or at the entrance of the vagina. Marson speaks of twenty-six cases at the Smallpox Hospital, out of a total of over fifteen thousand, in which the conjunctiva showed a single pustule, generally between the cornea and the internal canthus. It did not affect the sight or lead to any ill result.

Varieties in the eruptive stage.—The severity of variola follows the number of the pustules. When they are so crowded that they run together the disease is said to be *confluent.* This may either be the case over the whole of the body, or only over certain parts of it, or on the face alone, for the eruption is never more abundant than on the face.

If the pustules remain everywhere distinct from one another the smallpox is described as *discrete.* Between the two forms the state of the face decides, and there is seldom any difficulty in calling a case either confluent or discrete.

In discrete, as well as in confluent smallpox, the pustules run through all their stages. But there is a third variety, made up partly of discrete and partly of confluent cases, which is characterised by the premature subsidence or abortion of the eruption. For this, unfortunately, there is no received name. German writers* describe it as "*variolois*," a word etymologically misleading as well as barbarous; since it is not "like variola," but is true, though modified variola, and capable of generating the severer forms of contagion. In this country it is generally termed *modified smallpox,* because it is seldom seen except in persons who have been vaccinated. But variola occasionally passes off in the same manner in a patient who is unprotected by previous variola or vaccinia.†

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* Strictly speaking, I think that this is not quite correct. It is true that German writers give *variola modificata seu mitigata* as a synonym for their "*variolois;*" but in practice they confine the use of the latter term to cases of a certain moderate degree of severity, and apply it to all such cases, whatever may be the course of the individual pustules. Thus Curschmann speaks of *variolois* as sometimes leading to destruction of the papilla and to the formation of deep cicatrices, "so that what distinguishes the case from one of *variola vera* is merely the very much smaller number of pustules." We should call such a case one of very mild discrete *un*modified smallpox. On the other hand, I do not find German writers mentioning under *variolois* the examples of modified confluent smallpox which will presently be described.—C. H. F.

† *Abortive smallpox* would perhaps be a better term.

1. *Discrete smallpox.*—The eruption presents the characters which have been described in the individual pustules. Its amount varies greatly, from a few spots that can almost be counted on the fingers up to many thousands. At the time when they are acquiring their areola there is often a considerable degree of swelling of the surrounding parts, especially where the subcutaneous tissue is loose. Three or four pustules upon the eyelids may cause them to be puffed out like bladders, so that on the ninth and tenth days the patient may be unable to open his eyes. Trousseau cites a case of Van Swieten's in which a single pustule on the prepuce produced phimosis and great difficulty in micturition. He also remarks that the absence of a corresponding degree of tumefaction of the rest of the face renders the swelling of the eyelids more conspicuous in some cases of discrete smallpox than in the confluent variety of the disease.

As regards the general symptoms of discrete smallpox, the first thing to be noticed is that as soon as the eruption has fairly begun to develop itself, the fever which has existed during the initial stage subsides, and the temperature falls within thirty-six hours to normal, or even lower. Its decline is not always quite continuous, being perhaps interrupted by a slight evening exacerbation. At the same time the pain in the back and the sickness disappear, and the patient often feels perfectly well, with as good an appetite as when in health. This lull in the symptoms commonly lasts for three days; at the end of it he is, in all cases except those in which there are but very few pustules, again attacked with shivering and with febrile disturbance, which is known as the *secondary fever*, or the *suppurative fever*, or the *fever of maturation*. The pyrexia is generally remittent in type, the daily variations amounting to one or two degrees Fahr. The height to which the thermometer now rises varies with the extent of the inflammation in the cutaneous tissues, and therefore roughly with the number of the pustules; even in severe cases it is seldom above 102° or 103°. The pulse is quickened, being at from 110 to 120. The patient at the same time complains of headache, and is restless and sleepless. He is not unfrequently delirious, especially during the first night or two.

It must not be supposed that discrete smallpox is always unattended with danger. Trousseau relates the case of a girl, aged twenty-one, who had passed through a remarkably mild attack, but who was one evening suddenly seized with cerebral symptoms and difficulty of breathing, and in an hour she was dead; and he remarks that when this form of the disease does prove fatal, death occurs at an earlier date than in the confluent form —namely, about the eighth or the ninth day. In ordinary cases of discrete smallpox the fever lasts about three weeks, or even less, and the crusts have fallen by the time that normal temperature is regained.

2. *Confluent smallpox.*—The eruption fails to present its typical characters long before actual fusion of the pustules has taken place. Even before any definite papules can be recognised there is often a diffused redness of the face, which in itself could hardly be distinguished from the rash of measles. Watson mentions a case in which the appearances of urticaria, with its characteristic sensations, were at the outset so intermingled with the papules of variola that for twenty-four hours he doubted which of the two diseases was developing itself; and in other instances smallpox has been mistaken for erysipelas. Trousseau remarks that even where there are, in fact, multitudes of papules, the complete absence of intervals between them

may render one almost unable to detect any unevenness of the surface by passing the hand over the patient's forehead or cheek. The papules are always smaller than in the discrete form, and very early a milky fluid collects in their summits, giving rise to minute flat vesicles. The whole of the face has then a whitish-yellow colour, and its appearance is exactly like that of a mask of parchment, to which Morton, in his 'Pyretologia' (1718, Case xxxviii), long ago compared it. By this time, however, there is already considerable swelling, and it goes on increasing up to the ninth day of the disease, until the features are so altered that it is impossible to recognise the patient. The parts about the ears and the sides of the neck become enormously bloated, and the eyelids cannot be opened. This condition is very painful, and the scalp is often so tender that the pressure of the head upon the pillow can hardly be borne. (See models in Guy's Hospital Museum, 19—22.)

Somewhat later, about the eleventh or twelfth day, the hands and the feet become red, swollen, and painful. Trousseau, following certain of the old writers, attached a high prognostic value to this symptom; according to him its absence in confluent variola is almost always followed by death.

The *mucous membranes* generally suffer severely in confluent smallpox. Laryngitis often renders the voice hoarse, and sometimes causes a sudden and fatal fit of suffocation. Trousseau mentions three cases of this kind, in each of which death occurred unexpectedly on the eighth day of an illness that had previously run a normal course.

Another complication, perhaps due to extension of inflammation to the parotid gland, is *salivation*. Trousseau speaks of this as a characteristic feature of confluent smallpox. It begins, he says, about the fourth or fifth day, and goes on increasing until by the ninth or the tenth day one or two quarts may run from the patient's mouth within the twenty-four hours. Even during sleep there is a constant flow of saliva.

Again, *ophthalmia* is of frequent occurrence, which (unlike the harmless discrete pustules of the conjunctivæ) is often attended with sloughing of the cornea, and leads to permanent blindness.

From the very commencement of the eruptive period, the general symptoms of confluent smallpox run a course which differs from that of the discrete form of the disease. Instead of the temperature falling when the papules come out, it remains at 103° or 104°. There is often violent *delirium*, even during the daytime. It may be quite impossible to keep the patient in bed, except by tying him down with a folded sheet across the chest. If free, he must be most carefully watched, or he is very likely to throw himself out of a window or over the stairs. In those who have been intemperate the nervous disturbance often assumes the form of delirium tremens.

Convulsions are a frequent and often a very grave complication.

Retching and vomiting sometimes run on throughout this period of the disease; and *diarrhœa* is obstinate and troublesome. The urine contains albumen in a large number of cases.

When suppuration is established, there is a further rise of temperature, the *secondary fever*. It rises higher than in discrete smallpox, although, from the patient's previous state, this fever of maturation is less conspicuous.

The secondary fever is the most dangerous period of variola. Few patients die before the eleventh day; the most fatal days are the twelfth, the thirteenth, and the fourteenth. Towards the last there is generally coma; sometimes hyperpyrexia is present, as in a case of which Wunderlich gives a chart, and in which, before its termination on the eleventh day, the thermometer registered 109°. In many instances the immediate cause of death is bronchitis, pneumonia, pleurisy, or pericarditis. Even under the most favourable circumstances the fever runs on for at least ten days longer. Not infrequently, during the third week, the patient falls into a typhoid condition, with sordes, a dry brown tongue, muttering delirium, and subsultus; and these symptoms are justly regarded with dread.

When recovery is to take place, whether from the discrete or the confluent form of the disease, the redness and the swelling of the face subside, as the pustules undergo conversion into crusts. The pain from which the patient has been suffering passes off, but only to be followed by the most intolerable itching. His fever also declines more or less rapidly, being now proportionate to the amount of inflammatory action still going on. He regains his appetite, and becomes once more able to sleep. He opens his eyes, and gradually his features begin to reassume their natural outlines. At the time when the crusts are falling off, or a little later, he generally loses almost all his hair, and if the deeper layers of the skin of the scalp have been involved in the morbid processes there is always reason to fear that permanent atrophy of the hair-sacs may result.

The confluent variety of smallpox usually lasts about four weeks before the fever subsides.

3. A minor variety of smallpox, which sometimes occurs in confluent cases, is characterised by the occurrence of hæmorrhage into the pustules. This must not be confounded with the "hæmorrhagic variola," which proves fatal at an early period before the proper exanthem has come out (*supra*, p. 191). By way of distinction Curschmann calls it *variola hæmorrhagica pustulosa*. As a rule, those pustules which are seated upon the lower limbs are the first to show a purple colour. Bleeding presently takes place from the nose, kidneys, intestines, and other mucous surfaces. In women menstruation sets in, or abortion occurs if they are pregnant. The gums become spongy and bleed, as in scorbutus; the fauces show a sloughing "diphtheritic" affection, which is attended with a terrible fœtor. The constitutional symptoms are in most respects severe; but although the pulse is very rapid the temperature during the eruptive stage is seldom above 102°, and it not rarely falls to 95°, or even lower, before the patient's death. Curschmann says that this variety of smallpox occurs chiefly in persons over forty years of age, and in such as were previously out of health. He describes it as being almost always fatal; but remarks that in patients who have, in their delirium, got out of bed and walked about during the early part of the eruptive stage, the pustules on the legs may become filled with blood, without the case being particularly serious. A few petechiæ on any part of the surface are not a dangerous symptom.

4. Another very rare and fatal variety of the smallpox exanthem in which the pustules are confluent in separate patches was described by Marson as *corymbose*. His account of it will be found in Reynolds' 'System of Medicine,' but no one appears to have confirmed his observations except Dr Conolly, who figured a case in the Hardwicke Hospital at Dublin (quoted by Dr Moore).

5. *Modified smallpox.*—The course of the eruption is much less regular

in properly vaccinated persons than in the ordinary disease, whether discrete or confluent. During the initial stage no distinction can be drawn between it and them; for though there is a large proportion of cases in which that stage is but little marked, in some instances it lasts the usual time, and is attended with severe symptoms. When the papules are seen, they may appear on the trunk or limbs before there are any on the face, and with the exanthem the temperature falls, as in unmodified discrete variola—but with greater rapidity, and without any interruption in its downward path, so that in less than twenty-four hours it is normal.

It is thus evident that no definite period can be fixed at which the modified character of a given case of smallpox first becomes apparent. Sometimes the eruption dies away before it has passed out of the papular stage. Sometimes the papules undergo a partial conversion into vesicles, which then rapidly dry up. A special modification of this variety of the affection has long been known as the "horn-pox" (*variola verrucosa*); but writers by no means agree in their descriptions of it, for while Trousseau speaks of the vesicles as drying up and leaving in their places small, hard, corneous projections, which fall by a sort of desquamation between the tenth and the fifteenth days, Curschmann says that after the scabs have become detached, the solid bases of the pocks remain for a considerable time as warty elevations, especially on the face.

Again, in yet other cases of modified smallpox, the vesicles pass on into pustules. The change then appears to take place unusually rapidly, and the pustules themselves are often extremely small, and probably never reach so great a size as that to which they may attain in the unmodified forms of the disease.

Lastly, even confluent smallpox may occasionally run its normal course for ten or twelve days, and then suddenly subside, so that the case must be referred to the form of the disease which has been modified by vaccination.

Complications and results.—The complications of smallpox are numerous, and in unmodified cases are almost constant. Bronchitis is frequent, and often attended with lobular pneumonia; pleurisy, when it occurs, is apt to lead to empyema; and œdema of the larynx from ulceration and necrosis of the arytænoid cartilages has occasionally proved fatal. Salivation is frequent and distressing. The febrile albuminuria rarely leads to subsequent nephritis. Diphtheria is a dangerous complication and not a very rare one.

The *sequelæ*, beside the pitting, are chiefly various forms of local suppuration, like those which follow enteric and scarlet fevers: abscesses in the skin and lymph-glands, furunculi and ecthyma, tympanitis and otorrhœa, local gangrene and phlegmonous erysipelas. In severe cases the cornea may ulcerate, and the eye be lost, but happily these events, formerly common, are now become rare.

Convalescence is usually rapid, and the health and strength are not impaired as after enteric fever.

Infection lasts until the last scabs have been shed.

The *protection* afforded by smallpox is probably the most complete known. Relapses never follow, and although second attacks undoubtedly occur, they are more rare than in the case of typhus, measles, or scarlatina.

Post-mortem anatomy.—The marks of the characteristic exanthem are found on the skin and the mucous membrane of the mouth, pharynx, and œsophagus, of the nasal passages, larynx, and trachea. There is always evidence of bronchitis, and often of broncho-pneumonia. In hæmorrhagic cases mucoid and subserous extravasations are found.

In the pustules staphylococci are present, but no specific microbes; probably there is, as in severe enteric fever and scarlatina, ordinary suppurative infection in addition to that of the specific contagion.

Diagnosis.—Smallpox in its natural form can scarcely be confounded with any other disease. The vomiting and backache of its advent, the high fever, the constant exanthem, with its pustules and secondary fever, are too striking to be overlooked. But it is as modified by vaccination that variola meets us in civilised countries, and then it is sometimes overlooked, or supposed to be present when it is not.

The possibility of its presence should be considered in every case of lumbago with fever, of vomiting with fever, and of fever with a roseolous rash. The vaccination marks on the arms should be looked for, and if absent or imperfect the case should be isolated at once. A severe attack of influenza may simulate smallpox for the first day or two. The "triangular rash" of Hebra is characteristic, but a more general prodromal rash may resemble that of measles (except in not affecting the face so much) or that of scarlatina. When the characteristic papular exanthem follows on the third day the diagnosis of variola is usually plain, when none occurs on the fourth day it is certainly disproved. Hæmorrhagic variola may be easily confounded with purpura, or rather it is purpura, but secondary to a specific fever: and in the absence of local inflammation this can only point to variola, to scarlatina, or to typhus.

Often, however, a case is not seen until the papules have developed into pustules and the history is not available, or, as once happened to the writer, is designedly distorted. The diagnosis of smallpox then rests on the distribution of the pustules and the character of the fever. No one could mistake variola for acne unless totally ignorant of both; but it has been and probably will be mistaken for secondary syphilis and for chicken-pox. Special points of diagnosis will be again considered under Varicella, Typhus, and Syphilis.

Mortality.—Between ten and twenty about 25 per cent. of unprotected cases are fatal. More than half the unvaccinated patients above thirty years old die, and of those above sixty nearly 80 per cent. To children under five smallpox is as fatal as to adults between thirty and forty; about half of those who are attacked die.

Among sixty-seven cases recorded by Morton in Queen Anne's reign (before inoculation or vaccination were known), all but two being under thirty years of age, thirty-eight recovered and twenty-nine died—a mortality of 43 per cent. Of four cases complicated by abortion only two were fatal

Prognosis.—The points upon which this depends have been already indicated. They are, first, the number and quality of *vaccination marks.* The hundred; where three equally good were found, less than 1 per cent.; where three indifferent marks, more than 3 per cent. Two good marks kept the mortality down to 2·3 per cent., two poor ones only to 8 per cent. With one good mark, less than 4 per cent. of the patients died, and with one indifferent mark nearly 12 per cent. At the same hospital the mortality among unvaccinated patients generally was 37 per cent. (Marson).

In Dr Collie's book on Fevers will be found a series of tables (pp. 174-9) showing the effect of vaccination in diminishing the mortality of variola in young and old patients in the epidemics of 1871 and 1881. These confirm the results of Marson.

The second indication for prognosis is the abundance of the *exanthem*. Marson's statistics give the mortality among 2654 of unvaccinated patients as 4 per cent. in cases of discrete variola; 8 per cent. in cases of semiconfluent, including coherent, variola; and 50 per cent. in cases of confluent variola. A purpuric eruption is of serious omen—most so when it precedes the true exanthem; hæmorrhagic pustular smallpox is also dangerous, but not so dangerous as hæmorrhage in the papular or vesicular stage.

The most favourable *age* is from seven or eight to twenty. Above thirty the prognosis is grave, and above fifty almost hopeless.

Intemperance, delirium, convulsions, and want of sleep are indicative of danger.

Pregnancy is a very serious complication; abortion usually takes place, and the mother dies soon after. Cases, however, are recorded in which both the mother and the child have survived.

Treatment.—There is no special means of curing smallpox apart from treatment of the fever and of the local complications. The most rigid isolation is of course necessary for the sake of others, and all who come in contact with the patient should be revaccinated. Free ventilation and the utmost cleanliness are the first essentials. Diarrhœa should be checked. Laudanum and morphia are extremely valuable as hypnotics. Ammonia and senega are needed for bronchitis, and stimulants during the suppurative stage, especially when boils or abscesses appear.

Constant watching is necessary, in order to prevent the patient escaping from the sick-room or doing himself an injury in his delirium. But Dr Collie recommends that a restless patient should be allowed to get out of bed, and even to sit by the fire, if he persists in the attempt.

In order to ease the irritation of the skin and prevent scratching, as well as to soothe the fever, warm or lukewarm baths of about 100° or about 85° Fahr. are recommended, followed by inunction with oil or vaseline. The face should be covered with a thick layer of lead or zinc ointment, and the pustules should be pricked as soon as formed, in order to diminish scarring.

The good effect of ointments, masks, and other means of excluding light has been long recognised as the most efficient treatment to prevent deep suppuration and consequent disfigurement from scars. The patient has been kept in a dark room with the same object. Probably this depends on excluding the violet and ultra-violet actinic rays, the effect of which in producing dermatitis and pigmentation of the skin is familiar to observant Alpine climbers. These effects have been carefully and scientifically investigated by Dr Bowles (" Sunburn on the Alps." 1888 and 1890, " Influence of Light on the Skin." ' Brit. Journ. of Derm.,' vols. vii and ix, p. 258). See also an interesting paper by Dr Finsen of Copenhagen on the "Treatment of Smallpox" by the red rays of light, 'Brit. Med. Journ.,' December 7th, 1895, and again in September 30th, 1899.

Vaccination in unprotected persons exposed to contagion is imperative; and even if performed after the first symptoms of the disease have appeared, it probably has some effect in diminishing its severity.

But the true treatment for smallpox is prophylactic, not curative, and will next be considered.

VACCINIA.—*Syn.* Cow-pox, Attenuated Variola.—The terrible mortality from unmodified smallpox, and the scarcely less terrible effects it left behind —the scarred and hideous features, the sight impaired or lost, permanent deafness, and other disastrous results—can only be appreciated by those familiar with the general as well as the medical literature of the last century. Treatment of the disease had been improved by Sydenham, but its ravages were but little checked until the introduction of the practice of *Inoculation.*

This has been carried on in China and India from time immemorial, but it is not known how it was introduced into Turkey. An account of inoculation as a prophylactic for ordinary smallpox was first published in the 'Philosophical Transactions' for 1714, but it became popular seven years later by means of the letters and example of Lady Mary Wortley Montagu, the wife of the English ambassador at Constantinople. By inoculation with the lymph from a variolous vesicle the disease is reproduced with a shorter incubation period, with far less severe symptoms, and with a far smaller mortality than when contracted in the ordinary way.

Variola, however, thus produced was still variola, and as contagious as ever, so that the effect of general inoculation was to make cases of smallpox more numerous, though milder. Thus, although the mortality was reckoned at only three in a thousand, the total deaths from smallpox were increased. The Smallpox and Inoculation Hospital was founded in 1746, and inoculation was practised there down to 1822, but after 1840 it was prohibited by Act of Parliament. The operation was performed in China by putting variolous crusts into the nostrils, but in Europe in exactly the same way as vaccination, except that the lymph was variolous instead of vaccine.[*]

The practice of *vaccination*—the greatest achievement of medicine—was the fruit of the scientific temper and indomitable perseverance of the illustrious Edward Jenner. He observed that the milkmaids in the great dairy farms of Gloucestershire were subject to a particular eruption of the fingers, which was derived from similar pustules on the udder of cows. He ascertained the accuracy of a current belief that those who were so affected did not suffer from smallpox.[†] After a long investigation he was so satisfied of the fact that in 1798 he inoculated a boy with the matter from a cow's udder (*i. e.* with the cow-pox instead of with smallpox), and finding that this inoculation was as good a protection from the dreaded variola as the other, he made known his discovery.[‡]

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[*] See an account of inoculation and vaccination in China at the present day, by Dr Cantley ('Brit. Med. Journ.,' Oct. 5th, 1889, p. 761).

[†] This had been noticed before Jenner by Benjamin Jesty, a Dorset farmer, who actually vaccinated himself and his children in 1774.

[‡] 'Inquiry into the Causes and Effects of the Variolæ Vaccinæ, a disease discovered in some of the western counties of England, particularly Gloucestershire, and known by the [illegible]

The practice of inoculation with this "vaccine lymph" rapidly spread. It was adopted by the best physicians abroad as well as in England, and the process of "vaccination," thus begun, was at last made compulsory in all civilised countries. Vaccination does not absolutely protect from smallpox: inoculation did not, and an attack of smallpox by ordinary contagion does not absolutely protect. But variola, like other exanthems, protects in the vast majority of cases against a second attack; inoculated variola is a mild and comparatively safe form of the disease, and likewise protects from further attacks; vaccination is a still milder and perfectly safe form of the disease, and likewise protects from future smallpox.

The statistics given on page 200 show the truth of this assertion. It is also proved by the following example, quoted by Dr Eustace Smith. Dr Gayton had 1574 children under observation in the Smallpox Hospital at Homerton (1871-8). Of these 211 showed good vaccination marks, and only one of them died (0·5 per cent.); 396 showed incomplete vaccination, and 39 of them died (10 per cent.); 179 had been "vaccinated," but showed no scar, and 46 of them died (25 per cent.); while of 788, who unhappily had never been vaccinated at all, no less than 385 died (48·8 per cent.).

Similar evidence on a large scale is afforded by the compulsory vaccination carried out in the great Continental armies, by the results in our ow army, in the military school at Chelsea, and by observations in Sweden, i Germany, in France, in the United States, and in India.

It is certain that vaccinated persons are to a small extent liable to smallpox, especially if the scars are few and slight, or if the time since vaccination has been very long. Efficient vaccination is necessary to obtain th full benefit of Jenner's discovery. A second vaccination is desirable abou the time of puberty, and repeated vaccination is prudently submitted to by those who are going into contact with the disease, as doctors and nurse taking duty at a smallpox hospital or during an epidemic.

With delicate persons the local inflammation attending vaccination ma be severe, and cause some febrile action and several days' discomfort. Wit feeble infants the effects may be more serious. And it is now certain tha vaccination from a syphilitic child may introduce the virus of syphilis a well as that of vaccinia, especially if blood as well as lymph be inoculate This is, however, an occurrence so excessively rare that, while every pr caution is rightly taken against it by choosing healthy infants from whor to take vaccine lymph, the practical risk is infinitesimal.

A much more common but less certainly ascertained drawback to vacc nation is that the slight febrile disturbance which attends it may call fort local or general disorders, especially eczema and catarrhal inflammation Swelling of the axillary lymph-glands of the vaccinated arm and eryth matous œdema of the arm itself are frequent, and sometimes, especially i adults, troublesome. Erysipelas or pyæmia is a possible result, but nc more frequent than after any other slight puncture.

It has been suggested that these drawbacks, slight as they are, might b avoided by vaccination direct from the udder of a heifer affected wit vaccinia instead of from a vaccinated child; and this plan has been exte sively carried out. But it has its disadvantages. The operation is le certain of success, the febrile disturbance is usually more marked, and th maturation of the pustule and falling of the scab is considerably delaye Hence "arm-to-arm" vaccination from a healthy child to a healthy chil is probably the best method, although it is well, in order to meet objections, that calf lymph should also be available.

It was not until 1853 that vaccination was made compulsory by Act of Parliament. Subsequent Acts were passed in 1867 and 1891. Too many cases of inefficient vaccination still occur; and the Act has unhappily been sometimes allowed to become almost a dead letter, as in Leicester and Gloucester. In the latter city the result was a terrible epidemic of unmodified smallpox. But even this did not prevent unscrupulous agitation against the enforcement of vaccination, and in 1898 the Government was obliged, against its judgment, to allow "conscientious" objections to the practice. At the same time certain changes in the regulations were made, particularly transferring the place of vaccination from a station to the parents' house, and up to the present time (October, 1899) it appears that the numbers vaccinated have increased instead of diminished—an unexpected but happy result so far.

Course of vaccinia.—When vaccine lymph has been introduced into the lymphatic spaces of the skin, an incubation period of two days follows. On the third a papule appears, which in two days more becomes a vesicle. Towards the end of the first week a large umbilicated vesicle has formed, and attains its full development on the eighth day, when a congested halo surrounds it. The lymph then becomes turbid, and gradually dries up, the areola fades again, and by the end of the second week a scab has formed. This falls off about the end of the third week, and leaves a pitted depressed scar, at first darker but finally whiter than the surrounding skin. Meanwhile slight pyrexia has begun on the fourth or fifth day, increases at the beginning of the second week, and falls with the maturation of the pustule and the disappearance of the surrounding inflammation.

This process is sometimes slower, as when the vaccinated child is already in the incubation stage of another febrile disorder like measles; and it is sometimes quicker, or otherwise disturbed, as when the child is already affected by some inflammatory affection like eczema.

The course of a second vaccination is almost always rapid, and the constitutional disturbance usually greater, owing perhaps to the patient's age.

Occasionally cases are reported in which, beside the ordinary local effect of vaccination, a secondary generalised eruption takes place after a few days, with some fever. These rare cases are of small practical but great scientific interest.

Nature of vaccinia and pathology of vaccination.—Jenner himself believed that cow-pox was modified variola. This was long disputed, even after the experiments made by Mr Ceeley (1839) and by Mr Badcock (1840), but more recent observations and experiments go far to establish the fact.[*] It is now generally believed that variolous lymph inoculated on a cow's udder produces vaccinia; this inoculated in a human arm reproduces itself and protects from subsequent vaccination, as well as from subsequent smallpox.

Cows only are subject to vaccinia, because the contagion is conveyed to them from human variola in the process of milking. The disease is only derived by cattle from human beings, and dies out when not thus reinforced. It may also affect horses and probably sheep, though the *variola ovina* is in certain respects different. The vaccinia of cows is probably the same as one of the diseases known as the "grease" in horses.

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[*] The careful experiments carried out at Lyons by Chauveau threw fresh doubt on the question. He found that inoculation of vaccine lymph produces vaccinia in the cow, and that variolous inoculation produces variola. But the latter disease could not be thus propagated, and when both inoculations were made together the variola died out, while the [illegible]

If, as the writer believes, vaccinia is true variola modified by "cultivation," it furnishes the first and hitherto the most successful example of the method of protection from specific diseases by previous inoculation with an attenuated virus.

The cases of generalised vaccinia mentioned above (p. 203) strongly confirm this view.

For further details on the subject of vaccination, so interesting as an example of scientific experiment and reasoning applied to medicine, so important in its bearing on general pathology, and so deeply affecting the lives and happiness of the human race, the reader is referred to Dr Seaton's article in Reynolds' 'System,' and his 'Handbook of Vaccination' (1868), to Sir John Simon's Reports to the Privy Council, and to the late Mr Ernest Hart's excellent popular pamphlet.

The most complete historical account of vaccination is contained in Dr Crookshank's two volumes, published in 1889. Dr Acland has since written for the second volume of Allbutt's 'System of Medicine' (1897) a painstaking and particular review of the normal variations of vaccinia and its occasional complications, and Dr Copeman a full account of its pathology.

Perhaps the most weighty confirmation of value of vaccination is that given by the late Lord Chancellor Herschell, the chairman of the Royal Commission on Vaccination, which sat from 1889 to 1896, who, after hearing all that could be objected by the two or three physicians who dissent from the rest of the profession on this subject, made a statement which is printed at length in the 'British Medical Journal' for May 15th, 1897, p. 1247.

The broad facts on which we may safely rest the case for compulsory vaccination in infancy and, if possible, revaccination at or soon after puberty may be stated as follows:

1. The practice was introduced by a man of science and practical experience after adequate investigation. It was very properly criticised, as well as ridiculed, and within a few years was accepted by the whole of the profession in this country.
2. It was received with enthusiasm throughout Europe and the United States, and has been introduced wherever variola is known.
3. It has stood the experience of 100 years in all climates and races.
4. It is accepted by the most competent judges in all civilised countries.
5. It is most successful where most rigidly enforced, as in the German army, and less so where individual liberty is widest, as in England. The epidemic in the town of Gloucester in 1896 was a striking lesson of the mischief of allowing a preventable disease to be freed from the only known means of prevention.
6. Its scientific justification is that similar inoculations with attenuated virus have since proved effectual in the case of many other diseases.

VARICELLA

> " Each little pimple had a tear in it
> To wail the fault its rising did commit."
> DRYDEN.

History of the recognition of Chicken-pox—Its distinction from Smallpox—Incubation and onset—Characters of the eruption—Symptoms and course—Sequelæ—A disease of children—Diagnosis—Prognosis and treatment.

Synonyms.—Variola crystallina, spuria, volatica, Variolæ pusillæ (Heberden), the Chicken-pox.—*Fr.* Varicelle.—*Germ.* Windpocken, Wasserpocken. The word *varicella* is evidently intended as a diminutive of variola. The vernacular name is probably a corruption of *chickpease* (French *chiche*, Latin *cicer*), in allusion to the size of the vesicles.

Definition.—A specific infective disease with a characteristic exanthem and course.

At the end of the seventeenth century, soon after the final separation of measles from smallpox, English writers mention a variety of the latter disease popularly called " chicken-pox." The same affection seems to have been described in the sixteenth century by Vidus Vidius, and by Ingrassias, under the designation of " Crystalli." Vogel (1764) is said to have introduced the name Varicella. In 1730 Fuller asserted that chicken-pox and smallpox were really distinct diseases; but Heberden, in the first volume of the 'Medical Transactions of the College of Physicians' (1767), fully and conclusively described the differences between them. He also stated the chief reason which makes the recognition of chicken-pox important, namely, that those who had it might otherwise be deceived into a false security, and neglect keeping out of the way of the smallpox.

It was unfortunate that some of the earliest advocates of Jenner's vaccination, being anxious to show that the protection afforded by it was absolute, referred every suspicious-looking eruption in persons who had been vaccinated to chicken-pox. This, in its turn, led to a reaction, and its claim to be regarded as an independent disease has been disputed by several physicians, including even Hebra. Unfortunately, too, the great German dermatologist threw the subject into further confusion by employing the term varicella in an entirely new sense, namely, for all very mild cases of smallpox. It is certain that the varicella of Heberden is totally distinct from the mildest and most modified smallpox.

The proofs are (1) that it occurs in those who have been vaccinated, or who have had variola, just as readily and with the same characters as in those who are unprotected; and (2) that a person who has passed through it remains as susceptible as before to the vaccine virus or to that of smallpox. In the 'Lancet' for 1877 a case is recorded of an unvaccinated child who was admitted into St. Thomas's Hospital for chicken-pox, but who was placed on the floor containing the smallpox wards, because the diagnosis was at first uncertain. Two days afterwards vaccination was successfully performed, and eight days later still the child fell ill with modified variola.

The characters of the eruption of varicella, and the date at which it develops, are in themselves ample proof of its distinctness.

The lymph from the mildest eruption of variola is capable of conveying that disease by inoculation, but almost all of those who have tried to inoculate varicella in the same way from its vesicles have failed. Early in the present century Bryce performed this experiment upon thirteen children, who had never had either smallpox or cow-pox, without any result. It is true that, in a long series of cases collected by Hesse in 1829, there was a small minority in which inoculation appeared to succeed; but according to Thomas there are reasons for thinking that some error crept into these observations.

The nature of the contagium of varicella is not yet ascertained. It is certainly particulate. The disease may be conveyed by the vesicles and their dried-up lymph directly or through fomites. It is as contagious as smallpox, and protects as well from a future attack.

Incubation.—This seems to be of variable duration, usually a fortnight. It has probably been calculated upon the precarious basis of the interval between the dates at which different children of the same family have been successively attacked. Heberden stated it at eight or nine; Dr George Gregory at from four to six days. Bristowe says that in some cases it lasts exactly a week, but perhaps more commonly a fortnight. According to Thomas, it may be from thirteen to seventeen days; according to Gee, about a fortnight; according to Liveing, variable, about thirteen days with eruption on the fourteenth. Eustace Smith gives seven to fourteen days; Trousseau, from fifteen to seventeen; Dukes, fourteen to nineteen; Eichhorst, thirteen to sixteen; and MacCombie, thirteen to seventeen.

Onset.—This is usually simultaneous with the appearance of the eruption, so that there is no stage of fever before the exanthem appears, as in enterica, scarlatina, measles, and smallpox.

Thomas took the temperatures of children who afterwards developed varicella (no doubt where the disease was already in the family), and always found them normal or not raised above half a degree (Fahr.), which in childhood is of common occurrence in health.

Even where there are slight prodromal symptoms, as headache or cough, with loss of appetite and feverishness, they only last a few hours before the characteristic rash appears, so that the period of incubation must, as in the case of Rubeola (*sc.* notha), be reckoned up to that of the eruption.

In some instances, however, Thomas found that the child was feverish for a few hours before the rash appeared, and sometimes the fever lasted two or three days, and was accompanied with delirium. In a case which the writer saw some years ago convulsions preceded the exanthem.

The exanthem.—This begins as a series of small, slightly pointed red spots, which Trousseau compares with the rose-rash of enteric fever. Dr Gee says that they disappear when the skin of the part is stretched, this being a proof that there is no exudation into the tissue of the cutis, but only hyperæmia. In a few hours they pass into as many transparent tense vesicles, round or oval in form, and about as large as split peas. These sometimes have a red base; sometimes they are seated upon a perfectly colourless surface, so that the patient looks exactly as if he had been sprinkled with drops of clear water. They differ from the vesicles of smallpox in their superficial position: they have no thickened floor, they do not consist of a series of separate chambers, and when they are pricked they almost completely collapse. Most of the vesicles of varicella are rounded, particularly on the face and limbs; but on the back they are often oval, with the long axis cross-wise (Stowers). Most of them are without the central depression of smallpox; but in almost every case some may be found umbilicated, and occasionally most are so. They appear in crops, which come out in succession during the first, second, and third nights of the disorder. At length they acquire a yellowish appearance, and the fluid in them turns slightly opalescent, or may become puriform. After from twelve to twenty-four hours they begin to dry up and become flaccid; they are often ruptured, either by the nails of the patient (for there is often considerable itching) or in some other way; or they fall in first at their centres, so as to acquire a spurious umbilicus. Ultimately they form thin brownish-yellow scabs, which in a few days crumble away and leave reddish pigmented spots.

The eruption of varicella generally appears first on the upper part of the *back* or on the chest, rarely on the face. Formerly it was said to spare the face altogether; and although this is incorrect, it never comes out more thickly there than elsewhere, as is the case with variola. On the scalp it is almost always present. Afterwards it spreads to the limbs, but is more scanty there than on the trunk, and seldom reaches the hands or feet.

The vesicles come out, not in a single crop, but in a succession of crops, which may be prolonged over three or four days or even a whole week. Thus one sees papules and vesicles and crusts side by side at the same time; in this it is very different from smallpox. The total number of vesicles is sometimes not more than from ten to thirty, but according to Thomas it is generally two hundred or two hundred and fifty; and three times as many are said to have been observed. Only a few come out on the first day—perhaps a score. Then a hundred or more fresh ones are seen the following morning. The scabs which form as the vesicles dry up, fall off if left to themselves in a week or ten days, and leave reddish marks which gradually disappear; but sometimes a few small white cicatrices remain behind, and these may occasionally be depressed, and even pitted.

The *mucous membranes* take part in varicella. Thomas says that complaints of pain in micturition have often led to his observing vesicles upon the labia of girls, but that on the prepuce in boys they are less common. On the palate they often persist for some time, having slightly reddened bases; but on the lips, the tongue, and the cheeks they are to be recognised only in the form of small superficial ulcers.

Its varieties.—Some of the papules which, as we have seen, constitute the earliest stage of the eruption of chicken-pox, not infrequently abort and disappear, especially those which come out towards the end of the disease. Thomas has recorded a case—the nature of which was established by the fact that the patient's sister had just before had varicella—in which every single papule died away after thirty-six hours, so that no vesicles were formed.

On the other hand, the vesicles sometimes increase in size until they deserve the name of bullæ; this enlargement does not always begin until they have already become scabbed over, and they may go on spreading at the periphery of the crusts for a considerable time. Indeed, it would seem that sometimes varicella lasts longer than any other exanthem. Trousseau describes an epidemic in the Necker Hospital, in which, during from fifteen to forty days, blebs like those of pemphigus kept appearing on different parts of the patients' bodies, leaving ulcerations which lasted for six weeks or two months.

A few similar cases of bullæ succeeding chicken-pox have been recorded—some by Dr Pernet. The writer met with a small epidemic of this bullous varicella, which he described in the 'Journal of Dermatology' for April, 1897 (vol. ix, p. 148).

Mr Hutchinson ('Lect. on Clin. Surgery') saw a rash exactly like fading chicken-pox, which was said to have been out during a month, and which vanished spontaneously afterwards.

A hæmorrhagic eruption has been observed in this disease, but it must be very rare. McCombie says that the symptoms are severe, but that recovery usually takes place.

Course.—The general *symptoms* of varicella are very slight. In some patients the thermometer does not rise above normal during the first twenty-four hours, while the vesicles are coming out. In others there is slight fever of two or three days' duration, but it often passes off in a single day. Moderate fever may last four days. It is particularly to be noticed that the temperature in chicken-pox does not fall, as it does in variola, when the eruption begins to develop itself. Trousseau, however, speaks of successive onsets of fever, occurring regularly at night, and accompanying the different crops of vesicles; and Thomas seems to have observed something of the same kind, although he speaks of such exacerbations rather as tending to interrupt the usual nocturnal rise and morning fall of temperature. The maximum temperature is about 102°. In some slight cases there is said to be complete absence of fever throughout the course of the eruption. The defervescence is usually rapid.

As to other symptoms, the patient may be a little restless, but he seldom complains of headache or need be kept in bed. The tongue is clean, but there may be some thirst and loss of appetite. In a child suffering from chronic laryngitis, Dr Gee noticed decided increase of dyspnœa during an attack of varicella.

Sometimes, however, the symptoms are more marked, particularly in adolescents or adults.

In a case seen by the writer in 1886, the patient, nineteen years old, felt unwell one day, but went out to dinner, and was then obliged to leave the table, and fainted. Next morning the rash appeared on her face and shoulders, and continued in successive crops. The highest temperature was 102° F., and it did not rise with the development of the eruption. There were good vaccination marks. The febrile disturbance lasted about three days. One scar only remained on the forehead close to the hair, where a scab had been repeatedly scratched off. There was an epidemic of chicken-pox among children in the village, one of whom the writer saw.

The only other case attended by comparatively severe symptoms which the writer has seen was in a lady of thirty-two, who apparently took it from her child.

Relapses and sequelæ.—Some writers speak of relapses of varicella; and Thomas, although he denies that the disease is ever followed at once by a second complete attack, admits that he has seen a few vesicles appear as late as a month from the commencement of the eruption.

The question bears on a remarkable cutaneous affection which Mr Hutchinson believes to arise out of chicken-pox, and which he therefore terms "varicella-prurigo." He has recorded notes of sixteen cases, all of which are said to have begun with the characters of varicella, most of them having in fact been diagnosed by medical men as examples either of that disease or of modified smallpox. But it is curious that it never affected several children in the same family; indeed, there is only one instance out of the whole number in which more than one child suffered from the original disease; and any doubts as to the correctness of Mr Hutchinson's views are strengthened by the fact that he gives twelve other cases in which the same skin disease was supposed to have been caused by vaccination.

There is no question, however, of Mr Hutchinson's observation of gangrene as a complication of varicella, though it is happily a very rare one. Instead of the vesicle running the usual course, it becomes tinged with blood, and the scab is very large and black. The slight areola is more marked and of a dusky hue, and under the crust is an ulcer. These multiple gangrenous ulcers may prove fatal in young children. Dr Payne has observed, however, that in cases tubercle is discovered after death.

Predisposing causes.—Chicken-pox is almost confined to *children.* Dr Gee gives a table drawn up from the records of the Great Ormond Street Hospital, according to which infants under six months of age were often attacked, while there was a much larger number of cases among children between six and twelve months old than during any other period of equal length. Most patients are under six, and after ten years of age it very rarely occurs. Most writers say that they have never seen it in grown-up patients, but Heberden relates one case in which a mother caught it from her children, and another instance of it in an adult female was observed by Gregory. Among 584 patients recorded at Basle from 1875 to 1880 by Baader (quoted by Eichhorst), 382 were under five years old and 573 under ten, 7 were between eleven and fifteen, and only two undoubted cases between fifteen and twenty. As above stated, the writer has seen one case at nineteen and one at thirty-two.

Varicella is sometimes sporadic, sometimes epidemic. Thomas remarks that in large towns epidemics are not separated by intervals of several years (as with measles and smallpox), but occur every year or every half-year—in Leipzig regularly a short time after the opening of the infant schools.

This disorder does not affect any particular season, although Heberden speaks of it as occurring chiefly at the end of summer, and Gee thinks it is most common in the fall of the year, in September and October. It is highly infectious. The contagion is probably communicable through the air, and is also capable of adhering to solid bodies. That it possesses comparatively little tenacity may perhaps be inferred from the fact that the extent of an epidemic is not generally large.

Varicella protects efficiently against itself, but not against vaccinia or variola. Second attacks of chicken-pox are almost unknown.

Diagnosis.—The diseases with which varicella has been confounded are acne, pustular syphilide, and molluscum contagiosum, to distinguish which needs only knowledge and care—and variola, especially variola in vaccinated persons, and here the distinction is sometimes difficult. The slightness of the papules, their appearance in successive crops, their distribution and rapid course, will generally decide the question. If on the second or third day there is a vesicle, or a crust on the fifth, it cannot be variola (Heberden).

The *prognosis* of varicella is always good except in the rare cases of subsequent gangrene. There are no other complications and no proper sequelæ; but occasionally children are some time in regaining strength, and both Gee and Eustace Smith have seen tuberculosis follow chicken-pox.

No *treatment* is needful, except to ease the troublesome itching. The vesicles should as far as possible be protected from scratching, to prevent cicatrices. Since the contagion of varicella is very active, isolation is desirable, particularly when young children are in the house.

MUMPS

> "Where's Junius? In his cabin, sick o' the mumps, sir."
> BEAUMONT and FLETCHER, *Bonduca*, i, 2.

General characters of the disease—Clinical course—Orchitis and other complications—Anatomy—Prognosis—Treatment.

Synonyms.—Parotitis—Cynanche parotidæa (Cullen)—Parotitis epidemica.—*Scotticè*, The Branks.—*Fr.* Les Oreillons.—*Germ.* Ziegenpeter.

Definition.—A specific contagious fever, running a short course, with painful swelling of the parotid glands.

Characters.—The salivary glands, like the pancreas, are very little subject to the diseases which affect the liver and kidneys, or the mamma and testes. The most frequent affection is one which looks like a local inflammation, but is really part of a specific disease, transmitted by contagion, occurring epidemically, and protecting against future attacks. For this disease the popular name of Mumps is the most distinctive.

Like scarlatina, measles, rubella, chicken-pox, and whooping-cough, it is for the most part confined to children between five or six and fourteen; but, like them, it may readily be conveyed to unprotected adults. It is said to affect more boys than girls. In a Russian epidemic in 1885 there were 170 male and 90 female patients. The secondary orchitis is most often seen in boys about the age of puberty and in young men.

The contagion of the disease appears most often to be transmitted by the breath. It is active from the very first, and sure as well as active. No microbe has as yet been identified as the cause of the disease.

Mumps is endemic like measles and whooping-cough, but local epidemics may often be recognised, as in America during the civil war of 1862-3.

Clinical course.—Incubation lasts from two to three weeks. Dr Dukes gives fourteen to twenty-five days, other observers sixteen to twenty, and the Clinical Society's committee, 1892 (xxv), fourteen to twenty-five.

The earliest symptom is commonly an aching pain in the parotid region on one side, increased by every movement of the jaw in speaking or taking food. But sometimes malaise and pyrexia and sometimes vomiting precede these local symptoms by a day or two. Œdema very soon follows the pain; the hollow between the mastoid process and the jaw is filled up, and a diffused swelling makes the lobule of the ear project, while it spreads over the cheek towards the angle of the mouth, and downwards into the neck. The submaxillary glands are next involved, and a little later the opposite parotid; so that in the course of from three to six days the whole of the face becomes surrounded by a mass of firm doughy infiltration. There is an enormous double chin, and the natural contour of the throat is lost. The skin over the affected parts is but slightly reddened, and may be pale and waxy in appearance. Internally the swelling extends to the tonsils and the pharynx. The movements of the jaw are greatly impeded, and the teeth can only be separated to admit the end of a spoon, so that the patient must confine himself to liquid or very soft diet—milk, jellies, custards, and eggs beaten up.

Sometimes the saliva seems to be secreted in excess; but its quantity and quality are, as a rule, unaffected. The head is kept fixed in one position, with the face directed straight forwards; or if, as sometimes happens, only one side is affected, the head is turned towards that side. Pain and tenderness continue more or less severe; and sometimes the patient complains of earache, or of a continuous ringing sound, with partial deafness.

On about the fourth day the pyrexia ceases. It seldom runs high, but temperatures of 104° are now and then recorded, and typhoid symptoms have been occasionally observed. The brown dry tongue sometimes observed is, as Dr Goodhart says, due to the mouth being kept open.

Soon afterwards the swelling begins to subside, and so rapidly that within three or four days it entirely disappears. Thus the whole duration of the disease is usually a week or ten days, rarely a fortnight. There is sometimes desquamation of the cuticle over the affected parts.

Like other specific diseases, mumps is sometimes so mild that it would be overlooked but for the presence of other cases at the same time.

Complications.—In some cases, while the inflammation of the face and neck is passing off, or after it has ceased, the patient is attacked with acute "metastatic" inflammation of one *testicle*, generally (it is said) the right. According to Dr Duke's experience at Rugby, this is most apt to occur on the eighth day, but only if the boy is allowed to leave his bed. The whole gland becomes swollen and painful, and there may be acute hydrocele, with œdema of the corresponding side of the scrotum. After a short interval the other testicle is sometimes attacked. This painful complication is very seldom seen under fifteen years of age.

Now and then this secondary orchitis assumes a most alarming character, though apparently without real danger to life. High fever and delirium are sometimes present, or severe vomiting, or extreme prostration. In one case related by Trousseau collapse was suddenly developed; in another the patient fell into a typhoid state. The second case was the more obscure because the initial attack of mumps had been so slight and transient that it passed without notice, and nothing could be learnt about it until consciousness returned.

Some years ago the writer saw a case of orchitis from mumps in which the pulse became remarkably slow, and remained so for several days: the temperature also fell below normal, and the breathing was much reduced in frequency.

The inflammation of the testicle usually lasts from three to six days, and then rapidly subsides. Occasionally, however, it leads to permanent atrophy of the organ.

The liability of mumps, if appearing after puberty, to set up orchitis can only be referred to that correlation of distant organs in their proclivities to disease, of which there are many examples. Mr Stephen Paget has drawn attention to other instances of relation between parotitis and disorders of the abdominal viscera, of which the testis is one ('Lancet,' April 17th, 1886, and 'Brit. Med. Journ.,' March 19th, 1887).

In girls affected with mumps it is said that the *mammæ* or the vulva sometimes exhibit a like tendency to swelling and inflammation. This fact illustrates a case observed by Peter, in which a young woman who had amenorrhœa was several times attacked with parotitis at what should have been her catamenial periods, while on other occasions one of the labia became swollen and painful. According to some authors, the *ovaries* may be the seat of "metastatic" inflammation after mumps.

Other complications recorded are ophthalmia, acute bronchitis, bubo and urethritis, and acute inflammation of the middle ear. In other cases of deafness following mumps there is no tympanitis, and the lesion is referred to the labyrinth. According to Sir Wm. Dalby, it may be either "catarrhal"—when it passes quickly away, or "nervous"—when the condition is usually permanent.

Other very rare sequelæ are meningitis, facial paralysis, hemiplegia, and mania. Since none of the reported cases of meningitis seem to have proved fatal, it may be permitted to doubt whether there is really acute inflammation of the pia mater. The symptoms might be due to specific toxæmia, and this would also be a probable cause of the peripheral neuritis, to which nervous deafness and other paralytic affections may be plausibly ascribed.

Anatomy.—With regard to the exact seat of the morbid process in mumps there is still some uncertainty. It may affect any or all of the salivary glands on one side, or both; or, as Dr Goodhart believes, the cervical lymph-glands alone. The condition is probably a diffuse inflammation of the acini spreading to the stroma of the glands; and the connective tissue outside the salivary glands is involved in the inflammatory œdema. The secondary orchitis appears to be parenchymatous, and leads to destruction of the glandular epithelium of the tubules.

Protection.—Mumps, like other specific diseases, protects from itself. But sometimes, when one side has been affected, the other follows suit within a week or fortnight, or there may be a relapse affecting both sides. Apart from these relapses, cases of a second attack are occasionally met with: in one known to the present writer, a boy had mumps three times during his school life.

The *prognosis* of mumps is favourable. Even when the fever runs high, and is attended by orchitis with delirium, there is very seldom cause for alarm. Neither the inflamed parotid nor the inflamed testis ever suppurates; but the latter may atrophy, and become permanently useless.

Very little *treatment* is required in cases of mumps. The patient should be kept indoors and fomentations may be applied to the swollen parts. An ice poultice and belladonna are probably the best local applications for orchitis after mumps. Jaborandi (or its alkaloid pilocarpine) has been proposed as a specific remedy at the outset of the disease, and seems to deserve a trial.

WHOOPING-COUGH

"[illegible] lies under something like a chin-cough."
ADDISON, *Tatler*, No. 121.

[illegible]—*Clinical course—Complications and sequelæ—* [illegible] *and general ætiology—Anatomy—Pathology—* [illegible]

[illegible] Tussis clangosa (Glisson)—Tussis puerorum convulsiva [illegible] Med. iv. 5. § 8. 1685: also Heberden and Cullen)—[illegible] Sydenham. 'Proc. Integr.,' cap. xliv. 1695).—*Fr.* [illegible] (whence "hooping" cough, rationalised into [illegible] Keuchhusten.—*Vernacular.* Chin-cough, a corruption [illegible] Kink-cough, or Kink-host (Dutch Kinkhoest). [illegible] a catch in the breath (Skeat).

[illegible] specific infective disease, running an indefinite and often [illegible] with coryza and bronchitis.

[illegible] Hooping-cough, or whooping-cough, was probably [illegible] popular titles from time immemorial; but, like scar- [illegible] described by physicians in the seventeenth century—by [illegible] and by Sydenham. It is a very frequent and wide- [illegible] next to scarlatina, more fatal than any other in child- [illegible] under one year it is probably the most fatal of [illegible] to its infection, as they are to that of measles. [illegible] but it is so common that most adults are pro- [illegible] attack.

The *incubation* of whooping-cough after infection is [illegible] a fortnight as a rule, but often less.

[illegible] or first *stage*, as it is sometimes called, there is [illegible] some cough and some pyrexia. There may also be [illegible] and sneezing. The child (whooping-cough chiefly [illegible] seven) is pale, fretful, and restless. According to [illegible] sometimes remarkably frequent, recurring fifteen [illegible] in the minute: so that one might suspect the [illegible] the incessant cough. But, as a rule, there are [illegible] diagnosis, or even of suspicion, unless other cases [illegible] same family or neighbourhood.

[illegible] prodromal stage is very uncertain. Sometimes in [illegible] day or two, or it may perhaps be entirely absent. [illegible] or two, four, or even five weeks. Ten days is [illegible] length. In some cases it is believed to conitute the whole of the disease, the patient recovering without more disnctive symptoms; this was suggested by Cullen, and it has been recently upported by Dr R. J. Lee and Dr Eustace Smith.

In all but these somewhat doubtful exceptions a *developed stage* ucceeds, characterised by the appearance of the "whoop" with the cough. The change in question may occur either suddenly or very gradually. Some healthy children, especially if they happen to cough while crying, make a crowing inspiratory noise that is not very different from the true whoop; and children under a year old seldom whoop at all. The so-called whoop is not a sound *sui generis*. It is a deep inspiratory stridor, differing only in loudness and length from that which follows the first inspiration after any prolonged fit of coughing.

When, however, whooping-cough is well marked, there can be no mistake about it. Pyrexia is now absent, but the pulse continues very frequent. There may be a good appetite, and except for the cough the child may appear to be well. The cough comes on in paroxysms, of which there may be only ten or twelve in the twenty-four hours, or as many as sixty or eighty. If not above twenty it may be called a mild case. They are usually more frequent in the night than during the day. Each begins with a series of short explosions in rapid succession, and these expiratory efforts have no noticeable inspiratory movements between them; then, after the air in the lungs has been reduced to the utmost, there comes a long-drawn inspiration, attended with a characteristic whooping or crowing sound. Sometimes, as observed by Dr Newnham at the Evelina Hospital, the paroxysm begins with an inspiration. Such a succession of attacks may continue for several minutes, and generally end by expectoration of viscid mucus, or by vomiting.

These paroxysms of cough, if severe, cause great distress. The child, when it feels one coming on, runs to its nurse for support, or it clings to a chair or table so as to diminish the shock as much as possible. Patients old enough to take notice describe a tickling sensation in the larynx, or a feeling of compression about the throat, as though the air could not pass. Those who are younger betray the approach of an attack by restlessness and anxiety of face.

If the paroxysms are frequent and prolonged, cyanosis follows. The eyes protrude, the face and the neck swell, and a cold sweat breaks out, while tears run down the cheeks, although the child has no breath to cry.

Hæmorrhage is by no means infrequent; the expectoration may be tained bright red by blood (which probably comes from the fauces or rom the larynx), the nose may bleed, one or both of the conjuncivæ may become ecchymosed, the tears even may be mixed with blood, r the tympanic membrane may be ruptured, with the escape of blood rom the ear on one side or on both. Steffen (in 'Ziemssen's Handuch') says that albumen sometimes appears in the urine. He adds that momentary stoppage of the heart is not uncommon, and cites the case of girl six years old, where there was a temporary loss of sight during ach attack. He also speaks of a boy nine years of age, in whom, when evere paroxysms occurred, internal strabismus of the right eye was noticed, whereas the left looked straight forward, being held fixed with tonic spasm. Sometimes spasmodic movements of the muscles of the face occur. General *convulsions* are occasionally met with, and have before now proved fatal.

child may be out of breath for a time and glad to lie down; sometimes he complains of headache, and sometimes is dull or fretful. But he frequently begins to play again at once, and seems as lively as though nothing had happened. When there has been vomiting he often asks for food, and eats it eagerly.

Complications and sequelæ.—The mere violence of the cough may produce hernia or prolapsus ani. Subconjunctival ecchymosis also is not infrequent, and may serve as a hint to a diagnosis. Subdural hæmorrhage has been found in more than one case after death.

Among the more remote effects of the violence of the cough is the formation of one or more shallow whitish ulcers on the under surface of the tongue by the side of the frænum; they appear to be caused by the lower teeth, against which the tongue is forced outwards during the paroxysm. In this country Dr Thomas Morton first drew attention to these ulcers in a paper read before the Harveian Society in 1876; but on the Continent they had been previously described by Bouchard and others. Morton detected them in about 40 per cent. of his cases, generally between the third and the fifth week. He once saw an ulcer in an infant who had no teeth, when the tongue may possibly have been injured by the edge of the gums. The recognition of these sublingual ulcers may sometimes be useful in diagnosis.

Bronchitis and broncho-pneumonia are the most frequent and most important sequelæ. They are usually combined with lobular collapse.

Another result of whooping-cough is the production of pulmonary emphysema, and even the extravasation of air into the interlobular and subpleural areolar tissue. Thence it has been known to reach the mediastinum, and ultimately to diffuse itself under the skin of the chest, throat, and limbs. Or a pulmonary alveolus may rupture, so that pneumothorax results. Either of these accidents may cause a fatal result, but happily they are both rare. Empyema is a not infrequent sequel.

Ascending paralysis has been once or twice observed after whooping-cough, probably due to peripheral neuritis; and Dr Samuel West recorded a case of right hemiplegia with aphasia and athetosis, probably due to meningeal hæmorrhage during a paroxysm of whooping-cough ('Brit. Med. Journ.,' January 22nd, 1887).

In some cases, even when the paroxysmal stage of whooping-cough has completely passed off, the child nevertheless fails to recover its strength, and ultimately dies of marasmus; or pulmonary phthisis, with caseous bronchial glands, develops itself. Permanent deafness and otorrhœa are said to be occasional results of the injury sustained by the tympanic membrane during the paroxysms.

Protection.—Those who have once had whooping-cough are protected against future attacks. Indeed, Steffen says that its occurrence for the second time in the same individual, although not absolutely impossible, is far more rare than that of scarlet fever, smallpox, or any other exanthem.

Infection.—Whooping-cough is, unfortunately, most contagious in the first or prodromal stage, for that is when it is most easily overlooked. How long it continues contagious is uncertain, probably not during the whole of the whooping stage.

The contagion of whooping-cough is not easily transmitted to a distance and is very seldom conveyed by persons not themselves affected with the disease. However, one can hardly doubt that it might be spread by the use of handkerchiefs or towels contaminated by dried secretions from the air-passages of patients; and a case is recorded in which linen, sent to be washed on an island from a ship with whooping-cough on board, conveyed the disease to the island.

In the Clinical 'Transactions' (vol. xi, p. 238) Dr Bristowe recorded a case in which a lady carried the contagion from Sydenham to London upon her dress. During a visit to the former place a little boy suffering from whooping-cough was climbing upon her knee, and coughing and sneezing over her; she returned home the same evening, and early next morning one of her children was found playing over her dress, which had been laid upon an ottoman. This girl took the disease, and afterwards gave it to two other children. A further point of interest is that the boy himself had only begun to have a constant troublesome cough on the very day on which the lady visited him; in fact, he was staying away from home in the hope that he might escape the disease, which was prevailing among his brothers and sisters. The case is also important as tending to show that the period of incubation in whooping-cough is about a fortnight; for the girl fell ill exactly thirteen days after she was exposed to the contagion, and the two other children sickened after about the same interval.

Climate does not appear to have much influence upon the prevalence of the disease, except that perhaps cold and damp countries are most favourable to it; and Hirsch has shown that it is not more apt to be epidemic at one season of the year than another. When once established mild warm weather is favourable, and cold east winds unfavourable to the patient.

Sex and age.—There are doubtless great individual differences as regards susceptibility to the contagion. Female children are more liable to be attacked than males, and more of them die. The age at which whooping-cough is most common is between the second year and the eighth. Of Dr Goodhart's 314 cases 62 were under a year old, 212 were between one and four, 65 between four and six, and 13 between six and ten.

Barthez and Rilliet recorded the case of an infant whose mother had had whooping-cough for three weeks before its birth, and in whom severe paroxysms occurred on the second day. Sir Thomas Watson relates in his lectures how the grandchild of his bedmaker at Cambridge whooped on the very day of its birth, another child in the same house having been affected with the disease for three weeks before. Dr Eustace Smith found more than a fourth of a series of cases occurred in children under one year old.

Whooping-cough is sometimes observed in adults up to forty or fifty, or even a still greater age. Heberden met with one case in a woman aged seventy, and another in a man aged eighty. An eminent London physician suffered severely from an attack of whooping-cough when more than sixty-five years of age; and the late Sir John Russell Reynolds told the writer how another member of the profession who came to consult him for a short dry brassy cough, thought it indicated aneurysm. He had caught pertussis from a grandchild.

An association is often traceable between epidemics of measles and those of whooping-cough, children falling ill with the latter disease soon after having passed through the former. Such cases are peculiarly apt to be accompanied with severe broncho-pneumonia, and to terminate fatally, the measles having often already brought the lungs into a morbid condition. Sometimes, however, the relation between the two diseases is reversed, measles breaking out after whooping-cough has existed for some time. It is then sometimes noticed that the paroxysms become much less frequent and much less severe than before, and that they remain so as long as fever persists; but the same temporary improvement is often seen in whooping-cough when pyrexia appears from any cause.[*]

Anatomy.—In cases of death very few changes are found. The most important are in the lungs, which show patches of lobular broncho-pneumonia (pulmonary catarrh) secondary to catarrh of the trachea and bronchi, and, particularly in the case of infants, extensive lobular collapse. The bronchial mucous membrane is congested or ecchymosed, but the muco-pus covering it not abundant. The bronchial lymph-glands are often swollen—a result of bronchitis. If they are caseous, that is the result of secondary tuberculous infection, and only indirectly of the whooping-cough. In rare cases meningeal hæmorrhage has been observed, probably due to the paroxysms of cough.

Dr David Lees has put on record the interesting observation that after death from whooping-cough the upper orifice of the larynx between the arytænoid folds of mucous membrane is narrowed by the epiglottis being more doubled on itself than is usual.

Pathology.—There has been much doubt as to the true nature of this familiar disorder. Some writers have attributed it to pressure upon the vagus nerve by swollen bronchial glands; some have maintained that it is a mere catarrh of the respiratory mucous membrane. Beare, on the ground that the paroxysms of whooping-cough resemble those caused by a foreign body in the larynx, would have it that the fundamental lesion is an inflammation of the tract immediately above the vocal cords. But it is very doubtful whether laryngitis is constant or even frequent.[†]

It is clear in any case that whooping-cough is not produced by any local and accidental cause. It is a specific infection, as proved by its contagiousness and by the protection it affords. The cough and local symptoms are comparable to the diarrhœa of enteric fever, the sore throat of scarlatina, or the parotitis of mumps. And it is probable that the seat of the disease is rather in the nervous system than in the larynx and trachea. Any slight lesions found in the trachea and lungs may be well referred to the cough as their cause, not their consequence. If this be admitted, it seems reasonable to exempt the cerebrum, spinal cord, and peripheral nerves from participation in the disease, and to fix its seat provisionally in the respiratory centre of the bulb.

It must be allowed that in several particulars whooping-cough differs from other specific infective diseases. There is only slight and irregular fever; the course is not definite and self-limited like that of the eruptive fevers, typhus, smallpox, or rubella; and there is no exanthem.

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[*] There is a parallel instance in hydrophobia, and it is not impossible that Pasteur's discovery of the actual presence of the virus of hydrophobia in the nervous centres, and of its multiplication there, may hereafter be found to supply the key to the pathology of whooping-cough. One can easily imagine that the poison of this disease, having originally entered the air-passages from without, and having set up a catarrh there, is during the prodromal stage conveyed to some part of the central nervous system, and there sets up the peculiar spasmodic cough.—C. H. F.

[†] According to Rossbach ('Berl. klin. Woch.,' 1880) no morbid change can be seen with the laryngoscope during life; Meyer-Hüni ('Ztsch. f. klin. Med.,' 1880) saw reddening and slight swelling of the larynx and trachea, but not of the cords. Von Herff found decided hyperæmia of the larynx in the whooping stage, and pellets of mucus lodged on the posterior wall of the larynx.

May we not regard the incubation and prodromal stages as those which correspond to the course of an exanthem like measles, and the more obtrusive whooping stage, which gives its name to the disorder, as really a nervous sequel beginning in a toxic affection of the respiratory centre. but continued by the effect of frequent repetition, which in conscious functions we call habit? This view would agree with the increasing belief that whooping-cough is very contagious at first, and very little so in its later stages; and also with the effect of change of air, other diseases, or even accidents, in putting an end to a whoop of many weeks' duration.

It is in any case probable that the contagion of whooping-cough is an organism analogous to those which produce other infective diseases, and possibly it has already been seen with the microscope.*

Burger, in the 'Berliner klin. Wochenschrift' for 1883, asserted that the sputum in whooping-cough always contains large quantities of bacteria, which appear as rods of oval form, sometimes constricted in the centre, generally scattered irregularly, but occasionally arranged in chains. They are said to be easily brought into view by staining with fuchsin or methyl violet. Ritter and Kohn have also discovered microbes in the sputa, but there is at present no ground for regarding any of them as pathogenic or specific.

Prognosis.—It is exceedingly rare for a paroxysm of whooping-cough directly to destroy life. A very young child, however, may die from closure of the glottis, or perhaps from syncope, or from the rupture of an intra-cranial blood-vessel. When the attacks are very violent, and one follows another with extreme frequency, they sometimes end in apathy and stupor which may be fatal.

Much more often death ensues from the pulmonary complications of the disease, bronchitis and broncho-pneumonia; it is said that they kill half or two thirds of all the children attacked. As may easily be supposed, patients who before were weakly and delicate are more likely to succumb to whooping-cough than the strong and healthy. It is far more dangerous for infants than for older children, and among the poor than among the rich. The worst cases are those complicated by rickets or by adenoid growths in the pharynx.

There are differences of severity in different epidemics, and pertussis is more serious during the cold seasons of the year than in the summer. When it affects adult patients it is very distressing, but it is not dangerous, nor is it often prolonged.

Whooping-cough, unlike the specific fevers hitherto treated of, has no definite period. Its course is protracted but uncertain, and it is difficult to say when the specific malady ends and its sequelæ begin.

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* As far back as 1870 Letzerich figured in 'Virchow's Archiv' thallus filaments and spores, which he found abundantly in the sputa of patients with whooping-cough, and he asserted that he had succeeded in producing a like malady in rabbits by inoculating the trachea with the product of his cultivations. In the 'Jahrbuch der Kinderheilkunde' for 1876 Tschamer supported Letzerich, and maintained that an identical fungus, found on oranges or apples, is capable of giving rise to whooping-cough when inhaled. It is, however, not a fungus, but a bacterium, that we should expect as the *contagium virum*. This has been sought for by Koplik, Hensel, and other recent observers, but there seems at present to be no [illegible] that the specific microbe has been found.

After a variable period, a month or more, the violence of the paroxysms subsides. The expectoration becomes looser, more abundant, and more puriform. The cough becomes less spasmodic, and at last the characteristic whoop disappears after from six weeks to two or three months. In a case related by Trousseau its duration was only three days; the patient was a child three years old, an inmate of the Necker Hospital. After the paroxysms have ceased, symptoms of ordinary bronchial catarrh may remain for a time.

The child may be a long while in regaining its spirits or its strength, especially if the season happens to be winter, so that there is difficulty in getting it out of doors and into the fresh air. Sometimes, when the whoop has apparently passed off, a fresh attack of catarrh brings it back again for a few days in as marked a form as ever. Even after the lapse of a year it may be noticed that if the child "catches cold" the cough is attended with a similar sound.

Treatment.—We have no specific or very effectual treatment of whooping-cough. Sydenham depended on venesection, Fothergill and Armstrong advocated nauseating doses of antimony; and many other modes of treatment, as arsenic and nitric acid, have been from time to time introduced, to be in turn forgotten.*

Nevertheless treatment is not useless. In bad weather the patient should be kept in a spacious room, warm and equable in its temperature; for exposure to cold has a marked tendency to bring on the paroxysms; according to Hauke the presence of an excess of carbonic acid in the air has a like effect. But in fine weather the child should be taken out of doors every day. When the disease lingers in its course, nothing is so likely to bring it to an end as change of air, especially to the sea-side. The meals should be nourishing and frequent, and should be given directly after a paroxysm. Crying and excitement of every kind should as much as possible be prevented.

As regards drugs, belladonna, hydrocyanic acid, chloral, bromide of potassium or ammonium, hemlock, and henbane appear each of them to diminish the frequency and the severity of the paroxysms in some cases, and even to shorten the duration of the disease. But not one of these medicines can be said to succeed in most cases; and they often fail altogether. Alum in grain doses every four hours was introduced by the late Dr Golding Bird as an astringent in the later stages of the disease, and has often proved useful. Quinine also is sometimes serviceable; and the supposition that it checks the growth of the specific microphytes has led to its administration by insufflation into the air passages. Goodall and Washbourn add hydrochloride of cocaine in doses of $\frac{1}{16}$ to $\frac{1}{4}$ of a grain three times a day, as having been employed with some success.

It has lately become a common practice to treat whooping-cough by inhalations. The earliest attempts of this kind consisted in placing patients in the purifying chambers of gas-works, where the air is laden with tarry products—as well as with sulphuretted hydrogen and ammonia.

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* Some of them are not free from danger. In the same number of a medical journal which narrated successful treatment of whooping-cough by antipyrin and antifebrin, there appeared an account of a doctor in Germany who nearly killed his own child by repeated doses of antipyrin during whooping-cough.

A more recent plan is to impregnate the air of the patient's chamber with turpentine, or with eucalyptus, or with carbolic acid. A solution of phenol, for example, is diffused through the room by means of a spray apparatus, or by simply heating a vessel containing it. Children who are old enough may be induced to inhale a weak carbolic spray for fifteen or twenty minutes two or three times a day. Thorner, in the 'Deutsches Archiv' for 1878, reported very favourably of this practice; for about a week there was little change, but at the end of that time the symptoms of the disease began rapidly to subside. Successful results by similar means are met with in this country. Inhalations of the vapour from a boiling 2 per cent. solution of salicylic acid have also been recommended. Dr Goodhart has found both phenol and salicylic acid disappointing. He recommends Tr. Camph. Co. during the early stage, and during the later whooping period belladonna; with sodium or potassium carbonate, alum, bromides, or chloral hydrate only when belladonna fails.

Marshall Hall's proposal to protect the infant at night from draughts by a mosquito curtain has been tried with success by Dr Eustace Smith, who also recommends the child's room being filled with vapour from eucalyptus, creasote, or phenol: internally, hydrate of butyl chloral in one-grain doses, sulphate of zinc (gr. $\frac{1}{6}$), and belladonna or atropia; and, after the acute stage has passed, quinine in full doses.

INFLUENZA

> **" What evil star**
> **On you hath frown'd and poured his Influence bad ? "**
> **SPENSER.**

*History—Course and symptoms of the catarrhal form—its sequelæ—**Mild** cases—feverish colds—Cardiac form—Abdominal form—Nervous form—Mortality—Ætiology and pathology—the bacillus—Diagnosis—Prognosis **and** treatment.*

Synonyms.—Tussis epidemica (Sydenham)—Catarrhus a contagio (Cullen)—Catarrhus epidemicus—" the Chinese catarrhal fever "—" the Russian influenza."—*Fr.* La grippe.—*Germ.* Grippe.*—*Ital.* Influenza.

Definition.—A specific infective Fever, running a short course, with varied local symptoms, very apt to recur and to leave sequelæ.

History.—An epidemic, and probably specific disease, was well known to our fathers as the *influenza*, a name of Italian origin, which came into use in 1741, and denoted its external origin and wide-spread sway. Its last invasion as an epidemic was in 1847-8, and it was only a memory when the first edition of this book appeared in 1886. Since then it has reappeared with the severity of a new epidemic, and has now become endemic in Western Europe.

Like other specific diseases, its early history is obscure. It may, however, be probably identified with an " epidemic catarrh " recorded in 1173, with four similar epidemics in the fourteenth, and with four more in the fifteenth century. According to Dr Parkes (in his article in ' Reynolds' System '), there were eleven in the sixteenth century, sixteen in the seventeenth, and eighteen in the eighteenth. Between 1800 and 1850 there were ten, of which three were the most important: one, in its spread over different countries, occupied the years 1830 to 1833; another occurred in 1837, when " half London " was attacked; and the third in 1847-8. This last was a very severe epidemic. Many died from pleurisy, bronchitis, pneumonia, or pericarditis. At one time it was said that the whole staff of Guy's Hospital was laid up.

There was no reappearance of the disease, at least as an epidemic, for a period of more than forty years. But then, after having died out of the memory of the present generation, a severe epidemic of influenza appeared in 1889, and spread over the whole of Europe and most other parts of the world.

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* The word *grippe* is said to be derived from the Polish *crypka* or *grypka* (= *raucedo*).

When the newspapers of November 26th, 1889, announced "a curious epidemic raging in St. Petersburg," some authorities identified it with the dengue fever then prevalent in Greece and Turkey. The Russian doctors called it influenza, "the symptoms being fever and headache, accompanied by a running cold." Regiments were incapacitated and railways were interrupted by the suddenness and severity of the outbreak. The disease spread quickly through Poland to Hungary and Vienna, where there was a considerable mortality. At the K. k. allg. Krankenhaus between December 7th and 14th, 165 patients, 57 nurses, and 77 physicians were attacked. Other parts of Germany were visited, though less severely, as well as Italy, Spain, and France; afterwards the United States, Australia, and New Zealand. At the end of the year several cases had been reported at and near Chiswick, and by January and February, 1890, it was frequent in London, and our hospital wards and out-patient rooms were crowded with cases of catarrh and influenza. In the Foundling Hospital in January, 95 out of 315 children were attacked; and at the London Orphan Asylum 115 out of 500. At Hanwell, of 174 attendants 93 suffered, and of 1141 inmates of all ages 178. The disease after appearing in London visited the great provincial towns successively, and afterwards smaller places like Colchester and Cambridge, where at one college, St. John's, there were 70 cases. The severity of the epidemic was over in April (1890), but scattered cases occurred until the end of May.

Since 1890 the disease appears never to have left us. From time to time pauses have occurred with only a few scattered cases, and then they have become numerous again; but the severity has, on the whole, been less since influenza has become endemic than in the epidemic outbreak of 1889-90.

Course.—The incubation period is a short one, not more than a day or two.

Sir George Baker, in his 'Opuscula Medica' (second edition, London, 1771), gives the following account of the epidemic of influenza in the year 1762. It was characterised by alternate heats and chills, by a constant cough, sometimes dry, occasionally accompanied by a little thin mucous expectoration. There was depression of strength, a sense of weight and severe pain in the forehead and temples; inflamed, swollen, and watery eyes with photophobia; frequent sneezing and altered voice. There was painful rawness felt in the windpipe and chest, with, in some cases, a feeling of choking, and wandering pains in the arms, legs, and sides. The fever was chiefly nocturnal, but even then so slight that it rarely interfered either with sleep or with food. There was more or less perspiration, and when it was profuse the disorder was relieved or cured. The tongue was white and thickly furred; the urine was dark, and threw down a lateritious deposit. In all cases there was more depression of spirits and loss of strength than the character of the disease seemed to account for, and convalescence was often tedious and imperfect. Many cases of abortion occurred in London from this disease. In a few of the more severe cases there was a miliary eruption.

When Sir Thomas Watson was first called to two cases on April 3rd, 1833, the symptoms were just those which mark the commencement of an attack of fever, and he did not then know what was about to happen; but by the close of the following day all London was smitten with the disease.

The attack usually begins abruptly, often with a rigor; the temperature rises higher on the first day than is common in any disease except scarlatina. Severe headache follows, either frontal and deeply seated "behind the eyes"—as a medical student told the writer, just in the foramen rotundum,—or it is referred to the root of the nose and the frontal sinuses. At the same time there is coryza, with sore throat, injected eyes, and at first dryness, then running from the nostrils. The tongue is thickly furred, and there is complete anorexia. The patient feels prostrate from the beginning, sometimes drowsy, and sometimes wandering, or actively delirious. Lumbago and pains in the limbs, with the not infrequent vomiting, resemble the onset of variola. The sore throat, coryza, and subsequent bronchitis do not differ from those of a "severe cold." The bowels are usually confined, but sometimes there is sharp diarrhoea or severe sweats. A scarlatiniform rash has been occasionally observed. The urine is febrile, but very seldom contains albumen. The pulse is rapid, and usually full and with rather high tension—rarely weak and dicrotic, but sometimes intermittent. In pregnant women abortion may occur; or, if the menstrual functions have been suppressed from other causes, they are sometimes re-established. Facial herpes has been observed.

The course of the fever, as a rule, is not above three days, though sometimes four or five. The fall of temperature is very rapid, more so than in pneumonia. In one case, a strong, healthy young man was in full health on Saturday, had a temperature of 104·5° F. on Sunday, and by Tuesday afternoon was free from fever and pain.

If the attack lasts above five days, either pneumonia has developed or the catarrh of the air-passages has passed into ordinary bronchitis. The subsidence is occasionally more gradual, and sometimes marked with a critical sweating or diarrhœa, sometimes with epistaxis. The patient's convalescence is always slow, and he is long in regaining his strength.

Relapses are not infrequent, but few persons suffer more than one distinct attack in the same epidemic. On the other hand, to have had influenza confers no immunity against its recurrence on a subsequent occasion; on the contrary, those who have suffered once are likely to suffer again.

Sequelæ.—The most important sequel is acute fibrinous pneumonia.

In January, 1890, the writer saw in consultation with Dr Addison, of Colchester, a young man of twenty-seven ill with acute pneumonia of the left lung after influenza. He made a good recovery; but a brother three years younger had died a week before of the same disease, double lobar pneumonia after influenza; and a maid who had nursed him also died with acute double pneumonia. His mother, a younger brother, and a man-servant suffered at the same time from influenza, and the servant from pneumonia; but these three patients recovered.

Lobular pneumonia (pulmonary catarrh) is perhaps still more frequent, particularly in children and in elderly people, and it is not less dangerous to them.

In most cases great prostration follows, and in many there is muscular weakness with malaise for several weeks, or even for months, after recovery. In others the bronchitis common during an attack of influenza does not subside, as usual, in about a week after the fall of temperature, but becomes chronic; or in children may develop into broncho-pneumonia, with collapse; or it may end in acute tuberculosis or in phthisis. Another sequel, more common in children than in adults, is empyema.

Recovery from influenza has sometimes been followed by parotitis, necrosis, and distant abscesses.

There remain some varieties of this protean malady which differ remarkably from the most common and classical catarrhal, bronchial, and often pulmonary form just described.

Mild catarrhal cases.—When an epidemic of influenza is going on we meet with cases of coryza and sore throat with cough and rise of temperature, which are recognised as specific by the weakness and depression which follow them being quite disproportioned to the degree or duration of the fever.

But we also meet with similar cases when there is no epidemic of more severe forms, and the question arises whether these feverish colds are not some of them really *sporadic influenza*. They are generally assumed to be the result of a chill, and few who are subject to them but can recall instances of their following directly and unmistakably as the result of exposure to cold wind, or to rain, or to a draught of air. The first effect is a sudden feeling of chilliness, an involuntary shudder (*i. e.* a slight rigor), and a fit of sneezing. The reflex character of this paroxysmal neurosis is shown by its being caused by a bright light irritating the eyes, as well as by cold air in the face or snuff in the nostrils, and also by its being inhibited by pressure on the upper lip or bridge of the nose, and completely stopped by covering the face. Indeed, the action of the domestic remedy of smearing the nose with tallow or cold cream is to prevent sneezing by protecting the skin from cold.

But just as undoubtedly, a feverish cold sometimes comes on with no unusual exposure to cold, rather after sitting in hot rooms; it "runs through the house" like a contagious disease; it is accompanied by malaise and pyrexia; it lasts for a limited time, and then subsides.

In one point it differs from other specific fevers, but resembles epidemic influenza—it does not protect against itself; or rather, perhaps, it protects for only a short time. While relapses from an ordinary cold in the head are very frequent, a severe feverish cold rarely occurs except at considerable intervals.

The symptoms which follow the initial sneeze and shivering are pain in the throat felt on swallowing, together with headache and severe aching pains in the loins, the thighs, or the back and limbs generally. To this stage succeeds coryza and a short, dry, painful cough. The temperature rises, sometimes to 103° in an adult and often to 102°, the urine becomes febrile, the pulse quick, and the skin dry. After a day of distress the skin begins to act, and the symptoms are somewhat relieved, but the coryza becomes more severe, a little mucus is expectorated, the bowels are constipated, and some headache and pains in the limbs continue. After three, or at the latest four days, the feverish stage is almost always over, but there is great prostration of strength for so short an illness, and considerable bronchial as well as nasal catarrh. Finally there remains some cough and expectoration for a week or ten days more. Occasionally a sharp attack of diarrhœa seems to take the place of the nasal catarrh.

This is surely sporadic influenza, as seems to be proved by pneumonia sometimes following it, and by the specific bacillus having sometimes been found present (Lindenthal, 'Wiener klin. Wchnschrft.,' No. 17, 1897).

The *treatment* of these "influenza colds," as feverish catarrh used to be called, is first to avoid fresh chills, and secondly to keep warm. A hot bath or a Turkish bath is excellent, and for the time the patient feels well, but they do not cure; within an hour he is as ill as ever. He must go to bed, drink warm diluents, and get the skin to act. Dover's powder is certainly useful with most people. Some find antimonial wine more effectual in producing perspiration and relieving the pains and oppression. Low diet, salines, and laxatives are indicated, with constant warmth to the feet, the face, and the hands. Thus the patient gets comfortably through his malady. During convalescence quinine is the best tonic.

As prophylactics, covering the face and hands, and wearing cotton wool in the ears and nostrils, are the most effectual.

Varieties of grave influenza.—Of the severer forms of influenza there is one in which the chief force of the virus seems to fall on the *heart*. After an attack, often of no unusual severity, the patient finds himself subject to palpitation, sometimes to vertigo or syncope, and the pulse is weak, compressible, and sometimes irregular. Such cases may last for months, and cause great anxiety to the patient and the physician; but they do not, in the writer's experience, lead to permanent disease of the heart, nor has he known a fatal case. Dr Sansom has recorded cases of persistent tachycardia, and in one remarkable case the pulse continued for several months at a rate of only 30 to 40 in the minute.

In another form the *abdominal* organs suffer most. There is profuse diarrhœa, sometimes after copious bilious evacuations, ending in the discharge of clear, colourless mucus. Dr Simon, of Birmingham, has recorded several such cases ('Brit. Med. Journ.,' June 13th, 1891), and the writer has seen more than one of considerable severity.

Another and perhaps the most serious form of influenza is that whic
affects the *nervous* system. With only moderate pyrexia, and little or n
cough, the patient suffers from neuralgic pains after the primary malais
has passed off. Or he becomes depressed, tearful, or hysterical, and thi
may end in either suicidal mania or confirmed melancholy, but usually i
slow and gradual recovery. Peripheral neuritis has been observed as
sequel of influenza by Dr Buzzard and other physicians. Probably som
of the cases of bradycardia and tachycardia above mentioned are th
result of parenchymatous neuritis of the vagus or sympathetic cardi
branches.

Again, the convalescent from influenza may have most troublesom
giddiness and reeling gait, so that physicians may suspect him of tabes
cerebellar tumour, and policemen of being drunk. Some of these cas
have followed attacks so little marked, that Sir Samuel Wilks and som
other experienced physicians admit a form of influenza without any pyrexi
at all ('Lancet,' 1893, ii, p. 222).

Mortality.—It seems paradoxical to say that influenza is seldom fata
and yet that it always causes a great increase of the death-rate; but th
explanation is that the number of those who fall ill is greater, beyond al
comparison, than in the case of any other epidemic disease. In London i
1847 not less than 5000 persons are said to have died of influenza in si
weeks; but then it was computed that 250,000 persons were attacked. I
Paris above one fourth of the population suffered; in Geneva not less tha

one third. Those who die of influenza itself, and not of pneumonia or other sequelæ, are usually old or debilitated subjects, like the elder Dr Babington, who died in the epidemic of 1833, at the age of seventy-seven, and had previously laboured under emphysema of the lungs.

Graves found the epidemic of 1837 very fatal among the aged, yet saw Judge Day recover at ninety-three.

Sir George Baker writes of the epidemic in 1762, "*Leviter plectebantur infantes et liberabantur facillime.*" He found it most serious in advanced age, and particularly in asthmatics. Beside aged people, children under three or four years old sometimes die of influenza, usually with bronchitis and lobular pneumonia.

Parkes states that patients with disease of the heart, and some of those who have phthisis, pass through influenza without being the worse for it; but other writers have remarked that after its subsidence the progress of phthisis is accelerated. Dr Farr pointed out, in 1847, that the mortality was much greater in those districts of England in which the death-rate was generally high than it was in healthier places.

In February, 1762, the weekly death-rate at Warsaw ran up from 30 or 40 to 150. In London in 1837 the mortality in 2347 cases was 2·3 per cent., in 1847 about 3 per cent. (Peacock). In 1889-90 it was probably lower than this throughout England, but in Vienna and for a time in Paris there was a higher death-rate from influenza, owing chiefly to subsequent pneumonia.

Anatomy.—No characteristic changes are seen in the bodies of those who have died during an attack of influenza. The air-passages show the ordinary signs of bronchitis, and there may be great œdema of the pulmonary tissue. Hepatisation of one or both lungs is frequent, and lobular pneumonia, especially in children and aged subjects. Plastic exudation into the bronchial tubes has now and again been recorded.

Ætiology and pathology.—No other disease diffuses itself so widely ver the earth's surface. Not only is it capable of existing in all inhabited egions, but in some epidemics it has ranged over every quarter of the lobe; in places presenting all kinds of soil and every variety of climate. t cannot, therefore, be due to any "telluric emanation" or miasm; nor is related to ague, for Holland, which is infected with malaria, has escaped me European epidemics of influenza.

Again, the progress of influenza from district to district occupies time. he epidemic of 1762 appeared at Warsaw in February, reached Vienna t the end of March, and Madgeburg in April; at which time it also inaded Hamburg and London. It was much more severe at Venice and at Warsaw than in England and Germany, and did not visit Paris at all. In une it was epidemic throughout Alsace,* and in July attacked the British eet in the Mediterranean. After that month no cases were known to ccur in Europe. Beside London, the towns of Manchester, Lincoln, eicester, Exeter, and Norwich were seats of the disease, apparently by onveyance from the capital. Similar epidemics of catarrh or influenza ad appeared at Norwich in 1733 and 1743. It was less common in ountry places—an argument in favour of its being infectious.

The epidemic which raged in London in 1833 is supposed by Hirsch to have been related to one which occurred in 1830 in China, and which reached Moscow later on in that year. In 1831 it spread over Russia, Poland, Germany, France, Sweden, Italy; it next appeared in the Isle of Man, and lastly in New Jersey, on the other side of the Atlantic. In 1832 it occurred chiefly in Spain and in some of the United States. In 1833 it broke out again in the north of Europe, and after extending over Russia and Germany, and passing to Denmark, it reached London in April. It was also observed at different dates of this year in Switzerland, the Tyrol, France, Italy, and Egypt.

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* These accounts were received by Sir Geo. Baker from Drs Jackwitz, Mertens, Kothen, Pringle, and Reimarus.

The epidemic of 1889-90 was first observed in Russia and spread westward over the continent, attacking as usual the large towns earliest and most severely. It spread from Central Europe southwards over Italy and Spain, and northwards to England, where the smaller country towns were visited long after London, while many out-of-the-way places escaped altogether. In America and Australia the epidemic appeared later, and travelled over the United States in a westerly direction from the Atlantic seaboard to California.

Both in England and France epidemic influenza seems only to occur as the result of extension from other countries. There is doubt whether it has ever arisen in any part of Europe. Probably its home is in some remote part of Asia, perhaps Chinese Tartary. Hence the Russian name of the "Chinese catarrh," and the English name of the "Russian influenza."

The following examples show how rapid and sweeping an epidemic may be, and how difficult it sometimes is to trace the contagion.

In 1782 Admiral Kempenfeldt's squadron sailed from Spithead on May 2nd to cruise between Brest and the Lizard. On the 29th, there having been no communication with the shore, the men who formed the crew of one of the ships were attacked with influenza, and soon afterwards so many of the sailors on the other ships that by the second week in June the whole squadron had to return to port. In the meantime another fleet, under Lord Howe, had sailed, all in perfect health, for the Dutch coast. Towards the end of the month of May the disease appeared in several of his vessels also, although there had been no intercourse with the land.

On April 3rd, 1833 (the day on which Sir Thomas Watson saw his first two cases of influenza in London), a vessel called the "Stag" was coming up the Channel, and arrived at two o'clock off Berry Head on the Devonshire coast, all on board being well. The breeze was blowing from the land, and in half an hour forty men were down with influenza; by six o'clock the number was increased to sixty, and by two o'clock on the following day to 120. The same evening a perfectly healthy regiment at Portsmouth was attacked, and by the next morning so many of the soldiers were ill that the garrison duty could not be performed.

There have been numerous instances in which the complaint has first broken out in those particular houses of a town at which travellers had recently arrived from infected places; and there have also been examples of its having spared the inmates of prisons or convents, as though their isolation had served to protect them.

"Thus," as Dr Fagge wrote in 1880, "we are brought to the conviction that, unless the cause of influenza is something of the nature of which we have no conception, it must be a living thing, which is capable of reproducing and multiplying itself when once it has been introduced into a particular district or country."

It is possible that the contagion may be derived from horses or other of the lower animals. The human disease seems to be identical with the influenza to which horses are liable ("pink-eye"), which in 1872 is said to have attacked about 16,000 horses in New York alone. During epidemics of human influenza, horses, dogs, cats, and even birds are said to suffer. But the disease in horses does not appear to spread to stablemen and grooms, and monkeys in the Zoological Gardens at Regent's Park did not share in the recent epidemics. Indeed, Klein doubts whether even horses are liable to true influenza.

The bacillus.—The probability of influenza depending on a contagium virum has been raised to a high degree by the discovery of a specific microbe by Pfeiffer in 1892, present in the mucus and saliva of patients suffering from influenza. His statements were confirmed by Kitasato in Berlin, and by Klein* in this country.

It is a rod-shaped organism, very small, immobile, and refractory to stains. When staining with carbolic fuchsin is successful it affects the two ends of the bacillus, leaving the mid-part clear, so that it simulates a diplococcus. It grows best on gelatine mixed with blood. The chemical products to which probably its toxic effects are due have not yet been ascertained, as they have been in the case of diphtheria.

Diagnosis.—From the description above given it is manifest that influenza is one of the most variable and multiform of diseases. Nor would an attempt to give pathognomonic or even distinctive characters be of much practical use.

Influenza must never be forgotten as a possible explanation of many febrile and anomalous conditions without present pyrexia. Its most characteristic and constant features are its abrupt onset, its severe pains, its short course, the deep impression made on the nervous system, particularly the cerebral cortex, the vagus, and the splanchnic areas, its slow and uncertain convalescence, and the variety and persistence of its sequelæ. Often influenza can only be certainly recognised in retrospect; often a patient's own feelings, if he has suffered from it before, prove a better diagnosis than the symptoms seen from outside.

It may, however, be useful to enumerate some of the maladies for which influenza is most often mistaken, or which in their turn, though less often, are mistaken for influenza.

The most common severe catarrhal form has been often confounded with (acute) rheumatism, with variola and other exanthems, with the comparatively, but only comparatively, rare cases of enteric fever with an abrupt onset, and in foreign countries with malarial fever and with dengue. The slighter forms of influenza are constantly regarded as "ordinary colds" until the muscular prostration or some nervous sequel shows their true nature. Bronchitis and broncho-pneumonia from influenza have often been regarded as signs of asthma or of acute tuberculosis. Pneumonia after influenza is true pneumonia, with its own characteristic bacillus present—at least in most cases. It only differs by its greater danger from cases of different origin.

The cardiac sequelæ of influenza resemble those of diphtheria, and the condition sometimes seen in spirit drinkers with peripheral neuritis and weak, rapid action of the heart. Bradycardia is more characteristic.

The abdominal form of the disease may simulate cholera, as in a case seen by Dr Goodhart: or acute muco-enteritis, or irritant poisoning, particularly by animal products.

The acute nervous prostration sometimes caused by influenza, particularly if accompanied by vomiting and severe headache, may resemble tuberculous meningitis or tumour of the brain. The fact of nervous symptoms following an attack will often justify a favourable prognosis of conditions which resemble tabes, or of cases of insanity or melancholy, which otherwise would admit of little hope.

Prognosis.—An epidemic of influenza is only directly dangerous to infants and aged persons. But an attack may be complicated by pneumonia at any age. The sequelæ, though often worse than the attack, are very rarely fatal.

Treatment.—From an historical point of view it will always be interesting to know that bleeding and the administration of antimony were recognised by universal experience to be injurious in this disease at a time when they were regarded as almost essential to the cure of pneumonia and other inflammations. In the recent epidemics quinine has been found useful, even from the commencement of the attack. Another usual practice was, after having given one purgative dose, to prescribe salines during the first day or two, and afterwards ammonia with senega or serpentary. Parkes disapproved feeding the patient with hot beef-tea; it invariably, he thought, increased the headache and the languor. If the patient is young and healthy, it may be sufficient to let him drink freely of iced milk and soda-water, thin barley-water with slices of lemon, or weak white-wine whey. Except to elderly patients stimulants should not be given during the early stages. Afterwards a good supply of food should be allowed, and at this stage bitters and mineral acids are often necessary. During the stage of pyrexia the writer has found hot baths and diaphoretic drugs give most relief, and antipyrin has proved the most useful remedy for the severe headache.

For the cardiac complications and sequelæ strychnia is the best drug, particularly injected under the skin. Afterwards, for tachycardia, digitalis and alcohol are both of the greatest value. The pneumonia which follows influenza requires stimulants from the first, and most of the neurotic sequelæ are best treated by prolonged rest, fresh air, and London porter or Dublin stout, which are more useful and less liable to misuse than spirits.

TYPHUS

Ἥξει Δωριακὸς πόλεμος καὶ λοιμὸς ἅμ' αὐτῷ.
THUCYDIDES, lib. ii, cap. 54.[*]

History, nomenclature, and geographical distribution—Incubation—Course: first week, exanthem; second week—Crisis and convalescence—Mortality and mode of death—Post-mortem appearances—Complications and sequelæ—Ætiology; contagion of typhus: predisposing causes—Diagnosis—Prognosis—Treatment.

Synonyms.—Contagious or epidemic fever; "parish infection;" putrid fever; spotted fever, morbus pulicaris, febris petechialis; ochlotic fever (ὄχλος, a crowd); typhus fever, brain fever; jail fever, ship fever, camp fever or leaguer sickness; fourteen-day fever; Irish fever; febris Hungarica; typhus exanthematicus.—*Fr.* Fièvre typhus.—*Germ.* Flecktyphus.

Definition.—A specific contagious fever, with a characteristic exanthem. running a course of about two weeks.

History.—Typhus, in Watson's classical Lectures on Medicine, in Murchison's work on Continued Fevers, and in the first edition of this textbook, was placed first in the list of diseases, and it was "by merit raised to that bad eminence," for it was the first recognised and named as a separate kind of fever; and between the disappearance of the Oriental Plague from this country after 1666, and the appearance of the cholera in 1830, typhus was the most formidable infectious fever in the United Kingdom, particularly in the great towns. During the second half of the present century it has gradually ceased to be endemic, and epidemics have become smaller and more rare.

Its place in history is next in importance to that of the true or Oriental Plague. It is the common pestilence which has accompanied and followed wars, and which kept down the population in crowded cities and in the ill-cared-for prisons of the eighteenth century.

The name now in use was first applied to a definite kind of fever by Sauvages in 1759, and was adopted by Cullen and subsequent writers. Until then it had, from the time of Hippocrates downwards, been employed, in accordance with its etymology,[†] to designate a confused state of intellect, delirium and stupor.

The plague of Athens, recorded by Thucydides, was most probably what we now call typhus. However this may be, there is no doubt of the nature of certain epidemic fevers which prevailed in Italy, France, and Hungary in the sixteenth century, and of which accounts were given by Fracastorius of Verona, and other contemporary writers. During the course of the same century occurred the first three of the famous "Black Assizes" in this country, when judges, sheriffs, and jurymen were suddenly attacked with fatal illness, which had spread from the prisoners brought up for trial. One of the older names of the disease is *jail fever*. Another is *Morbus castrensis* or *military fever*, from the ravages which it has committed among soldiers and camp followers, from the time of the Thirty Years' War and the siege of Reading in 1643 down to the Crimean campaign. Other names, again, are *spotted fever* and *brain fever*, from some of its more conspicuous symptoms. Of late years it has been universally called Typhus in this country and in France; but the Germans are obliged to term it *Typhus exanthematicus* or *Flecktyphus*, owing to their unfortunate habit of calling Enteric Fever *typhus*.

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[*] "A Dorian war shall fall,
And a great plague withal," as Hobbes translates the oracle.

[†] Τῦφος, smoke, mist, cloud, confusion and cloudiness of ideas, akin to Latin *stupeo*.

As Typhus fever or one of its synonyms, the disease included several kinds which were long held to be mere "varieties" affecting the skin, the bowels, or other organs in different cases, as Influenza or Syphilis does. We now regard them as different "species," proved so by their "breeding true." We have seen how, between 1835 and 1850, the *fièvre typhoïde* of Paris was shown to be not a local modification of the *spotted typhus* of London and Edinburgh, but a separate disease (*supra*, p. 111). About the same time Famine typhus was separated as a third specific malady, to be described in the following chapter. Oriental Plague, or true Pestilence, had always been regarded by the best observers as separate from putrid and spotted contagious fever, and is no longer spoken of as Bubonic Typhus. Thus typhus, as the term is now understood, is of strictly defined and comparatively small extent, while its importance is further restricted by its being nearly banished from civilised Europe.

Distribution.—On the Continent the disease with which we are now concerned has always been less common than in the British Isles. Both in Great Britain and in Ireland it has prevailed with great severity on repeated occasions during the last two hundred years. In the present century there have been epidemics of Typhus in 1803, in 1817-19, in 1826-28, in 1836, in 1843, in 1846-48, in 1856, and from 1861 to 1870, particularly in 1866-69, the last in London, although scattered cases have since occurred. In Dublin the last serious outbreak was in 1880-82. It must be noted, however, that in some of the earlier of these epidemics there was a large admixture of cases of Relapsing Fever, which was not known to be distinct from typhus until 1843, but can even now be recognised in the history of great fevers by its small mortality.

Typhus in the poorer districts of Edinburgh, Glasgow, Liverpool, and Dublin still occasionally occurs, as it did until recent years in London, but, as an epidemic, it no longer leaves its haunts in cities and invades the country. It is most prevalent in winter, probably from greater overcrowding and less ventilation.

On the Continent its course has been chiefly epidemic. A terrible visitation of typhus decimated the French army in the Crimea. It has been seen more or less frequently in the Baltic provinces of Russia, in Poland and in Hungary. The last epidemic in Germany had its seat in East Prussia and Silesia in 1867-8. Typhus is rare even as an occasional visitant in the south of Europe, and it appears to be unknown in Japan, in India, and the tropics generally; but it is not uncommon in Northern China.

The disease was introduced into America in 1847 by an infected immigrant ship, and in 1867 by the same means into Australia, but fortunately it has never established itself in either continent. There have, however, been outbreaks in New York (735 cases in 1881-2), Philadelphia (1883), and Montreal (1877).

Typhus has never been seen in South Africa, nor in the equatorial regions of that continent; but it occurs in Egypt every spring, and Dr Sandwich, of Cairo, has described it in the 'St. Thomas's Hospital Reports' for 1886.

The original home of typhus is unknown. Many epidemics have been traced to Russia and Tartary, and perhaps China is the most probable primitive focus of the disease.

It affects all ages, and Buchanan has shown that even young children are by no means exempt.

Typhus is unknown among animals. Mosler injected fresh blood from typhous patients into the veins of dogs without any result. Zülzer reported more successful results in rabbits.

Entrance of the contagion.—No materies morbi has yet been detected,* though there can be no doubt of its existence. It probably gains entrance in most cases by the breath, and through the stomata of the air-vesicles invades successively the lymph, the blood, and the tissues. The contagion is very sure, but readily diluted and dissipated, and probably not very persistent.

Incubation.—This is of variable length. Few cases afford an opportunity of determining it, for the disease rarely follows a single definite exposure to contagion. But Murchison collected for the 'St. Thomas's Hospital Reports' in 1871 no fewer than thirty-one instances more or less directly in point. In two of them the effect was apparently immediate: the patient was conscious of an offensive odour proceeding from a case, and was at once attacked with headache, prostration, nausea, and rigors; and all the other symptoms developed themselves in due course. In one instance the period was not more than two days; in two, only four days; in one (that of Murchison himself) exactly five days; in one not more than six days; in two not more than ten days. Similar short incubation periods have been given by other writers: thus Lebert, in 'Ziemssen's Handbuch,' mentions five to seven days; and in some cases the disease has followed exactly eight days after exposure. On the other hand, Murchison found that in most of his thirty-one cases the period was longer: in four it was exactly twelve days; in thirteen others it was within a few days of this, on one side or the other; in one patient not less than thirteen days, in another not less than fourteen days, in the third exactly fifteen days, in the fourth not less than twenty-one days. The period of incubation would seem, then, to be very variable, but most frequently somewhat under a fortnight.

During the incubation the patient appears to be well; but sometimes towards its end there is a little malaise, with headache, pains in the limbs, and loss of appetite.

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* Microbes have been described in the blood and tissues by Hallier in 1868 (a fungus), by Mott in 1873 (dumb-bell cocci), and by Hlava of Prague in 1889 (strepto-bacillus).

Course.—The onset of the disease is generally definite and usually sudden: much more often so than in enteric though less than in relapsing fever. The patient is attacked with headache, and with pains in the back and limbs. He feels chilly, and sometimes shivers; after a while he may perspire, but the chilliness presently returns, and he is glad to sit cowering over the fire. He feels weary and disinclined for exertion. He is thirsty, but appetite is completely lost. His tongue is œdematous, pale, and coated with fur, which is at first white, afterwards yellowish. There is nausea, but not often vomiting. The bowels are constipated. The urine is scanty, dense and high-coloured. He is restless, and his sleep is disturbed by painful dreams and sudden starts. So far these are only the common early symptoms of any specific fever, and in slighter degree they attend a feverish cold. Next follow more characteristic symptoms.

Every day the patient becomes more prostrate; if unable to seek relief in bed, he totters as he walks, and his hands tremble when he attempts to use them; until, on the third or the fourth day at latest, he can support himself no longer.

From the first the patient's aspect is dull, heavy, and oppressed. The eyes are injected and suffused. The face is of a dusky colour, the flush being general, and not limited to the cheeks as in enteric fever. As the disease advances, the expression becomes more and more vacant and stupid, and it is seldom that the patient himself feels anxiety about his illness. Towards the end of the first week there is generally *delirium*, especially at night. The advent of this symptom is said to be earliest in persons who have been intemperate, and in those who have been subject to mental anxiety and fatigue. In some exceptional cases it sets in during the first night, and Murchison speaks of having seen cases which were at first mistaken for mania; but, as a rule, it is only after the lapse of several days that the patient's mind begins to wander. About the same time he generally ceases to complain of headache.

The *temperature* rises rapidly in typhus. Wunderlich stated that it is generally 104°—104·9° on the first evening, and that by the fourth evening it is seldom under 104·9°, generally about 105·8°, and often higher still. Experience in England has been that the average figures are much lower than these. According to Murchison the highest temperature attained at any period of the disease is generally about 104° or 105°; it is scarcely ever as high as 106°, except in children, and it sometimes fails to reach 103°. The maximum is seldom reached before the fourth to the seventh day, and when it has been attained, the thermometer varies little during the first week. A high range of temperature in the first week generally precedes severe cerebral symptoms in the second; but a rise in the evening is of comparatively little significance, so long as the thermometer falls each morning; what is really serious is a high temperature without intermission.

The *pulse* does not usually rise above 100 during the first two or three days; afterwards it ranges, as a rule, between 100 and 120. When it is much higher the case is severe if the patient is an adult; but in children a very rapid pulse, even at the commencement of the disease, is not an evil sign. Occasionally the pulse remains below 100, and Murchison cites instances, observed by himself and others, in which it was not above 40, and sometimes down to 28, for days together. In certain cases, however, of slow radial pulse the heart's beats have been twice as frequent as the pulsations felt at the wrist. A slow pulse is not regarded as a favourable sign in typhus.

Respiration during the earlier days follows the pulse rather than the temperature.

The exanthem.—Typhus is attended by a characteristic eruption, the "mulberry rash," as it was named by Sir William Jenner.

In rare cases this is preceded by a rose-rash, which may almost be mistaken for the eruption of scarlet fever. In the museum of Guy's Hospital we have models, illustrating this remarkable roseola, which were taken from two women under the care of Dr Wilks in 1864. The parts represented are the abdomen, and the forearm with the hand; but in each case the rash is said to have covered the patient. It was of a bright crimson colour, punctate, macular, or diffused. One of the women was a nurse in the hospital, so that she was under observation from the first. The roseola in each case faded before the mulberry rash came out.

The characteristic *mulberry rash* generally appears on the fourth or the fifth day of the fever; sometimes, as in a case admitted into Guy's Hospital in 1874, it may be discovered as early as the third day, and sometimes as late as the sixth. Occasionally it is absent; but observations made with great care at the London Fever Hospital in 1864 showed that this occurred in scarcely more than 2½ per cent.—among nearly 2500 cases of all ages. The exceptions were mostly in children. When it does come out in them it is often peculiarly abundant and well marked, so as to resemble the rash of measles; and in such cases it may be seen on the face, whereas in adults it is generally confined to the trunk and limbs. One usually looks for the typhus rash on the chest and the abdomen; but Dr Buchanan says that the earliest maculæ are to be found on the back of the wrists, the borders of the axillæ, and the epigastrium. The face, though flushed, is seldom the seat of the macular rash.

It consists of more or less numerous spots, of indeterminate form, the largest three or four lines in diameter, isolated or irregularly confluent. They may at first be slightly raised, so that one can feel them with the finger; and as they may then be of a somewhat florid colour, it is sometimes difficult, when they are few and separate, to distinguish them at this stage from the rose spots of Enterica. At this time, too, they disappear on pressure. But in the course of a day or two they alter in appearance; their hue becomes darker and more dingy; they are no longer raised; and when the finger is pressed upon them they either remain unaltered or assume a yellowish tint. This change is due to the escape of blood from the vessels into the substance of the cutis; in other words, the maculæ have become petechial. They remain visible on the dead body if the case should end fatally while they are still present. In many instances they are from the very first of a livid or purple colour, and do not fade on pressure.

Within forty-eight hours from its first appearance the mulberry rash is complete. During this time fresh spots may come out, but they are added to the old ones. There is not, as in enteric fever, a succession of crops of maculæ, one set appearing while another is fading away. Murchison satisfied himself on this point in a large number of cases, surrounding every spot with a circle of ink so as to identify it. In addition to the distinct maculæ, there is also a faint, irregular, dusky red mottling, which looks as if it were more deeply seated, and has therefore been inaccurately called a "subcuticular rash." After the first day or two no fresh spots appear, but the maculæ persist until the end of the fever.

It is important to notice that the severity of a case of typhus is generally directly proportionate to the amount of eruption, its depth of colour, and the rapidity with which it becomes livid or petechial. Murchison adds that the exceptional cases in which no rash appears have generally a mild course; but according to Lebert they are often severe and sometimes fatal.

Subsequent course.—During the second week of the fever, in which the exanthem is fully out, the symptoms become more and more severe. The patient is sleepless, and usually passes into a state of continuous *delirium*. Sometimes he is noisy and violent, shouting, talking incessantly, singing, and struggling to get out of bed. In this condition a patient may throw himself out of the window, and some years ago a man was brought into Guy's Hospital for a suicidal wound of the neck inflicted in the course of typhus. After two or three days the delirium becomes quiet and muttering; and often it is so from the first. Sometimes excitement comes on as night approaches, while the rest of the day is passed in a state of stupor and prostration. By the middle of the week there is generally complete unconsciousness. When loudly spoken to, the patient perhaps opens his eyes and stares vacantly; or, if told to put out his tongue, he may separate his jaws, and leave them gaping.

Murchison, who himself went through two attacks of typhus, tells us that the imagination is far from inactive during the delirium of typhus. He took a great dislike to a nurse and to a valued friend; and because they once tied him down in bed he fancied that they intended to murder him, that they were shutting him up in a dungeon, that they followed him to India, Burmah, and other countries which he had visited in former years, and to which he had escaped. Dr Gueneau de Mussy, who caught typhus in Dublin in 1843, afterwards remembered imagining that he was tied down in bed, and gradually consumed by spontaneous combustion. Another fancy was that he saw the front of a particular house in Paris in a state of phosphorescence, and a child suspended by the neck from a window. He also believed that he saw one of his friends killed in the street; and so strong was the impression that even during convalescence he continued to feel concern for his loss.

During the second week of typhus *deafness* is very commonly present; indeed, it begins about the fifth day. It sometimes affects one ear, sometimes both. Many cases do well in which deafness has been complete; and from the time of Fracastorius there has been a tradition that it is a favourable sign. For this belief, however, there do not appear to be any good grounds; deafness is certainly present in many cases which end fatally.

The *pupils*, in the advanced stages of typhus, are contracted and insensible to light—as minute as pinholes, according to Graves. Jenner first laid stress on this myosis as a distinction between typhus and enteric fever; and Murchison says that neither during active delirium nor in profound stupor has he seen dilated and insensible pupils in typhus.

The muscular *prostration*, during the second week of typhus, becomes extreme. The patient sinks down in bed, and lies on his back, unable to raise himself, or even to turn on either side. If the clothes are turned down to look for the maculæ, he takes no notice. One of the shrewd remarks of the last resident apothecary of Guy's Hospital, Mr Stocker, was that to find a fever patient lying on his side was a good sign. The fæces are passed involuntarily. The urine dribbles away incessantly, so that unless it can be caught in a proper receptacle it soaks the sheets and produces great irritation of the skin. But before it begins to run off it may fully distend the bladder, the muscular coat of this organ being paralysed as well as the sphincter. One must, therefore, never omit to examine the hypogastric region at every visit, even if the nurse tells us that the urine is passed in abundance. Other results of muscular weakness are loss of power to speak, to protrude the tongue, and sometimes even to close the eyelids, so that the cornea may slough.

At this stage of the disease *tremor of the muscles* is almost constantly present. The whole body may be in a state of agitation, especially in old people and in those who have been exhausted by work or by intemperance; indeed, in drunkards a state of delirium tremens often seems to be added to the typhous symptoms. Sometimes the tremor is limited to the hands and tongue. Murchison speaks of having sometimes seen nystagmus, or choreiform spasms of the limbs. Much more frequent are those jerking movements of the muscles of the forearms which are commonly called *subsultus tendinum*. Or there may be twitchings of the face. Jenner saw two cases in which the face acquired a peculiar expression from a spasmodic action of the inferior recti muscles of the eyeballs in association with the levatores palpebrarum; in each instance the movements were excited when either of the arms was suddenly raised. Another variety of spasm is picking or fumbling at the bedclothes—a bad sign, for which we have the pedantic names "floccitatio" and "carphology."

The mulberry *rash* generally remains visible until the fall of temperature. The maculæ may even become darker and more distinct. But the faint general mottling fades more quickly, and in mild cases, where this is the only eruption, nothing of it may be left when the fever has still several days to run.

The *temperature* during the second week is, as a rule, rather lower than in the latter part of the first week. This slight fall between the seventh and the tenth day is of good omen, and its absence unfavourable. Henceforth, and up to the time of the crisis, there are usually slight recurring remissions of one or two degrees. A decided rise during the second week indicates the supervention of some complication, generally in the lungs.

Shortly before death the temperature often rises very high. Dr Moore records a case in which it reached 109·1° Fahr. on the nineteenth day. Another, met with by Dr A. W. Foote, was 107·2°; and the same figure (43° C. and 109·4° Fahr.) was recorded by Wunderlich and Murchison. But, except just before death, hyperpyrexia appears to be very rare in typhus.

The rate of the *pulse* varies little from day to day, but its general tendency is to become more rapid. Its volume and force decline until in severe cases it is so small and feeble as to be imperceptible. Sometimes it is dicrotic, but Lebert remarks that this is not nearly so common as in enteric fever. Irregularity of the pulse is more frequent, and sometimes there are intermissions. Stokes pointed out an important sign of the chief danger of the disease, namely, diminution or loss of the heart's impulse, with disappearance of the first sound. For several days, even when recovery is to take place, it is often impossible to feel the heart beating, and with the stethoscope only the second sound can be heard. In other cases a systolic bruit is developed, no doubt from temporary dilatation of the mitral or tricuspid orifice.

The *breathing* in the second week of the disease is almost always hurried, being at the rate of thirty or forty in the minute. Some degree of hypostatic congestion of the lungs is a constant symptom, not a complication. It often begins about the middle and sometimes early in the second week, and is indicated by diminution of resonance over the bases behind. The respiratory murmur is feeble, and masked by the coarse râles which gradually spread over the greater part of the chest. Bronchial catarrh is always associated with this condition, and the patient may spit up considerable quantities of frothy tenacious secretion, mixed perhaps with streaks of blood. But sometimes cough and expectoration are entirely absent.

The *tongue*, in mild cases, may remain moist, though furred, throughout the whole course of the fever. But usually during the second week it is dry, rough, and brown. When the disease is very severe it becomes contracted into a ball ("like a parrot's tongue"), and covered with a dark brown or black crust; this crust may be irregularly cracked; but the fissured condition of the tongue itself, which is so commonly seen in enteric fever, is rare in typhus. The lips and teeth are covered with dark, dirty crusts, called *sordes*. They consist of an accumulation of epithelium, which becomes dark from desiccation or black from blood or from the remains of beef-tea and other food. Increased care and skill in nursing have in this, as in many other points, deprived the clinical pictures drawn by earlier writers of some of their most striking features. An entire loss of appetite lasts to the end of the disease; and sometimes the patient refuses to take nourishment, nor does he in the latter period of the fever appear to suffer from thirst. Occasionally vomiting is a persistent and troublesome symptom, but this is exceptional. The abdomen is sometimes flat or even concave, and tympanites is very rare. As a rule there is *constipation*, but looseness of the bowels is not infrequent. Murchison spoke of diarrhœa as occurring in about 5 or 10 per cent. of all cases of typhus. But in 1866 Buchanan said that it had been seen in at least one third of the cases which had come under observation at the London Fever Hospital during some years; he was inclined to refer its greater frequency in the experience of some physicians to the larger amounts of liquid food forced upon the stomachs of their patients.

The *urine* generally remains dark in colour during the second week of typhus, but sometimes it is pale and even alkaline. The amount of urea voided is increased compared with the amount of nitrogenous food taken. At this period of the disease the *chlorides* fail to be excreted by the kidneys, even when the patient is made to take large quantities of common salt. The urine sometimes contains no chlorides at all, but Buchanan says that usually two or three grains are passed in the twenty-four hours, just enough to produce an opalescence on the addition of nitrate of silver. *Albuminuria* is common in fevers generally, and in typhus appears to be present in more than half the cases. It may, however, when associated with blood and tube-casts, indicate acute febrile nephritis.

Crisis.—Such is the condition of a patient suffering under typhus until about the fourteenth day, when in favourable cases a marvellous change takes place. He falls into a sound and quiet sleep, from which he awakes a few hours later, rational, refreshed, and cool. At the same time the thermometer shows a rapid fall to normal or even lower—a difference generally of from 4° to 6° Fahr. The fall may be complete within twelve hours, but according to Lebert often takes thirty-six hours, and sometimes two or three days.

Murchison says that in nearly one half of the cases of typhus the date of the crisis is the thirteenth or the fourteenth day; in more than three fourths it is from the thirteenth to the sixteenth. Lebert gives a much larger proportion of cases as subsiding between the tenth and the twelfth days, some even between the sixth and the ninth. These short cases mostly occur in children.

The crisis is sometimes accompanied by perspiration, or by diarrhœa, or by the excretion of urine depositing lithates in abundance; but there is no reason to suppose that such symptoms are essential to the defervescence, as was formerly supposed. Afterwards the temperature generally remains normal, except that for a night or two there may be a slight evening rise.

Convalescence advances rapidly. The tongue becomes clean and moist, and the appetite is ravenous. Within three or four weeks the patient has recovered his bodily strength, and is able to return to work.

A *relapse* of typhus is exceedingly rare. Jenner, A. P. Stewart, and Murchison never saw a case. An instance of it was recorded in 1869 by Ebstein, the interval being twenty-five days. At the London Fever Hospital no such case occurred among 18,268 cases during twenty-three years after 1855. In that year Buchanan had a nurse under his care who, after recovering from an attack of the disease, was taken ill a week later, and went through it a second time: on each occasion there was a mulberry rash.

Mode of death.—Many cases of typhus do not run on long enough to exhibit a crisis. The mortality from the disease at the London Fever Hospital has been 15 per cent., but Murchison estimated that, if slight cases and those occurring in children be taken into account, it is about 10 per cent. It differs, however, slightly in different years, and it is said to be higher during an epidemic than at other times. Among soldiers in camp, or in a besieged city, the death-rate has sometimes been enormous. In the Crimea one half of the French troops who were attacked is believed to have perished; and still more terrible instances are on record. In certain epidemics death has been known to occur on the second or third day, or even after a few hours' illness. Such cases have been described as a special variety of the disease—*typhus siderans*, and the patient was described as *sideratus*, planet-struck. Murchison speaks of having seen several cases end fatally on the sixth or eighth day, usually from pulmonary congestion. But, as a rule, a patient does not die of typhus until towards the end of the second week.

The exact mode of death varies. Sometimes it is with "typhoid" symptoms and coma; sometimes it is through the lungs with cyanosis: sometimes it is with failure of the heart, the pulse being imperceptible, and the skin cold, livid, and bathed in a profuse sweat. In some of the last-mentioned cases the patient lies for a day or more in a peculiar state, to which Jenner has appropriated the name *coma vigil.** He has his eyes wide open, so that he might be supposed to be awake, but he is absolutely insensible to all that goes on about him, and his face is devoid of expression. Occasionally death is preceded by a rapid elevation of temperature, as in a case at Guy's Hospital in 1873, when it reached 108·7°.

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* As Dr Moore remarks, the same term was formerly applied to a different and far less important symptom by Chomel.

Morbid anatomy.—The appearances observed in the bodies of those who have died from typhus are not characteristic of the disease, but are effects of the febrile state through which the patient has passed, and are found in cases of enteric fever, pneumonia, erysipelas, and pyæmia. The *rigor mortis* is brief and incomplete, putrefaction takes place early, and there is much cadaveric discoloration of the skin. The *blood* is fluid and dark-coloured, and it stains the lining membrane of the heart and of the great vessels. There is often but little emaciation, yet the *muscles* are soft and friable, and when death has occurred at a late period of the fever their fibres are granular or waxy-looking under the microscope. A similar change is found in the substance of the *heart*, and this cardiac degeneration has no doubt much to do with the fatal issue. The *spleen* is but little enlarged, weighing on an average seven ounces, though sometimes as much as fourteen; its tissue is very soft, and may be reduced to a mere pulp, which escapes when the capsule is divided. Jacquot is said to have observed a case in which instant death resulted from rupture of the spleen. The *liver* is soft and hyperæmic: in an advanced stage of the disease it is often fatty. The *lungs* are in a state of more or less extensive hypostatic congestion. The affected parts are bulky, of a dark red or purple colour, and soft; serous fluid oozes abundantly from their cut surface, and from the bronchi.

There is an entire absence of those intestinal lesions which we have described above as characteristic of enteric fever, and the petechiæ of the exanthem may usually be seen on the body.

Other changes which have been noted are independent of the disease. They either belong to a certain period of life, or denote intemperate habits, which predispose to death in typhus. Thus the brain has often been found atrophied, with an excess of fluid in its ventricles and upon its surface.

Occasionally the *kidneys* show signs of Bright's disease, and the nephritis is sometimes recent, and secondary to the fever. Murchison speaks of having found the kidneys weigh nineteen, twenty, and even twenty-three and a half ounces.

Complications and *sequelæ* of typhus are neither numerous nor frequent. Jaundice is extremely rare. Murchison met with only fifteen cases, and in one of them the liver was in a state of acute yellow atrophy, while in another leucine and tyrosine were found in the urine. One well-marked instance of jaundice occurred at Guy's Hospital in 1869. The patient, a man aged forty-nine, died on the fourteenth day of the fever.

Retention of urine may occur, and if not relieved, or relieved without care, may easily lead to cystitis. The bladder should always be felt for.

Lobar pneumonia is not uncommon. Sometimes, when the patient appears to die of the severity of the disease, instead of there being merely hypostatic congestion of the dependent parts of the lungs, the lower or the upper lobe is found to be hepatised. Of about forty consecutive fatal cases of typhus examined at Guy's Hospital by Dr Fagge, six had well-marked pneumonia. In one instance an attack of pleurisy occurred a week after the subsidence of the fever; and after death the left side of the chest was found to contain several pints of fluid with flakes of lymph. Pneumonia is much more rare as a sequel than as a complication. Murchison repeatedly saw gangrene of the lungs after typhus, and gangrene of the extremities has also been recorded.

The occurrence of a convulsion is an unfavourable symptom, but recovery took place in twelve among one hundred and thirty-two cases in which this complication occurred at the London Fever Hospital between 1862 and 1869. It is towards the end of the second week that convulsions are most often observed. Acute meningitis may occur, but it is an exceedingly rare complication. Mania sometimes sets in during convalescence, and may compel the removal of the patient to an asylum; but in the long run it seems always to end favourably. Peripheral neuritis may follow typhus, and is probably the cause of paraplegia as a sequel of the disease.

Hemiplegia, with or without aphasia, is an occasional sequel, but in all likelihood it depends upon embolism of one of the cerebral arteries by a fragment of thrombus derived from the left auricle or ventricle; for clotting of blood in their recesses is favoured by the feeble circulation and temporary dilatation of their walls. Murchison once saw ulcerative endocarditis in a case of typhus, with large vegetations and infarction of the spleen. Embolism of the arteries of the limbs is doubtless the cause of gangrene of the feet, which has now and then occurred as a sequel. Bedsores ought hardly to arise in this disease, since the state of coma is of comparatively short duration. Thrombosis of the femoral veins is not often seen after typhus. Inflammatory swellings which have been compared with the *buboes* of plague occasionally form in the parotid and submaxillary regions, and go on rapidly to suppuration.

When a pregnant woman is attacked with typhus she often passes through the disease to her full time, but sometimes a miscarriage occurs between the tenth and the fourteenth days. As a rule she afterwards does well, and the child, if not too immature, is generally saved.

Varieties of typhus.—The course of this disease is, as a rule, remarkably uniform; but it is the custom to distinguish mild cases, which last only ten days—mostly in children; and cases are recorded in which the crisis has occurred as early as the eighth day. It is in these patients that the rash is ill-developed, or sometimes, it is said, entirely absent. Such cases have been distinguished as *typhus mitior* or *mitissimus*, and correspond to those which Dr Collie, in his 'Monograph on Fevers' (1887), describes as "Typhus simplex." Those which last fourteen days and show the symptoms above described, he calls "Typhus gravior;" and cases with extreme prostration and stupor without much delirium, which usually end in coma about the eighth day, "Typhus gravissimus," corresponding more or less with the Typhus siderans above mentioned.

Coincidence with other specific fevers.—Typhus has been recorded as occurring along with scarlatina by Peacock, with variola by Murchison, with diphtheria by Gairdner, and with enteric fever by Moore. The only other fever which has been often observed as a complication is erysipelas.

Ætiology.—That typhus is directly transferable from the patient to others is established by the clearest possible evidence. Instances are commonly observed in every hospital into which cases of typhus are admitted: if into general wards, those who are already patients may be attacked; if into special wards, the medical attendants and nurses. When cases are left in private houses or lodgings, the disease passes not only to inmates, but also to occasional visitors like doctors and clergymen. On the other hand, the removal of a single case of typhus from the building in which it arose is often effectual in preventing other occupants from taking it.

The diffusion of the disease can often be traced from point to point in a town or in a district. Thus Alison relates how the son of a shoemaker in Edinburgh lay ill with typhus in a room in which his father and two apprentices were at work. Afterwards both of the apprentices were attacked in their own homes, apart from one another, and at considerable distances from the workshop; and there followed seven cases of typhus in one house, and seven in the other.

An epidemic which occurred at Carlisle in 1781 was found by Dr Heysham to have started from a particular house in Richard Gate; one of the persons affected there was a weaver, who on his recovery communicated the disease to his fellow-weavers in a large workshop, and by them it was spread all over the town.

The contagion of typhus is probably exhaled both by the skin and the lungs, and it may perhaps cause the offensive odour which is so perceptible close to severe cases. This odour has been compared to the smelll of rotten straw, or to that of mice. Murchison regarded it as *sui generis;* he says that he has known nurses in the London Fever Hospital distinguish typhus from other fevers by it alone. It is given off chiefly during the second week, and there is reason to believe that the contagion is then most powerful. The late Dr Perry, of Glasgow, maintained that the disease was not contagious before the ninth day; for he found that at the Glasgow Fever Hospital patients who happened to have been sent in for erysipelas, pneumonia, bronchitis, or other inflammatory affections escaped typhus so long as they remained in the wards for acute cases, but caught it when they were transferred into a convalescent ward; and he adopted the plan of keeping such patients in the fever wards until they were sufficiently well to go to their homes, with the result that during several months none of them were attacked. Murchison, however, although he admits that typhus is most contagious after the first week, thinks that the spreading of the disease in convalescent wards is due to the patients coming more closely into contact there, and especially to their wearing their own clothes, saturated with the fever poison before admission. He believes that the human body soon ceases to give off the poison after the subsidence of the fever.

Typhus is not nearly so apt as the contagious exanthemata to be propagated by means of inanimate objects, or of human beings themselves unaffected by it; but occasionally clothes and bedding may become vehicles (*fomites*) for the transmission of typhus. Laundry-women are liable to contract the disease without direct communication with the sick. Barker and Cheyne, in their account of the epidemic of 1800, relate that a child discharged from a fever hospital took to another institution a bundle of clothes which had not been disinfected; a woman who opened it perceived an exceedingly disagreeable odour, and in a few minutes became ill with what proved to be the beginning of the fever.*

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* Haller, of Vienna, thought that dark-coloured materials were more apt to absorb the poison than light-coloured ones. I well remember I used to notice, when demonstrator of

Occasionally typhus seems to be conveyed by persons not themselves affected, or perhaps by their clothes. Murchison relates that in January, 1867, a patient in a surgical ward at the Middlesex Hospital was attacked after she had been there for four and a half months; and she had been daily visited by a nurse who was in close attendance on a patient with typhus downstairs. In 1861 an Egyptian vessel, the "Shiah-Jehaad," introduced typhus into Liverpool, where thirty-one persons caught it. But although Dr Duncan, on the testimony of the surgeons who attended the men on board this ship, thought that they suffered from no other disease than dysentery ('Trans. Epid. Soc.,' 1861), Dr Parkes came to the conclusion that typhus had really been prevalent among them before they arrived in port ('Army Med. Rep.,' vol. ii).*

Very few certain cases have been recorded of catching typhus by contact with the bodies of those who have died of it; but Murchison, when he was attacked in Edinburgh, had been dissecting in a close room, in which there were many such bodies, and he had never entered the wards of the infirmary, nor seen a case of the disease. On the other hand, at St. Bartholomew's Hospital, in 1838-9, the dissecting room received seventeen bodies dead of typhus; but among six students of the hospital who alone took it, four had not dissected at all, and the other two, who had dissected, had been also exposed to contagion in the wards.

The poison of typhus is easily rendered inert by free dilution with air. Some writers have stated that there is greater risk of the disease spreading from the lower to the upper stories of a house or of a hospital than in the reverse direction; and this, if true, would show that the upward currents of air that exist in all inhabited buildings are capable of carrying the infection with them. But experience has abundantly proved that it never passes from one house to another through the atmosphere. When the London Fever Hospital was one of a row of houses in Gray's Inn Lane, no case of typhus arose in the others; and afterwards, when it occupied its second site at King's Cross, on the same plot of ground as the Smallpox Hospital, and but a few yards off, Dr Tweedie was able to state that during eight years not one of the officials of the latter institution contracted typhus. Murchison believed that if a patient with this disease is placed in a large, well-ventilated apartment, the attendants incur little risk, and the other residents in the same house none whatever. The late Mr Stocker always maintained that one or two cases of typhus in a large ward would not spread.

Predisposing causes.—We need not now argue that typhus is always due to contagion from a previous case, and is never generated *de novo.*

It is, however, instructive to remember that the spontaneous origin of this, as of other contagious fevers, was almost universally believed up to recent times. Indeed, no less an authority than Virchow supported the opinion that the poison of typhus can be generated by the concurrence of filth, starvation, crowding, and misery; the same view was maintained by

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anatomy, that the dissecting-room smell adhered to me more strongly when I wore dark clothes.—C. H. F.

But the 60th aphorism of Dr Keil, of Northampton, is that "black cloaths *cæteris paribus* draw the least moisture of any" ('Medicina Statica,' 1720).—P.-S.

* She left Alexandria in November, 1860, and, after calling at Malta, arrived at Liverpool on February 16th, 1861. But, according to Dr Parkes, some of the men may have brought the poison with them when they embarked at Alexandria; they numbered 476, and included not only Arabs, but also Nubians and Abyssinians.

the late Dr Hudson, of Dublin, and was strenuously argued by Murchison. But all the facts adduced may be explained if we admit that impairment of health lowers resistance to the typhus poison, and bear in mind that overcrowding is an obvious help to the spread of contagion.

Of the supposed instances of the spontaneous generation of typhus collected by Dr Murchison, the most important, in consequence of the care and pains which he devoted to its investigation, is perhaps the group of seven cases which arose in Meridian Place, Bermondsey, in March, 1859—a time when the disease was at least very uncommon in London, for during ten and a half months previously only two examples had been seen in the Fever Hospital. But the real bearing of this and similar observations is that those who were attacked by the disease had been huddled together in narrow, ill-ventilated dwellings. They were often destitute and famished as well; but that this is not so essential to the development of the disease as overcrowding, seems to be shown by the fact that at Dundee, in 1865, an epidemic of typhus was brought about by the inhabitants of the surrounding country flocking into the town in consequence of work being abundant and wages good. It would be impossible to find a better illustration of the conditions under which typhus is a fatal epidemic than was afforded by the "Shiah-Jehaad;" the men on board were crowded together in the most shocking manner; some of them were in a state of starvation; the filth and stench between decks were abominable; and, to crown all, the hatches had been battened down on account of bad weather.

There does not seem to be anything of the individual predisposition to or immunity from the infection of typhus which we observed in scarlatina and some other specific fevers. Every one is capable of taking this disease except those who have already passed through it, for the immunity conferred by typhus is almost absolute. At the London and Glasgow Fever Hospitals all the nurses who have not had the disease before contract typhus within three or four months after entering upon their duties. In 1833 Dr Tweedie stated that with one exception every physician connected with the London Fever Hospital had been attacked with typhus. Similar experience is recorded from the Fever Hospitals of Dublin, Cork, and Edinburgh.

Certain accidental conditions appear to favour contagion; one is *intemperance*. Murchison says he has known several instances of persons exposed for months to the poison of typhus in its most concentrated form, who were not attacked until immediately after a debauch. Chronic alcoholic poisoning probably acts in a similar way: it was once noted that more than one half of the patients admitted with typhus into the Edinburgh and Glasgow Infirmaries had led intemperate lives—but this perhaps was not conclusive evidence.

Other favouring circumstances are excessive *bodily fatigue*, *mental anxiety*, and *want of sleep*. In support of the popular doctrine that a dread of typhus increases the risk of taking it, Murchison cites the case of an Edinburgh medical student who so feared it that he could hardly be induced to enter a ward in which there were any cases; he was one of the first students to be attacked during the epidemic of 1847. Convalescents from other complaints are believed to be predisposed to typhus, and in armies it has been frequently observed to follow scurvy. There is no evidence that typhus is less likely to occur in phthisical persons than in others, but, as Tweedie long ago remarked, butchers appear to have certain exemption from typhus. Butchers admitted into the London Fever Hospital for typhus have generally been out of employment and ill-fed.

Poverty and *starvation* are certainly predisposing causes. No less than 95·6 per cent. of the 18,000 typhus cases observed at the Fever Hospital during twenty-three years had been inmates of hospitals or dependent on parochial relief, and many of them had been on the verge of starvation for weeks or months. Epidemics in Ireland and on the Continent have repeatedly followed the failure of crops and the wide-spread destitution consequent; so that although relapsing fever is the true "famine fever," that name was formerly given to typhus, and not without warrant.

Other so-called predisposing causes probably act only by facilitating contagion. Thus epidemics occur rather in the winter than in the summer, because the poor for the sake of warmth block up every hole by which air can enter their dwellings during the cold season. But in some years more cases of typhus have occurred in July or September than in January. So far as is known, the variations of temperature which occur in temperate climates have no direct influence upon the prevalence of the disease.

We have seen that typhus is absent or exceedingly rare in tropical countries, although it was believed by some observers to occur in the gaols of India. But this exemption depends on the climate only, for in London negroes and East Indians have been admitted into the Fever Hospital with characteristic symptoms of typhus.

It is worthy of notice that a considerable proportion of the patients themselves attribute the disease to their having "caught cold" or got wet. In 1856 a young man was taken into Guy's Hospital who gave as the history of his illness that he went for a long walk on damp ground, and felt that he took cold; two days later he had headache and fever, and the typhus eruption followed in due course. No doubt the infection had already been received when the chill was felt. Such facts are only worth recording because they teach caution in accepting similar statements with regard to pneumonia, rheumatism, and other acute diseases.

Age and sex.—Typhus may occur in persons at all ages, from one month to eighty-four years old. Buchanan showed that children are in no degree exempt, though in them the disease is comparatively mild. The quinquennial period at which the disease is most common is from fifteen to wenty; one half of the cases admitted into the London Fever Hospital have been in patients between ten and thirty.

The proportion of males to females among typhous patients scarcely differs from that in the population generally.

Protection.—A second attack of typhus is as rare as one of smallpox; ar more so than a second attack of measles or scarlet fever. It does, however, sometimes occur, and there was a notable instance of it in the case of Murchison himself, the interval between the attacks being ten years. He knew of two other physicians who contracted this form of fever twice, and n one of them it proved fatal on the second occasion. The late Sir George Buchanan had typhus twice.

A remarkable fact, of which Murchison saw six examples, is that, during n epidemic, a person exposed to the infection may have what appears to be n abortive attack of typhus, with fever, dry tongue, and even slight delirium, but no distinct rash; may recover, sometimes after exactly four-attended with the characteristic eruption. Such a "*typhisation à petite dose*" seems to afford no protection whatever.

Diagnosis.—This is not difficult when one has an opportunity of watching a case throughout its whole course; but there is sometimes great difficulty in forming an opinion upon a single visit.

We suspect typhus when there has been exposure to the specific contagion, or when at least cases have already occurred in the neighbourhood. But at first it may be impossible to decide whether, supposing both are *prevailing*, the case is one of typhus or measles, or even to exclude enteric fever, some cases of which begin exactly in the same way. As a rule all uncertainty is removed by the development of the eruption; but as a rare exception this may be absent. The locality of the rash of measles and its later appearance are more important than Lebert's point of the harassing cough which belongs to measles, whereas the bronchitis of typhus comes later, and is seldom troublesome. Certain cases of *purpura* might be mistaken for typhus; but the eruption is petechial from the first. Strange to say, the copaiba rash has been mistaken for typhus, as in an instance quoted by Dr Hudson.

At the end of the first week the diagnosis of typhus from *Enteric fever* is rarely a matter of serious difficulty, even in children. Apart from the evidence of dates and the dissimilar exanthems, the one is epidemic and rapid in its evolution, while the other is sporadic and gradual in its onset and course. Fulness of the abdomen and dilatation of the pupils point to enterica; a flat belly and contracted pupils to typhus or meningitis.

Pneumonia, although formerly often mistaken for typhus, and although Murchison says he has seen many instances sent to the London Fever Hospital, ought to be always recognised by its physical signs, even when it occurs as a complication of typhus.

The distinction between *meningitis* and typhus is one of far greater difficulty. Among the symptoms which point to the former disease are an anxious and pained expression of face, intense headache, especially when concurrent with delirium (for in fever pain almost always ceases before delirium sets in), the presence of convulsions early in the case, the repeated occurrence of vomiting, and the presence of optic neuritis, which is not observed in typhus. The patient, moreover, is irritable rather than apathetic, and resents instead of enduring examination. Epidemic cerebrospinal meningitis is sometimes accompanied by an exanthem, and has probably been mistaken for typhus.

Further questions arise in the diagnosis of cases which come under observation at a time when the patient is comatose, with "typhoid" symptoms fully developed. Pyæmia may then be mistaken for typhus, or acute atrophy of the liver, or, in patients advanced in years, bronchitis, with a dry brown tongue and stupor.

A similar "typhoid" condition, which is perhaps most apt to be mistaken for the later stage of typhus is *uræmia*. Patients have often been sent to the London Fever Hospital whose symptoms were all of renal origin; and at Guy's Hospital in former years the doubts with regard to such cases have not always been entirely cleared up. Conversely, in 1865, a man who was in a surgical ward for stricture became feverish and drowsy, and his illness was attributed to ascending nephritis, until a mulberry rash was discovered upon him; he died, and the kidneys were found to be healthy. It must be borne in mind that albuminuria is common in typhus, and the existence of chronic renal disease, even when established by *post-mortem* examination, affords no proof that typhus was not also present. The absence of a rash and of decided pyrexia are probably the most useful points of distinction.

Prognosis.—In making a forecast at the beginning of a case of typhus, the most important consideration is the *age* of the patient. The older he is, the greater is the danger. In children this disease seems never to be fatal except by some complication. Murchison found that at the London Fever Hospital the average typhous death-rate for all ages was from 15 to 19 per cent.; at the same hospital after his time it was 18·6 per cent. (1871-80), at Stockwell 22·5, and at Homerton 19·7 during the same ten years (Collie); in Dublin (1882) only 10·5. The death-rate in persons above thirty was 35·39 per cent., in those above forty 43·48 per cent., in those above fifty 53·87 per cent., in those above sixty 67·04 per cent. Drs Washbourn and Goodall give the following figures from the hospitals under the Metropolitan Asylums Board for London, 1871-94:—Total mortality 437, out of 2166 cases, or 20·2 per cent. Mortality under fifteen, 2·2; fifteen to twenty, 7·8; and above twenty, for successive periods of five years, omitting decimals, 20, 23, 29, 35, 48, 44, 60, 75; and 71 above sixty. Dr Moore records the recovery of an Irishman from typhus at the age of 104!

Secondly, typhus is likely to terminate fatally in persons who have been intemperate, who have been exhausted by fatigue of body or mind, or who have been suffering from want of food. The greater frequency with which these several conditions occur among adult males, as compared with females, is probably the reason why the mortality is higher in men, 22·4 to 18·3 (Metropolitan Asylums Board); among children between five and fifteen it is lower in boys than in girls. Patients who have gone on struggling against the disease during the first few days often become rapidly prostrate. The sooner they are put to bed the better their chance.

During the progress of a case, the danger may be measured by the intensity of the cerebral disturbance and by the degree of prostration. Moreover, the earlier the date at which severe symptoms are present the worse is the prospect. Sleeplessness continued throughout several days is of evil omen, and so is the occurrence of convulsions. A very abundant rash is usually a bad sign, especially if the spots are dark and rapidly become petechial. A pulse over 120 is always a serious matter in an adult; and when it exceeds 150, death is almost certain. Relaxation of the sphincters before the tenth day is unfavourable; but towards the end of the second week it is not uncommon in severe cases, which may nevertheless do well. Great lividity of the face and limbs and coldness of the distal parts of the body with profuse sweating are among the most threatening symptoms. A well-marked "typhoid" state, however, with dry brown tongue, sordes and subsultus, is observed in many patients who recover. Indeed, even in what appear to be the worst cases, one must never give up hope until the last.

Treatment.—The progress of typhus cannot be arrested, nor can it be specially treated; but much may be done to help a patient's recovery, and many of the most distressing symptoms may be alleviated or removed.

tions for the reception of fever cases

dates from the commencement of the present century, but so recently as 1842 many London physicians, among whom was Dr Bright, were in favour of mixing such cases with others in general wards, rather than of setting apart special wards for them, or of collecting them in buildings devoted to that particular purpose. However, so far as typhus is concerned (and the same may be said of relapsing fever) this was certainly a mistake. For it has now been proved by experience that if a sufficient cubic space of 2000 feet be allowed, and if ventilation be well attended to, the death-rate is not greater among typhus cases accumulated in the same ward than when they are scattered. Moreover, in every hospital in London, where such cases have been placed in general wards there have been terrible examples of the spread of fever to other patients. To cite but a single instance: in 1862 one or two cases of typhus were admitted into one of the large medical wards in Guy's Hospital; seven other patients took the disease, and five of them died.

Every large town ought to have a fever hospital, capable of enlargement during an epidemic by the erection of temporary buildings.

A patient suffering from typhus should be placed upon a spring bed, or upon a hair mattress, with not many bedclothes. The temperature of the ward or room should not exceed 60°, and may be allowed to fall lower with the window open, while the pyrexia continues. After the first day he should not be allowed to get up even to pass his excreta. It is important that his nurse should be strong enough to raise him when necessary, and instructed enough not to lift him into a sitting posture. From time to time he should be turned over to one side or the other, and kept from rolling back by pillows.

Food.—A nourishing diet is of the highest importance. Dr Graves of Dublin desired as his own future epitaph the testimony "He fed fevers." Beside milk, eggs, and beef-tea, the patient may have broth made of mutton, veal, or chicken, meat juices and extracts, jellies, custards, blancmange of isinglass or ground rice, white-wine whey, &c.; and vermicelli may be given in the beef-tea, small pieces of bread or toast in the broth or in the milk.

For beverages we have the choice of barley-water, toast-and-water, lemonade, tamarind-water, currant-water, effervescing drinks, and cold weak tea. Before many days have passed, however, the patient acquires a distaste for everything but cold water. He should drink often, in order to keep up free excretion by the kidneys.

Food must be pressed upon him and administered at regular intervals of from one to three hours; to give it more often than this is probably injurious rather than useful. It is important that his strength should not be allowed to run down at night, or during the early morning, when the risk of exhaustion is always greatest. When, as is sometimes the case, he obstinately refuses food, it must either be introduced into the stomach by means of a tube passed through the nostril, or, better, by closing one nostril and pouring the liquid nourishment through the other by means of a short funnel; an aural speculum answers the purpose well. If necessary, nutrient enemata or suppositories must be resorted to.

Stimulants.—Alcohol need seldom be given to children, but it is almost always required for patients over forty, and those who have been intemperate need it earlier and in larger quantities than others. The state of the heart affords the best indication; the necessity for stimulants is greater in proportion as the cardiac pulse is feeble, the first sound low, and the radial pulse rapid, compressible, or, above all, irregular. The propriety of continuing to give alcohol may often be determined by its effect on the heart's action. If the pulse becomes quicker than before, it is probably doing harm; if slower, it may be expected to do good. It is most needed during the second week; very seldom before the appearance of the mulberry rash. Low muttering delirium is often controlled by stimulants; a dry, brown tongue becomes moist, and other "typhoid" symptoms become less marked. On the other hand, severe throbbing headache and maniacal excitement are often aggravated.

Probably there is no direct advantage in employing one liquor rather than another. Some recommend, on various grounds, brandy or whisky or rum, others port wine; Buchanan speaks highly of beer. The amount must vary with the urgency of the symptoms. It is seldom necessary to give more than eight or ten ounces of brandy in the twenty-four hours. Of port wine as much as a bottle may be taken with advantage by patients who are desperately ill; but only small doses should be allowed at a time, repeated at intervals of an hour or two. Where there is great prostration, with cold sweats, hot punch or hot wine-whey is particularly useful.

Twenty or thirty drops of brandy may be administered subcutaneously. Ether, carbonate of ammonia, camphor, or musk may also be given by the mouth with good effect, particularly when alcohol fails.

Treatment of symptoms.—The *pyrexia* itself seldom requires direct treatment in typhus. When the temperature rises high, cold sponging or the wet pack is better than cold baths.

Quinine in large doses of ten or twenty grains, although it lowers the temperature for a time, does no real good. Indeed, the experience of Christison in Edinburgh, of Peacock in London, and of Haller in Vienna was that quinine is positively injurious; and this was the final conclusion of the medical officers of the French army in the Crimean epidemic. Antipyretic drugs like salicylates, thallin, antipyrin, antifebrin, are unsafe.

Murchison's favourite medicine at the London Fever Hospital was dilute hydrochloric acid, in doses of twenty minims, with a little syrup and tincture of orange. When there is insatiable thirst, he recommends a very weak infusion of cascarilla or quassia.

The early headache, which is often a very distressing symptom, may be checked by laudanum, particularly when it is accompanied by an inability to sleep.

Sleeplessness at any stage of typhus, when it lasts for thirty-six hours, should be combated by fifteen minims of laudanum or a quarter of a grain of morphia at night. When the patient is in a state of maniacal delirium, Murchison recommended chloral hydrate in a dose of twenty grains, but when the heart's action is feeble or irregular it does harm. Paraldehyde in half-drachm doses may then be prescribed, or chloralamide. Other useful sedative drugs are bromide of potassium, Indian hemp, and hyoscyamus. Each must be given in full doses to do any good.

When profound stupor appears to threaten the patient's life, Murchison advised that a cupful of strong coffee should be given every three or four hours. He also advised dry cupping to the loins, or mustard poultices, or flannels wrung out of hot water, when the urine was scanty and albuminous. Should high fever accompany coma, cold affusion on the head may be employed with great advantage.

For the pulmonary complications of typhus, ammonia is the chief remedy. But in some cases turpentine is said to be even more decidedly effectual; it is given in doses of fifteen minims every three hours.

For inflammatory swellings, usually in the parotid region, both Murchison and Buchanan recommend the application of a blister at an early stage, for it sometimes appears to prevent the occurrence of suppuration. When an incision is required, it should be made early.

Prophylaxis.—The true treatment of typhus, as of other specific fevers, is preventive. Its disappearance from England is due first to sanitary legislation, diminution of crowding and increase of cleanliness and comfort among the poorer classes. The isolation of cases in fever hospitals is the second prophylactic measure. The infection of typhus does not spread far, and is easily checked by dilution; moreover it does not last long. Four weeks from the invasion, or ten days of "quarantine" after convalescence, are sufficient safeguards.

So far as medical men and nurses are concerned, the danger of the extension of typhus may be greatly reduced by retaining for as long as possible those who have already passed through the disease, and by invariably selecting persons under thirty for vacancies that may arise.

Convalescence.—After the subsidence of the pyrexia, the patient may eat as soon as his tongue is clean and his appetite returns. If wine or brandy has been given, beer should be substituted while the case is still under treatment, so that there may be no excuse for carrying on the habit of tippling when health is restored.

When pyrexia has disappeared, the patient rapidly recovers his strength and appetite. Relapses are extremely rare; neither A. P. Stewart, Jenner, nor Murchison met with a single case. Sequelæ, as deafness and phlegmasia dolens, are almost equally uncommon—in striking contrast to their frequency after enterica. Moreover, while after a tedious convalescence the typhoid patient is often weaker for years, typhus, like acute pneumonia, leaves no harm behind it. Hence the joys of returning strength are unalloyed by misgivings for the future. The contest with this disease is severe and often perilous, but it is neither protracted nor indecisive. *Horæ momento cita mors venit aut victoria læta.*

RELAPSING FEVER

Λιμόν τε καὶ λοιμὸν γενέσθαι.—HERODOTUS, vii, 171.

History, and geographical distribution—Incubation, onset, and course—Relapse—Convalescence—Sequelæ—Death and morbid anatomy—The spirillum—Famine as a predisposing cause—Diagnosis—Prognosis—Treatment.

Synonyms.—Febris recurrens, Typhus recurrens, Febris recidiva. Bilious remittent fever; Seven-day fever; Five-day fever; Famine fever; Morbus pauperum; Irish fever; Yellow fever in Ireland (Graves); Typhinia (Farre, 1859).—*Fr.* Fièvre à rechutes.—*Germ.* Rückfallsfieber, Hungertyphus.

Definition.—A specific, contagious fever, with a peculiar microphyte, running a short course, and followed by one or more relapses.

History.—Typhus, as described in the preceding chapter, is only the residue of the larger group of typhous "continued" fevers. The most important advance made by a long series of researches during the first half of the present century was the differentiation of enteric fever; but during the same period a third fever also was recognised, which is generally known as Relapsing or Recurrent Fever. Other names are given above.

Probably we may identify this disease with that referred to by Hippocrates ('Epidemica,' lib. i, const. 3) as a fever at Thasos, which in some patients "departed on the seventh day, remained absent for a week, and then recurred for three days."

It was undistinguished from other so-called "continued" fevers. unless "bilious" and "synochal" varieties may refer to it, until 1770, when an Irish physician, Dr Rutty, described an autumnal epidemic prevailing in 1739 and 1741 as a fever of from four or five to six or seven days' duration, with intense headache, terminating in a critical sweat. . . . Patients were subject to a relapse, even to a third or fourth time, and yet recovered. It followed two seasons of great scarcity, and was far from being mortal (quoted by Dr Moore).

Barker and John Cheyne wrote in 1821 of the fever prevalent in 1800 and 1801 in Dublin, which generally ended on the fifth or sixth day by sweating, and was very liable to recur; and the same physicians described a later epidemic of the same disease, which was very prevalent in Ireland (1817-19), and also in London (3000 cases in 1818) and Scotland (2715 in Glasgow, 2400 in Aberdeen), 1817-18

Famine fever was again in Dublin in 1826, along with typhus. as recorded by Graves and Stokes, and in 1842-3 it was common in Scotland and Ireland.

It was common in Great Britain and Ireland in 1847-8, and again after a lull in 1851, when there were 256 cases in the London Fever Hospital. In 1854 only one, and since 1855 no cases were observed until 1868-9-70.* Since then a few deaths have been recorded here and there, one in Scotland in 1879, and three in Ireland in 1890.

Being far less fatal and of shorter duration than typhus, it was in 1826 regarded by Irish physicians as a mild variety of that disease, which was also prevalent at Dublin in that year. But in the year 1842 Dr Henderson, of Edinburgh, brought forward good grounds for believing that it was a distinct fever and due to a different poison. The same view was strongly upheld by Jenner in 1849-51, chiefly on the following grounds:—that the symptoms and course of the two diseases were different; that even when they prevailed together in the same town, one could never be traced to infection from the other; and thirdly, that neither afforded protection against a subsequent attack of the other. To these arguments may now be added the fact that in relapsing fever the blood always contains a microphyte, which is absent in typhus; so that there is now no doubt of the specific and independent character of relapsing fever.

Distribution.—In the great epidemic of 1847-8, and also in 1851, it was noticed, both in London and in other large towns of Great Britain, that most of the patients were destitute Irish, many of whom had recently left their own country, and that neither the English nor the Scotch were attacked.

On the continent of Europe Relapsing Fever has less often prevailed, but has probably formed part of some epidemics of typhus. It was recognised in Silesia in 1847, but, with this exception, it was unknown on the Continent until in 1863 it was observed in Odessa, and in 1864 at St. Petersburg. Next, in 1867, it broke out for the second time in Silesia, and in the following year it prevailed in Berlin and in other German towns, as well as in Breslau, where it was epidemic in 1872-3.

Griesinger in 1851 saw a fever in Egypt which he called Bilious Remittent, and afterwards visiting London saw cases of undoubted Relapsing Fever, and identified the two diseases. Dr Sandwith informs the writer (1896) that relapsing fever occurs in Egypt every spring.

In September, 1869, Relapsing Fever appeared at Philadelphia, and in November at New York. The patients were chiefly poor Irish or Germans, and it seems to be almost certain that the contagion was imported from Europe. On two or three former occasions it had been introduced into the same cities by Irish immigrants, but it never spread among the American population.

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* The first case was in July, 1868, that of an Irishwoman who had been for several years in London. Four days afterwards another case was brought into the Fever Hospital, a Polish Jewess, who lived near the first patient. Three weeks later there arrived from the street in which the Jewess lodged a girl who had lived all her life in London. The only other persons attacked in London that year were eight Jews, who were admitted into the German Hospital at Dalston. In October a severe outbreak occurred at Tredegar, in South Wales. In the autumn of 1869 relapsing fever became epidemic in several of the large cities of England; in London the disease reached its height in December of that year, and then gradually declined until June, 1871. The last cases were eight in Blackfriars in December, 1872. The relapsing fever of 1870 in Edinburgh is well described by Dr Muirhead in the 'Edinburgh Medical Journal' for that year.

It has repeatedly prevailed in India, and the well-marked epidemic of 1877 at Bombay was carefully studied by Dr Vandyke Carter ('Med.-Chir. Trans.,' vol. lxi). In 1864-5 it appears to have been observed in China.

Protection.—It is now ascertained that the same person may have relapsing fever more than once. It does not protect against typhus, nor does typhus protect from famine fever. The late Sir Robert Christison had relapsing fever three times. Dr J. C. Steele (then of Glasgow, for many years at Guy's Hospital) told the writer that, in the epidemic of 1848, persons who had previously suffered from typhus were not attacked by relapsing fever; and the same thing is said to have been noticed recently in epidemics at St. Petersburg and at Breslau. Dr Henderson, however, related six cases in which typhus occurred first and relapsing fever afterwards; and Murchison says that of thirty-one persons who contracted relapsing fever in the London Fever Hospital in 1868-9 no fewer than thirteen were known to have had typhus. Dr Hudson relates that in 1848 he attended a medical friend with relapsing fever, again three months later with typhus, and again several years after with enteric fever.

Incubation.—The period of incubation for this disease is very short; Murchison says that it sometimes breaks out immediately after exposure to contagion, sometimes not until twelve days have elapsed; but the usual period observed in this country has been four to ten days. Lebert finds it from three to seven days. During the interval no symptoms whatever are present.

Onset and course.—Relapsing Fever begins suddenly. The patient is seized with chilliness or with actual rigors; his head aches, he feels giddy, he has pains in the back and limbs, and his skin becomes burning hot. One of the distinctions from typhus is that it is giddiness rather than prostration which sends the patient to bed. Indeed, he is sometimes able to walk to the hospital on the third day of the fever.

The thermometer indicates a rapid rise of temperature. This begins even before the rigor, and within twelve or twenty-four hours it reaches 104° or 106°. The pulse, too, becomes quickened much earlier than in typhus: it is seldom below 110, and not infrequently reaches 140 on the second day. The respiration is not quickened in proportion to the pulse and temperature. The tongue is moist, and covered with a white or yellowish fur. There is great thirst. Appetite is generally wanting; but sometimes it remains good, or even voracious, and the patient is able to take solid food without harm. In other cases, however, there is much nausea, and sometimes vomiting. Pain, with tenderness on pressure, is often present in the epigastric and hypochondriac regions. By percussion increased dulness of the liver may be detected, and the spleen is usually enlarged, so that its edge reaches below the costal cartilages. Jaundice often occurs on or after the third day; in some epidemics it has been observed in a fourth of the cases, but generally not oftener than in a twelfth. In these cases the urine contains bile-pigment; but the fæces remain dark. The face is flushed and the eyes are injected, but the countenance is not usually dusky, nor is the expression stupid and confused as in typhus.

As a rule, there is no exanthem; but several observers have noticed a roseola in certain exceptional cases, and in the epidemic in Silesia of 1847 a rose-rash on the second or third day was the rule. Murchison, who saw a rash in only eight out of about six hundred cases, says that it consisted of small spots, or of a reddish mottling, sometimes resembling measles, sometimes undistinguishable from typhus at an early stage; yet always disappearing under pressure, and fading after a few hours or within three or four days at the latest. It came out sometimes during the first attack, sometimes in the relapse; and either on the third day or immediately before the crisis. Petechiæ are not uncommonly present; no doubt fleabites have frequently been taken for them, but Murchison says that large numbers may come out after the patient's admission into hospital. The surface of the body often remains very dry, but sometimes on the second or third day there is profuse sweating, which may last for hours without relieving the patient. Sudamina may be developed in large numbers, and are probably the cause of desquamation of the cuticle, which frequently occurs later on. Some cases of undoubted Recurrens have been described as Miliary fever.

When relapsing fever occurs in a pregnant woman, gestation is almost invariably brought to an end, whatever may be its period; but it is curious that the abortion or miscarriage is not seldom delayed until the relapse. The child, if not stillborn, survives only a few hours. This is a marked contrast with what occurs in typhus.

In the regular course of the disease the patient's condition undergoes but little alteration for about a week. The temperature and the pulse remain high, though oscillating up and down, and the pulse-rate sometimes reaches 150, 160, or even 180. The patient continues to suffer from severe headache, and from intense pains in the muscles and joints. Sleeplessness is almost always a marked symptom, and the mind generally remains clear. Relapsing fever is altogether attended with far more suffering than typhus; persons who have passed through both diseases invariably look back upon the former as the worse.

Crisis.—As a rule the fever suddenly subsides, on the fifth, sixth, or seventh day. Sometimes, however, the crisis takes place as early as the third day, and sometimes as late as the tenth. Just before it takes place the pyrexia reaches its *acme*, and this last rapid rise may be as much as 4° Fahr. In some exceptional instances maniacal delirium suddenly comes on at this time; the patient screams and struggles violently; but within a quarter of an hour becomes calm again, and has no recollection of what has occurred. In other cases the crisis is said to be ushered in by epistaxis, diarrhœa, or the appearance of the catamenia; but the most frequent sign of the crisis is profuse sweating. The change is wonderfully complete. In the course of a few hours the temperature becomes normal, or even lower than normal, having fallen 8° or 10° without a break; Murchison cites one case in which there was a range of 13° in six hours, and another in which the difference amounted to 14·4° in twelve hours. At the same time the pulse drops from 120 or more to about 70, the tongue becomes clean, and the patient loses all his pains. Lebert remarks that the period from evening to morning is that at which the crisis is most apt to occur. Litten has seen it sometimes preceded by a *pseudo-crisis*, the temperature falling below normal, but within twenty-four hours rising to as high a point as before, again to fall on the following day.

Interval.—During the *interval* which follows, the patient feels perfectly well. At first he is rather languid; but his appetite is good, he quickly regains his strength, he leaves his bed, and sometimes insists on returning to work. His temperature for two or three days is almost always lower than in health, and afterwards normal. The pulse is at first rather above the natural standard; and then remarkably infrequent, perhaps not more than 44 or 50. At the same time it is unstable, and may rise to over 100 when the patient assumes the erect position.

Relapse.—In exceptional cases this apparent convalescence is really the end of the disease. But, as a rule, it is abruptly terminated, after seven days, by a sudden reappearance of all the symptoms of the first attack. Sir Robert Christison used to relate a striking anecdote of his colleague, Dr Hughes Bennett, who took relapsing fever at the commencement of the epidemic of 1843, when its characters were known only by the older physicians in Edinburgh. "When he had detailed his case," being then supposed to be convalescent, "I told him he had sustained an attack of my old acquaintance, . . . whose face I had not seen for a good many years; that he was not yet done with it, and that he would have another attack, commencing with rigor, on the fourteenth day. Dr Bennett, surprised—I will not say incredulous,—replied that the relapse had no time to lose, as there were only three or four hours of the fourteenth day to run. It did, indeed, lose no time, for I must have scarcely reached home from his house before the rigor set in with violence."

Sometimes instead of occurring on the seventh day of the interval, the relapse begins on the second, or the third, or the fifth day; sometimes it is postponed for several days, and even as late as the twenty-fifth day. Lebert says that it usually sets in at night.

This second attack is generally shorter than the first one, lasting three days, or not more than one or two; but sometimes it is prolonged to five days, or even to seven. In the details of their symptoms and in their severity the two attacks may resemble one another exactly, but in some patients the relapse is the worse, especially when the original attack was very mild; in others it is far less severe, and it may be so slight as almost to escape notice. When well marked, it ends in a sudden crisis, just as before.

Recovery.—The patient now generally passes on to a permanent recovery, but sometimes there is a third attack, which begins between the twenty-first and the twenty-fourth days of the disease; it is almost always mild, and seldom lasts more than forty-eight hours. There may occasionally be a fourth or a fifth attack.

Convalescence after relapsing fever is slow, compared with that after typhus. The patient is long in regaining his strength. Although the duration of the disease up to the second crisis is only about eighteen days, he is seldom able to return to work within six weeks.

Complications are not frequent. The most serious—lobar pneumonia—is also the least common. Diarrhœa is frequent, and still more so is jaundice. Among 903 cases reported from the London Fever Hospital by Dr Collie, there were 48 of icterus, 16 of diarrhœa, 27 of epistaxis, and 1 of hæmaturia.

Pneumonia was not infrequent at Breslau in 1872-3. Lebert says that it was generally double, that it occurred chiefly in those who had been intemperate, and that it sometimes set in during the attack, sometimes in the relapse. It has been known to lead to pulmonary gangrene. Bronchitis is commonly present, but is seldom severe. In St.

Petersburg hæmorrhagic pachymeningitis is said to have been observed. The spleen has sometimes ruptured and caused death by hæmorrhage, or an embolic block has broken down and set up fatal peritonitis. Dysentery has been a frequent and dangerous complication in some epidemics. The occurrence of abortion is sometimes fatal. Inflammatory swellings or "buboes" in the parotid regions or in the groins are said to have been often the cause of death at St Petersburg, but in England they appear to have been more frequent in cases which recovered.

Sequelæ.—Relapsing fever sometimes leaves traces behind after recovery. Severe articular pains often continue during convalescence, and effusion into the knee- or ankle-joint has now and then been seen.

Another sequel, which sometimes does not set in for several days, or weeks after the subsidence of the fever, is a form of ophthalmia. This is described as beginning with an amaurotic stage, in which the retina, or perhaps the choroid, is alone attacked. Afterwards, in a second, inflammatory stage, when the more superficial structures are involved, there is intense pain. Fortunately, it is rare for both eyes to suffer, for recovery is always tedious, and there is sometimes permanent loss of vision.

Exitus lethalis.—It is only in exceptional cases that relapsing fever proves fatal. In this country the mortality has been about 4 per cent. In Bombay, however, Dr Vandyke Carter estimated it at 10 per cent. In persons advanced in years the disease is decidedly more dangerous than in younger patients.

Death occurs sometimes by syncope. At and after the crisis the pulse is often very weak, small, or irregular, and there may be temporary impairment of the first sound of the heart and of its impulse. These symptoms usually soon disappear, but a patient who may have had a mild attack, and who appears to be doing well, is now and then found pulseless, cold, livid, and unconscious, and dies in a few hours. This sometimes takes place at about the period of the first crisis, but sometimes during the interval. In three such cases, observed by Murchison, the heart was found fatty and dilated.

Another mode of death is by suppression of urine with convulsions and other uræmic symptoms. In some instances of this kind, recorded by Dr Henderson as far back as 1843, Dr Maclagan discovered urea not only in the blood, but also in fluid from the cerebral ventricles. It is probable that in these cases the kidneys were diseased, and that albuminuria was present during life; but the urine may contain albumen, and even blood, in relapsing fever, without any serious consequences resulting.

Again, the disease may be attended with typhoid symptoms and jaundice, and these cases often terminate fatally; this Griesinger described as a distinct "bilious" form of fever, with hæmorrhages into the skin and from mucous surfaces, including that of the stomach, so that the vomit may be black, as in yellow fever. The same comparison was made by Stokes and O'Brien in Ireland. The interval is imperfectly marked, the pyrexia running on more or less continuously from the first attack to the second. Heydenreich, in St. Petersburg, and Carter, at Bombay, have each shown that the spirillum was present in these cases of bilious recurrent fever, so that there is no doubt of their true nature.

Anatomy.—The *spleen*, if death occurs during the attack, is found to be more or less soft, and much enlarged, more so than in enteric fever. Sir Wm.

Jenner once found it weighing thirty-eight ounces, and Küttner is said to have met with a spleen four and a half pounds in weight. It sometimes contains embolic wedges. In the one case which came to an autopsy at Guy's Hospital during the epidemic of 1869-71, the spleen, which weighed fifteen ounces, had a remarkable appearance. It contained a number of small yellow softened patches or abscesses, the largest being of the size of a horse-bean, which seemed to follow the branchings of minute veins. Dr Moxon could discover no thrombi in any of the vessels, even with the aid of the microscope. Very similar appearances were observed by Litten, at Breslau, in the epidemic of 1872-3. The *liver* is also increased in size. As a rule, the *kidneys* are gorged, and the epithelium in the renal tubules is in a state of cloudy swelling.

Ætiology: the pathogenic microbe.—Relapsing fever was the first specific fever in which the nature of the contagium was discovered and its pathogenic significance demonstrated. It is morphologically a vibrio, and has been named *Spirillum Obermeieri*,* after the late Dr Obermeier, of Berlin, who first described it in 1873.†

Its ordinary appearance is that of a delicate, homogeneous, spirally twisted filament; its length is from $\frac{1}{1000}$ to $\frac{1}{500}$ of an inch, or equal to from one and a half to six times the diameter of a red blood-disc (15—40 μ). It is never still, and its motion is compounded of a rotation on the long axis, a progression forwards or backwards, and a lashing movement, which lengthens it out and makes the twist disappear for a moment.

All observers admit that the presence of the spirilla is coincident with the attacks of relapsing fever, and that during the interval it cannot be detected. Birch-Hirschfeld, however, once found it for two days after the second crisis, and several writers, including Litten, have stated that it is seldom to be seen during the first two or three days of the disease.

Heydenreich, of St. Petersburg, found that while it appears before the thermometer begins to rise, it ceases to be discoverable before the commencement of the crisis. He has specially inquired into the influence of heat upon this organism outside the body, and he finds that it remains alive and active much longer when it is kept at a temperature of 60° or 70° F. than at blood-heat, whereas at fever temperatures it dies more quickly still. His hypothesis accordingly is that the pyrexia, which is itself caused by the existence of this organism in the blood, proves directly fatal to it. In all probability the filament breaks up into a number of minute granules, and some of them may perhaps constitute the germs from which fresh crops of spirilla are afterwards developed. Heydenreich has occasionally seen some of the filaments beset with granules so as to resemble a necklace, and the late Dr T. R. Lewis, of Calcutta, speaks of having once noticed a beaded appearance. If the germs remain alive in the blood throughout the whole of the interval, they must be the resting spores, for the spirochæta itself dies at the temperature of the blood in about twenty hours.

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* Others refer it to the genus *Spirochæta;* for Ehrenberg in 1833 distinguished two genera of *schizomycetes* under these names, the difference between them being that *Spirochæta* possesses greater flexibility than *Spirillum*. This distinction is preserved in the nomenclature of Cohn and Flügge; but the two "genera" are closely allied, and the names are applied almost indifferently to the microbe discovered by Obermeier.

† It seems doubtful whether the form found in the blood of relapsing fever is identical with that which had previously been observed in water containing vegetable matters (*Spirochæta plicatilis*, Ehr., Cohn.) and in the saliva of healthy persons (*S. denticola*). Dr Vandyke Carter stated that the dimensions of an organism of this kind which he found in the water of a tank at Bombay were much larger than those taken from his fever patients ('Med.-Chir. Trans.,' vol. lxi).

During a single attack, Heydenreich finds that there are extraordinary fluctuations in the numbers of these microphytes from day to day. He therefore supposes that successive generations are more or less constantly produced throughout the fever. Sometimes after they have been present for two or three days they suddenly cease to be discoverable; but a few hours later they reappear in greater or less abundance. Their recognition in the blood, at the end of an interval, has repeatedly enabled him to foretell the approach of a more or less marked relapse. On the other hand, Dr Carter, at Bombay, sometimes detected the spirochætæ when there was absolutely no rise of temperature; while in other cases a one-day febrile attack would occur at the proper time for a relapse, without the blood containing the spirillæ. Dr Carter remarked that the numbers of the microbe must often amount to hundreds of millions, for dozens are seen in the field of the microscope at the same time. It is possible that they may aggregate into masses with blood-corpuscles, and seriously interfere with the circulation. In fatal cases they cease to be discoverable after death. None of the solid organs have hitherto been found to contain them. Heydenreich could not detect them in the urine, nor in the conjunctival secretion, in the fluid from the pleura, the intestine, or the bronchial tubes.

Spirochæta (*v. Spirillum*) *Obermeieri* has been successfully cultivated out of the body, but for a long time all attempts to reproduce the disease failed. Injections of blood infected with it into the circulation of dogs, rabbits, or guinea-pigs were made by Obermeier himself without result, and the same negative result followed inoculation of sheep. Nor did he find that the disease was propagated by the penetration of minute quantities of blood from patients with relapsing fever into scratches upon the hands of healthy persons. Dr Carter has since recorded the fact that a few days before he himself was attacked he had scratched his finger in making an autopsy upon a fatal case. Finally he succeeded in inoculating monkeys with the spirillum taken from the blood of patients suffering from relapsing fever. The spirillæ were found after death in the viscera of the animals experimented upon ('Lancet,' 1879 and 1880). This completed the chain of evidence, that this microbe is the contagium vivum, and the sole cause of relapsing fever, according to the criteria laid down on p. 18.

Mode of spreading.—Direct contagion from the sick is the chief mode of propagation of this disease, and it is exceedingly apt to pass from hospital patients to nurses and clinical clerks. In 1843 the post of house physician in a fever hospital at Edinburgh had to be filled six times in five months, owing to successive incumbents being attacked. In the London Fever Hospital, during the years 1869 and 1870, twenty-seven of the nurses and officers, and five patients admitted for other diseases, contracted relapsing fever. In 1870 a nurse from the London Fever Hospital was transferred to St. Mark's Hospital; and about the same time a wardmaid from St. Mark's paid a visit to the Fever Hospital. These two women both fell ill at St Mark's, and gave relapsing fever to five other persons there. Two striking instances of direct contagion were recorded by Mr Reid, of Glasgow, in 1843.

(1) At Dalmarnock Colliery there was a large building of three stories, which was entered by separate stairs and contained forty families; some

Irish people brought relapsing fever with them into the uppermost flat, whence it spread and attacked twenty-two persons in two months.

(2) Into a house of two apartments, in which eleven human beings were lodged, a person from a neighbouring village introduced the fever; everyone there caught it, but all the occupants of the next house escaped, although they were almost as closely crowded together.

That the disease may be transported to a distance by infected clothes seems to have been proved by two cases recorded by Dr Parry, of Philadelphia, in 1870; and in 1843 it was noticed in Edinburgh that a large number of laundry-women contracted relapsing fever, although they had no other communication with the sick than washing their clothes and bedding.

Litten has related how a mason, who himself remained well, but who slept in a street in Breslau in which the disease was prevailing, carried the contagion to his mother, who lived where no case had before appeared.

The facts of this Breslau epidemic of 1872-3, which are to be found in the 'Deutsches Archiv' for 1874, strongly support the conclusion that microbes are given off either with the breath or from the skin, and are received into the air-vesicles with the inhaled air. Litten has clearly shown that at Breslau they were not conveyed in drinking-water.

Predisposing cause.—The cases observed by Litten in Silesia seem to have belonged to the poorest classes, with the exception of the physicians who took the fever in the hospital; and English writers have generally regarded as the cardinal fact in the ætiology of this disease its occurrence in a starving population. It is the *famine fever* of Ireland. Murchison cites instance after instance to prove that those who have suffered from it in England and in Scotland have, with certain exceptions, been in a state of extreme destitution. When it occurred in Silesia in 1847, the inhabitants, in consequence of a succession of bad harvests, had been reduced to subsist on clover, grass, mushrooms, and the roots of trees, Carter states that it was brought to Bombay in 1877 by the peasantry flocking into the city from famine-stricken districts. History shows that as Typhus is the pestilence of war, of camps, and of sieges, so Relapsing fever is the pestilence of famine. Relief of extreme destitution in districts where relapsing fever was prevailing has repeatedly been followed by the subsidence of the epidemic. Starvation renders persons more susceptible to the contagion, probably by weakening the power of the living cells of the body to cope with the invading microbes.

At the same time persons well nourished and in perfect health may take the disease if exposed to the contagion. There are many cases of physicians, nurses, students, and others, well fed and healthy, who caught the disease from patients. In 1870, at London Fever Hospital, out of 136 such persons (not patients) thirty-four took the fever.

Age and sex.—Relapsing fever may attack persons of all ages, from five months to seventy-five years, but most of the patients are between the ages of fifteen and forty. There are more male than female patients, the reason probably being that more men than women are beggars, hawkers, and vagrants.

Diagnosis.—During the first day or two relapsing fever may be mistaken for typhus or for smallpox. Since it frequently prevails at the same time with typhus, the early diagnosis between these two diseases is important, because of the difference in gravity of prognosis. How useful the detection of the spirochætæ may be, was shown by Dr Carter at Bombay, where there was often difficulty in distinguishing the effects of malaria from the more continued varieties of relapsing fever, and where so multiform were the phases of the spirillum fever that about 25 per cent. of his cases might be termed irregular.

Prognosis.—Most patients attacked by relapsing fever recover. It is far less fatal than typhus or enterica. Out of 441 cases collected by Murchison from the records of the London Fever Hospital only 11 proved fatal; and in the great Scottish epidemic in 1843, of 6300 cases only 260 died. On a basis of over 14,000 cases he estimated the mortality at less than 5 per cent. Among 903 cases afterwards collected by Dr Collie there were only 11 deaths, *i. e.* 7 among 572 men and 4 among 331 women. As usual in epidemics, the worst cases come first, and the disease becomes gradually milder. Age is a serious circumstance, as in typhus and enteric fever. Petechiæ, delirium, and scanty urine are grave symptoms.

Treatment.—This is not so successful as might be hoped, for we have as yet no means of preventing the relapse. With this object quinine and arsenic have been employed both in England and in Germany in vain. At Breslau carbolic acid, and in America the sulphites, the hyposulphites, and the preparations of chlorine have been used with no better result. Perhaps the salicylate of soda, or the sulphocarbolates, or perchloride of mercury, or some other parasiticide may be more effectual.

An emetic at the onset of the disease affords much relief to the severe hypochondriac pains. Patients often find comfort from frequent sponging of the surface with cold or tepid water, and from cold affusion on the head. Throughout each attack castor oil, or some other mild aperient, should be given. Murchison recommended the systematic administration of diuretics, which he believed often prevent the supervention of uræmic poisoning. He directed that from one to two drachms of nitre, one drachm of dilute nitric acid, and half a drachm of tincture of digitalis should be taken in divided doses in the twenty-four hours. He often gave opium or hydrate of chloral for the relief of headache, or of the muscular and arthritic pains.

Patients should be taken to a hospital as early as possible, and placed together in wards set apart for the purpose, since Litten seems to have shown that the accumulation of cases neither augments the mortality nor appreciably increases the danger of medical attendants and nurses.

THE PLAGUE

"The pestilence that walketh in darkness."

History and geographical distribution—Incubation, onset, and symptoms—Diagnosis—Anatomy—The specific bacillus—Mortality and prognosis—Treatment—Conditions of contagion and causes of the decline of the plague. The Sweating Sickness—History—Symptoms and pathology.

Synonyms.—Pestis vel Pestilentia, λοιμός, Pestis inguinaria, Typhus bubonicus, Black death, Levantine or Oriental plague.—*Fr.* La peste.—*Germ.* Die Pest. In India known as the Bombay plague, the Pali plague, and Mahamuree.

Definition.—A specific contagious fever, with a short course and high mortality, characterised by inflammation of the lymph-glands, and by the constant presence of a specific microbe.

History.—The English word Plague (*plaga*, a stroke) was formerly applied indiscriminately to every fatal epidemic disease; and when the malady now to be described first appeared in England in the fourteenth century, it was called the "Black Death." But other epidemics formerly included under the comprehensive terms plague and pestilence are now distinguished as Variola, Typhus, Cholera, and Yellow Fever. Hence we can now use the word Plague without ambiguity for the bubonic or Oriental Pestilence.

The history of the true plague, identified by its mortality and its buboes, can be clearly traced back to an Egyptian epidemic under Justinian, about 550 A.D., and notices by medical writers show that it was recognised as early as the time of Trajan. It seems improbable that the "Plague of Athens" described by Thucydides was the Oriental Pestilence.*

Epidemics of the plague were frequent during the Middle Ages, when it was probably almost constantly endemic in Persia, Syria, and Egypt. The Black Death of 1348-9 began in China, spread to the Crimea, and thence by a ship's crew to Genoa, devastated the whole Continent, and put a stop to the war between England and France. Its ravages in Florence were recorded by Boccaccio. It probably reduced the population of England by one third, or even more, and thus led to important economic results. The same epidemic invaded Syria by way of Armenia, and thence spread over Egypt and the whole of Northern Africa, the hotbed of plague in the days of the Roman empire.

There were several epidemics in the fifteenth and sixteenth centuries, and again in 1609, in the first year of Charles I (1625), and during the civil war (1635-47); but the most destructive and happily the last in England was the Great Plague of London in 1665, so graphically described by Defoe, although he was but two years old at the time, and could only know what he tells by hearsay. It came to us from Amsterdam, after having devastated Italy and Central Europe. In 1665 no less than 68,596 deaths from the plague appeared in the bills of mortality for London.

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* There is no mention of buboes or of hæmorrhage, and the symptoms recorded are unlike those of the Oriental plague. Murchison believed the plague of Athens to have been yphus, Littré and Daremberg that it was smallpox, and a book has been published to rove that it was scarlatina. It may have been any of these diseases, or relapsing fever, r measles in a virgin soil. Except fever and contagion, the recorded symptoms do not

There was a terrible epidemic in the south of France, known as the Plague of Marseilles, in 1720, when the scenes described by the great English novelist were renewed. The disease did not visit Western Europe again, but epidemics occurred during the eighteenth century in Sicily and Poland (1743), Wallachia and Russia (1771).

In the early part of this century there were outbreaks of the plague in Malta, at Noja in Southern Italy, upon the Lower Danube, and in the Balkan Peninsula. The last outbreak in Constantinople was in 1841. Up to 1844 the plague was frequent in Egypt, and after a long interval it has lately reappeared (1897-9). The disease also vanished from Smyrna and Syria, and hopes were entertained that it had become entirely extinct in the Mediterranean area. But in 1858 it appeared again at Benghazi, in Tripoli; and it is now known to have visited the highlands of Western Arabia in 1853. There is also reason to believe that it has never been absent for many years together from parts of India; in 1834 an epidemic which occurred in Rajpootana became known as the "Pali plague," and as recently as 1877 it prevailed at Kumaon, on the southern slopes of the Himalayas. Between 1863 and 1876 there were six or seven outbreaks in Western Asia, some in Persian Kurdistan, others upon the Lower Euphrates. In 1877 it showed itself at Resht, to the south-west of the Caspian Sea. From this place it probably made its way to the Lower Volga, where it prevailed, in the district between Astrakhan and Tsaritzin, from October, 1878, to February or March, 1879, and excited great alarm throughout Russia, and in Europe generally.*

This terrible disease, long obsolete in Europe, has thus of late years become rare in the Levant; but it still occurs epidemically at intervals in Yunnan (China), in Kumaon (Northern India), in Irak-Arabi and Syria, in Kurdistan and Northern Persia, in Arabia (1889), and in the single town of Benghazi, in the province of Barca. It is believed to exist as an endemic disorder in certain parts of Northern India. A terrible epidemic devastated Canton and Hong Kong in 1894, and another visited Bombay in 1896-7. The plague lasted from May till the beginning of September, and killed 2550 persons.

In the present year (1899) this ancient scourge of Europe has reappeared in places where it has not been heard of for nearly two hundred years, and at Oporto and some smaller places in Portugal the Oriental plague with its distinctive characters has destroyed many lives, including that of at least one physician.

The plague does not appear in the tropics, and is stopped by the hot season in subtropical regions. Nor, with few exceptions, does it survive the cold of winter in European climates. The most favourable temperature is from 60° to 85° Fahr. It has never spread to the New World, to Australia,† or to South Africa.

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* The English Government sent Surgeon-Major Colville and Dr J. F. Payne to investigate the disease on the spot, but it had already disappeared on their arrival at Astrakhan.

† The journals report an outbreak of Plague in New South Wales (April, 1900).

Clinical course.—The *incubation* of the plague is believed to be from two to seven, usually from three to five, days. In Hong Kong this period was not less than six nor more than nine (Sir William Robinson's despatch, 'Colonial Reports,' No. 148).

Symptoms.—The disease sets in abruptly, and runs a short and severe course. It begins with shivering and muscular pains, great malaise, and depression; with dulness and mental apathy—typhus in the original meaning of the word. Intense pyrexia then sets in, the temperature rising to 106° or 107° on the first day; violent delirium is common, and the symptoms rapidly assume a typhoid form. Death not uncommonly occurs at a very early period, before there have been any symptoms characteristic of the malady; it may be preceded by the appearance of petechiæ and large vibices in the skin; there may also be hæmaturia, hæmoptysis, and vomit blackened by altered blood.

If life is prolonged to the second or third day there appear one or more *buboes*, attended with severe pain and tenderness. The seat of this affection may be either the groin, the armpit, or the neck. According to Liebermeister, a plague-bubo is sometimes so small as to be detected only on careful examination, but it may attain the size of a hen's egg, though more often not bigger than a walnut. The apparent size is increased by œdema. Its formation is commonly attended with subsidence of the delirium and of fever, the skin becoming covered with a profuse sweat, and the pulse falling to 90 or 100. It may ultimately disappear without bursting; but usually it breaks, or is opened by the surgeon, and gives exit to a mixture of blood and pus, and this may be followed by a tedious process of suppuration, which greatly prolongs the patient's illness.

Cutaneous gangrene may be associated with the buboes, but is much less frequent and of more fatal augury. It appears most often upon the lower limbs, on the buttocks, or on the neck; sometimes there are not less than a dozen of these so-called "carbuncles" in the same case. Bullæ and pustules may also be seen.

Petechiæ and vibices are usually present over the surface of the body, and these were the "plague-spots" and "tokens of the plague" in the popular accounts of the pestilence in England. They are more numerous than the petechial spots of typhus, and probably gave rise to the epithet which distinguished the pestilence of the fourteenth century as "the black death."

The fever often falls by lysis after a course of a fortnight, but occasionally by crisis in the first week. Death may take place with delirium and coma in the first five or six days, or even earlier—even on the day of attack. Pestis siderans is the name given to these acute and fatal cases.

Hæmoptysis is the early and constant symptom mentioned by the describers of all the mediæval epidemics (see Hirsch and Hecker). Hæmatemesis and melæna are less common; epistaxis, menorrhagia, and hæmaturia also occur, and the latter two are said to be of fatal import.

Cases with suppurating buboes are the most favourable; they often last beyond a week, and sometimes beyond a month.*

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* "Those that die of the plague die a very easy death generally: first, because it is speedy; secondly, because they die without convulsions. They do but of a sudden fetch their breath a little thick and short, and are presently gone, just as you squeeze wind out of a bladder" (Boghurst).

Pneumonic plague.—Sometimes pneumonia, which is a frequent complication of the plague, forms the chief clinical feature of the disease.

Dr L. F. Childe, writing from Bombay to the 'Guy's Hospital Gazette' (July 17th, 1897), describes cases of broncho-pneumonia with hæmoptysis, in which no buboes were present during life, although the internal lymph-glands were found to be swollen, red, and soft, and the specific bacillus was present in cultivations from the lungs and spleen. He thus tells the history of the fatal illness of a surgeon, who contracted it in his official duties.

Surgeon-Major M— was in his usual health on January 2nd, 1897, when he had a rigor in the morning. During the day a bad headache developed; he had nausea and vomiting with pains in the limbs. At 2 p.m. temp. 103·4°, pulse 116, resp. 25. After a bad night he felt worse, and the temp. rose to 104°, pulse 110, resp. 23. Jan. 4th (third day), temp. 104·6°, pulse 113, resp. 25; tongue moist; no enlarged glands. During the night he began to cough and brought up some watery sero-mucous fluid, slightly blood-tinged, with pain in the left axilla. Here moist sounds could be heard like those of early pneumonia, and also below the left clavicle; the rest of the lungs and the other organs appeared to be normal, as did the lymph-glands. The symptoms were not like ordinary pneumonia, for the onset was different, and the condition of the tongue and mouth different; there was no dyspnœa or pneumonic disproportion of pulse and respiration, and the sputum was not at all like rusty sputum, for it was loose and free, and coming up with the slightest cough; it was watery, looking more like serum than mucus, and it was slightly pink, not rusty yellow. Also the patient's general condition was far worse than could be explained by the small amount of pneumonia present. I examined the sputum under the microscope, and found it full of bacilli looking like those of plague, and cultures were made from which a pure growth of the plague bacillus was obtained. During 4th and 5th patient became steadily worse, his temperature remained about 104°, and his expectoration became most profuse; the moist sounds were heard over a larger area, as well as slightly at the bases; the respirations increased to 35 and then to 45, and the pulse to 120 and 135; and he ultimately died early on January 6th (the fifth day of the disease).

The nurse who attended him unfortunately died of a similar form of plague. She became ill on the evening of January 7th, and showed symptoms of pneumonia on the 8th. She rapidly became worse, and died on the 10th. Her sputum was not nearly so profuse as in the former case, and symptoms of exhaustion came on much earlier. She also had no glandular pain or enlargement whatever, and bacteriologically her sputum was exactly as described above.

Pestis minor.—There is a much milder form of this disease, which often precedes or accompanies an epidemic of the plague, and has been called the masked, aborted, or lesser pestilence, endemic miasmatic pest (*pestis larvalis s. minor*, *peste fruste*, *pestis ambulans*). There is no hæmorrhage in these cases. The buboes are few and chronic, and the pyrexia moderate. The illness lasts about a fortnight. So slight are some of these cases that they have been regarded as a separate malady; but there seems to be no doubt of their true nature, notwithstanding the absence of cases intermediate in severity. This mild variety usually precedes an outbreak of the severe form, as it did in 1664 in England. It appears not to be contagious.

Diagnosis.—It is not always easy to distinguish the plague from other malignant fevers until the character of the prevailing epidemic has been determined. Dr Milroy, in Reynolds' 'System,' cites the remark of Heberden that, "on first breaking out, the disease has never been known to be the plague;" and he says that in Constantinople or in Cairo no physician ever ventures to give a name to an epidemic until a case occurs in which a bubo or a carbuncle is seen.[*]

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[*] In a small treatise, *Pestis descriptio*, by Gratarolus, of Bergamo, published at Paris in 1561, twelve signs of the disease are given, most of which are indicative of any severe fever; but the twelfth is *omnium certissimum: si cum febre post aures aut sub alis aut*

The diagnosis has often been left open from that reluctance to admit unpleasant truths which has always contributed to the spread of an infectious disease. According to Dr Milroy, a difficulty has often arisen, during the prevalence of the plague, from the occurrence of glandular pains and swellings, or of carbuncles, in persons who remain well enough to follow their occupations, and who speedily recover almost without treatment (pestis minor). Liebermeister remarks that these cases are often observed when an epidemic is declining.

The early severity of the symptoms and the presence of the characteristic petechiæ and buboes are the distinguishing points for diagnosis, and in doubtful cases inoculation of rats will decide the point.

Morbid anatomy.—*Post-mortem* examinations throw little or no light on the pathology of the plague. The viscera are soft and blood-stained, and the spleen is more or less swollen. The serous membranes are ecchymosed. Some of the internal lymph-glands are enlarged; perhaps a chain of swollen glands, extending up from the pelvis along the front of the spine, or a mass lying in the mediastinum; their tissue is of a bright red colour, or full of points of suppuration; and blood may be diffused and extravasated into the surrounding structures. Briefly, the autopsy is like one of typhus with the addition of swollen and suppurating lymph-glands.*

The contagion.—That the plague is propagated by an infective virus derived from the bodies of the sick is now doubted by no one, although in former times there were "non-contagionists" with regard to this, as to every other disease.

The specific microbe of the Oriental plague was discovered by two Japanese pathologists at Hong Kong in 1894, Kitasato and Avyama: the latter was himself attacked by the disease, but recovered (see 'Lancet,' August 11th and 25th, 1894). Their results were confirmed by Dr Yersin, who was sent thither from Paris, as they were from Japan, to study the epidemic by modern methods of inquiry; also by Dr Cantlie at Hong Kong, and by Dr Surveyor at Bombay. The microbe is a short thick bacillus. It grows freely in agar-agar or serum at the temperature of the normal or febrile human body. It is very abundant in the lymph-glands and spleen, and present, though not in large numbers, in the blood. When a pure cultivation is injected into rats or other rodents it reproduces fever with "plague-buboes," and it reappears in large numbers in the spleen and lymph-glands.

The contagion may be transmitted from man to man by contact or by fomites, but it is also capable of transmission from infected soil and houses—*i. e.* plague might be called miasmato-contagious (*cf. supra*, p. 16). This conclusion had been reached by Boghurst, in his 'Loimographia,' 1666, and in modern times by Liebermeister ('Ziemssen's System'), by Payne ('Encycl. Brit.,' art. Plague), Creighton ('History of Epidemic Diseases'), and other observers, who based their belief on the singularly local character of the disease; on the fact of persons removed from the soil, as the vast river population of Canton in 1894, and the sailors and bargemen on the Thames in 1665, escaping during the epidemic; on infection persisting in certain houses or streets, and on its being spread by

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circa inguina potissimum bubones sine manifesta aliqua causa, aut in aliis etiam partibus carbunculi subito oriantur. Among the other symptoms *sanguinis sputum* is mentioned.

* The first account of an autopsy is given in a curious work by Dr Geo. Thomson called 'Loimotomia, or the Pest anatomised,' published in 1666.

rats and mice; and lastly on the old belief, confirmed by recent observation, that "plague does not go upstairs." The infection is easily dissipated by free ventilation, and travels very slowly from place to place.

Many of the clergy and physicians who remained firm to their duties in the great plague of London escaped, and the same exemption was noticed by the French army doctors at Cairo in 1800.

The virus seems often to be conveyed by articles of clothing, wool, silk, hair, paper, books, &c. In 1665 the local epidemic at Eyam, in Derbyshire, so well known by the heroism of the vicar, Mr Mompesson, began with the case of a tailor who had just received a box of clothes from London (150 miles off), and who was watching them hanging before the fire when he was taken ill; at that very time the disease in the metropolis was at its worst. The bedding and the linen of those who have had the plague are particularly apt to be carriers of infection to other persons, sometimes after a considerable interval of time. When epidemics used to occur in Western Europe the disease was supposed to reach Holland direct from Turkey in bales of cotton or silk, and to be brought over to England from Holland in a similar way. Nevertheless the exportation of cotton from Alexandria to Liverpool and Marseilles during the prevalence of the plague in Egypt in the year 1835 was not prohibited, and yet no cases appeared in England or in France.

It seems more than probable that the plague is often conveyed by rats, and possibly by flies also.

Predisposing causes.—In European Turkey the plague used chiefly to appear in the spring and early summer, whereas in Egypt the warm winter was its usual season. In Nubia and in other countries with a hot dry climate it has hitherto been unknown. Outbreak of plague as an epidemic is favoured by dirt, particularly by fæcal contamination of the soil. Secondly, it is favoured by warmth of the external air and of dwellings. It usually begins in spring, and disappears when cold weather sets in; but is also stopped by extreme heat.

Overcrowding, dirt, and poverty greatly encourage its development. At Malta in 1813 it was noticed, according to Dr Milroy, to be far less common in the upper stories of the lofty houses of Valetta than in the basements. The higher parts of a district sometimes escape when the lower are infected with the plague; thus it is said that the citadel of Cairo and the village of Alem Dag, near Constantinople, used to remain free, although communications with the cities below were not interrupted. It seems less apt to occur in persons over fifty years old than in those who are younger, and at least in English epidemics affected more adults than children. In the two most recent epidemics, in Hong Kong, 1894, and in Bombay, 1897 (both within the tropics), the violence of the disease was confined to the native quarters, and few Europeans caught it; a fact, no doubt, due to the want of cleanliness in the former, for many of those who undertook the work of cleansing and visiting died, like Captain Vesey of the Shropshire Regiment at Hong Kong, and my friend Dr Manser at Bombay, who fell victims to the pestilence they were combating.

Treatment.—There is no plan of treatment, nor is there any kind of drug, which is proved to be of use in this terrible disease: prevention, not cure, is the way to meet it with success. It may, therefore, be well that bleeding and sweating failed in the hands of the best physicians in the seventeenth century, and that quinine and brandy have failed in India and

China. Nor does active treatment of the buboes seem to do more than add to the patient's sufferings.

Two specific methods of dealing with this formidable disease have been devised: the first on the model of antitoxic serum in Diphtheria, the second on the model of vaccination in Variola.

Treatment by injection of serum from a horse rendered immune to the plague by repeated injections of the virus has been tried by Yersin and Calmette, and is said not only to protect rabbits from the effect of the virus, but also to antagonise what has already been administered.

Dr Haffkine, at Bombay, reports the injection of prophylactic fluid, *i. e.* inoculation with the attenuated virus, in a large prison at Byculla (June, 1897). The result was two cases and no deaths, compared with twelve cases and six deaths among prisoners not inoculated. Subsequent experience has confirmed these results, but one objection not yet obviated is the severity of the symptoms which the inoculation causes.

Prognosis.—The fatality of the plague in the seventeenth century exceeded that of all other diseases. It usually destroyed from 70 to 90 per cent. of those whom it attacked; very seldom less than 60 per cent. Moreover it often carried off half the population of a town or of a district in which it prevailed, and completely rooted out whole families, so that no survivor remained. The "Riley Graves," near Eyam, in Derbyshire, still tell of the fate that befell two families in that place during the epidemic of 1666: one, that of the Talbots, consisting of seven persons, was utterly eradicated within twenty-five days; the other, that of the Hancocks, lost seven out of eight members, the only one left being the mother.*

It is probable that part of the excessive mortality from this disease was due to the fact that, instead of isolating each case at the earliest possible moment, the ancient practice was to close up every infected house, preventing both ingress and egress, and confining the sick and the healthy together.

The outbreaks of the plague in the present century, though severe and terrible in their mortality, have differed much from such invasions as the Black death in Florence or in Norfolk in the fourteenth century, from the great Plague of 1665 in London, or that of 1720 in Marseilles. The mortality has not been so large among those affected, and the majority of cases have been among the poor and dirty crowds of Canton, Hong Kong, and Bombay; while the better cared for and cleanlier Europeans and also the well-to-do natives have usually escaped. The exceptions have been chiefly among those whose duties as physicians or sanitary inspectors or Government officials led them into unusually close contact with the infected dwellings. The most favourable cases have been observed to occur towards the decline of an epidemic. The prognosis of Pestis minor is uniformly good.

Both in the history of past epidemics and in recent experience it has been noticed that the Plague has not an unlimited duration like Typhus, Scarlatina, Enterica, and Diphtheria. After gradually increasing in the number of cases, it remains at its height of severity and prevalence for some months, and then gradually abates and leaves the district for several years.

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* On the church wall at Penrith is inscribed "A.D. 1598, *ex gravi peste, quæ regionibus hisce incubuit, obierunt apud Penrith* 2260, *Kendal* 2500, *Richmond* 2200, *Carlisle* 1196. *Posteri avertite vos et vivite.*" (Quoted by Dr Allbutt.)

Prophylaxis.—There is every reason to believe that the improvement in the hygienic conditions of Western Europe during the last two centuries has been the chief reason why the plague no longer appears among us, and that Hirsch is wrong in attributing this result mainly to the system of quarantine. And this is fortunate, for it is certain that at the present day, if the disease were again to approach our shores, it would be impossible to maintain that system effectually. Liebermeister cites in detail the proceedings that were adopted to prevent the extension of the plague from Noja in Italy in 1815. The town was surrounded by two deep ditches, a triple military cordon was drawn round it, the soldiers had orders to shoot down anyone who attempted to pass, and no articles were allowed to be sent out except letters which had been first dipped in vinegar. The efficacy of such measures is illustrated by the following instance. Dr Aitken states that during an epidemic at Marseilles a large nunnery was "shut up," with the result that all the inmates remained free, although there was an infirmary on one side for those ill of the disease, and a burying-ground on the other side for those who died of it. This writer, however, speaks of quarantine as a barbarous and unnecessary nuisance; and it is well known that the best English authorities hold similar opinions. Indeed, the chief reason why our Government from time to time enforces quarantine regulations, in our possessions in the East and elsewhere, is that if we did not do so, other countries would at once refuse intercourse with our more exposed ports, on the mere chance of our becoming a source of danger to them.

The true method of meeting plague is by destroying the suitability of the soil. When houses are kept clean, and the ground is no longer defiled by excrement, pestilence will be as obsolete in Hong Kong and Bombay as in London and Marseilles.[*]

THE SWEATING SICKNESS.—A few words may be added with reference to another disease more obsolete than the Plague, which afflicted England at the end of the fifteenth and half the sixteenth century.

The *sudor anglicus*, as it was called, was an infective febrile epidemic disorder characterised by a rapid course, profuse perspiration, and limitation to this island, or rather to its inhabitants, for it was said to single out our countrymen wherever they might be abroad.[†] It first appeared in 1485, and was supposed to have been introduced by the motley host which Henry VII led to Bosworth field; for the first recorded cases happened between his landing at Milford Haven, on August 7th, and the battle a fortnight afterwards. On the 28th it broke out in London, about the same time at Oxford, and spread over the whole country with a great mortality. It ended as suddenly as it began in December of the same year. The second epidemic was in 1507, the third in 1517, the fourth in 1528, and this alone spread to Hamburg, and thence to all northern and central Europe, devastating Scandinavia, Poland, Hungary, and Switzerland, but sparing France, Italy, and Spain, and most of Germany. The fifth and last epidemic in 1551 was confined to England, Calais (then still an English town), and Flanders. This was described by Dr John Kaye (better known as Caius); it, like previous epidemics, is said not to have invaded Scotland or Ireland.

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[*] In re-writing this article I have followed that by Dr Milroy in 'Reynolds' System.' on which Dr Fagge's chapter in the first edition of this volume was based; Dr Payne's article in 'Allbutt's System;' Dr Lubbock's article from 'Hygiene and Diseases of Warm Climates' (1893); and Dr Cantlie's account of the plague in Hong Kong, 'Brit. Med. Journ.,' vol. ii, 1894. It is right to say that the writer has never seen a case of Relapsing fever or of Oriental plague.

[†] Dr Payne remarks that this was either because they brought the disease with them or if settled in foreign parts they were merchants in communication with England.

The onset of the "English sweat" was marked by rigors, headache, and pains in the back and limbs; after a short cold stage like that of ague, there followed sudden and profuse diaphoresis, with rapid pulse, thirst, and no doubt raised temperature. No exanthem is noted, and there is no mention of sudamina. The course of the malady was remarkably short; fatal cases ended in a few hours with stupor and signs of collapse; but after twenty-four hours the danger was nearly past. Relapses, however, sometimes occurred.

Various guesses as to the ætiology of this curious disease were made; but it seems pretty certain, first, that it was not due to any of the supposed causes—the English "constitution," dirt and squalor, moist seasons, British fogs, Continental heat, poverty or luxurious living; and secondly, that it was (what Caius denied) an infectious disorder, spreading by human intercourse.

Dr Payne, in an able article on the subject in the last edition of the 'Encyclopædia Britannica,' argues that this strange epidemic was not really an isolated phenomenon, but closely related to "Miliary fever" (*Schweiss-freisel*, *suette miliaire*, or Picardy sweat), an epidemic disease of short duration marked by fever, sweating, and sudamina, not very uncommon in France, Italy, and Germany during the last and the present century, and apparently endemic in Northern France. Of this disorder, which appeared in 1718, there is an interesting account in Heckel's 'Epidemics of the Middle Ages,' translated by the younger Dr Babington for the Sydenham Society in 1844 (p. 315).

If this miliary fever of Picardy is really identical with the *Sudor Anglicus*, we have a close parallel to the endemic *pestis minor* and the epidemic plague, as well as to endemic and epidemic cholera, and endemic and epidemic measles.

CHOLERA

Hæc igitur subita clades nova pestilitasque
Aut in aquas cadit aut . . . . .
LUCRETIUS.

History—Name—Onset and early symptoms—stage of collapse—stage of reaction—the course of this stage—So-called "cholera-typhoid"—Cholerine—Complications—Anatomy—Pathology—Ætiology—contagion—the Comma Bacillus of Koch—propagation of the disease—Diagnosis—Prognosis—Prevention—Treatment.

Synonyms.—Epidemic cholera, Asiatic or Indian cholera, spasmodic, serous, malignant, or blue cholera; Cholera pestifera, Cholera morbus, Passio cholerica. In India the word *morxi*, used by the Portuguese in the sixteenth century, probably denoted cholera. The French in the Deccan called it "*mort de chien*." The native term in Hindoostan is *haiza*; in China, *Ho-loan*.

Definition.—An acute specific disease, propagated by infection conveyed by water, running a short course, and most dangerous when appearing in an epidemic form.

History.—Like Typhus and like Plague, Cholera is an endemic disease in certain parts of the world, which from time to time has made more or less wide, and more or less severe epidemic incursions. Like them, its terrors have in recent times greatly abated, and its banishment from Western Europe may be regarded as complete, and may be hoped to be final.

The best ascertained home of cholera is Bengal, where it certainly existed during the eighteenth century, and probably from the earliest times. Mr Macnamara cites numerous notices of cholera epidemics both before and after the appearance of the English in India. In the winter of 1817-18 it appeared as a new and fatal disease in the camp of the Marquis of Hastings, then engaged in the Mahratta war on the banks of the Sind. Cholera was recognised the same year at Jessore in Bengal, and there also was regarded as an unheard-of pestilence. During the next few years (to 1823) it spread slowly over a large part of Asia.

The first appearance of Cholera in Europe was in 1830, when it spread from what was apparently its endemic seat in Northern Persia to Russia, and thence to Germany. Its first outbreak at Paris occurred in the midst of the carnival of 1831, and everywhere it came with such suddenness and was so rapidly fatal that it created as much terror as the plague in former times. In October, 1831, it reached Sunderland, and London in the following January. After spreading over the whole of

Europe the new pestilence crossed the Atlantic, and was very fatal in the cities of the United States and of Brazil. This epidemic was not over until 1837.

The second European epidemic was in 1847-8, when the mortality in London was very great. The well-known surgeon, Aston Key, was one of the victims.

The third, like the other invasions, began in the East and slowly spread to England in 1853. It was one cause of mortality during the Crimean war, particularly in the French and Sardinian armies, and was very destructive in America. In London its effects, though severe, were remarkably confined to certain districts.

The fourth epidemic was in 1865-6, and the fifth in 1870-73. The next important invasion of Europe by Cholera was confined to Italy, the south of France, and Spain (1884-6). At Naples and at Marseilles it was very severe.

The last epidemic (1891-3) spread rapidly from India, by Persia, to Central Europe. It was most severe at Hamburg.

Cholera has been endemic in China since 1820, and in 1858 invaded Japan, where there was a severe epidemic in 1879.

Certain regions have hitherto escaped Cholera: Iceland, the Orkneys in Europe, Chili and Peru in the New World, the Cape Colony in Africa, and the whole of the Australian continent, beside many islands.

Name.—The term "cholera" does not seem to occur in the Hippocratic writings, but it is used by Aretæus and by Alexander of Tralles. The description by the former writer ('De causis et signis acutorum morborum,' lib. ii, cap. v) refers to severe diarrhœa with vomiting; the evacuations are said to be first fæcal, then mucous (φλεγματώδεα, *pituitosa*), and lastly bilious (χολώδεα); pains, cramps, scanty urine, feeble and frequent pulse are given as the symptoms which accompany this disease; none of them characteristic of Asiatic Cholera, as distinct from severe diarrhœa.

The word χολέρα is usually and probably rightly derived from χολή, and means bilious diarrhœa. So Celsus understood it: *bilis supra infraque erumpit, primum aquæ similis, deinde ut in ea recens caro lota esse videatur, interdum alba, nonnunquam nigra vel varia. Ergo eo nomine morbum hunc* χολέραν *Græci nominarunt* (lib. iv, cap. xi).*

The name applied to the new disease was extremely inappropriate, for Indian cholera is marked by an absence of bile in the matters vomited or discharged from the bowels. The epithet "Asiatic," or more properly "Indian," was applied to the new malady by way of distinction from so-called "English Cholera," or "choleraic diarrhœa." But the term cholera should be applied to the epidemic disorder alone.

Course.—After exposure to the contagion of the disease there is a short period of *incubation*, always under ten days, most often perhaps three or four days, and sometimes not more than twelve or twenty-four hours. Goodeve cites an instance in which a detachment of sepoys, on their march from one place free from cholera to another, passed through a

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* Alexander Trallianus gives another etymology: *Intestina vero* χολάδες *veteres appellabant ut etiam Homerus testatur. . . . Hujus gratia etiam affectum* χολέραν *nuncuparunt* (lib. vii, cap. xvii). Hesychius's Lexicon (quoted by Liddell and Scott) gives a second explanation of the word as an application of χολέρα, "the gutter of a roof down which the rain is discharged:" but this is nonsense.

[illegible] was attacked after [illegible]

[illegible] the earliest symptoms is usually [illegible] attended with griping pain and a sense of [illegible] vertigo and [illegible] at the London Hospital [illegible] that she was attacked with [illegible] that she went down to her [illegible] were slight diarrhoea for three [illegible] or more in the [illegible] have been suffering [illegible] the ever-pursuing [illegible] stage [illegible] in which [illegible] the aspect of the [illegible] symptoms appeared. The [illegible] few hours to two or [illegible]

[illegible] and then the disease [illegible] very often—according to [illegible] in the early morning, perhaps [illegible]

[illegible] collapse without the [illegible] purging. These cases, called [illegible] for after death the [illegible] rice-water secretion.

[illegible] is violent *purging*: the con[illegible] and the discharge [illegible] the water in which rice has [illegible] spoken of as "rice-water stools" [illegible] whitish-grey material, which [illegible] and immense [illegible] At one time it was [illegible] were present, but they appear not [illegible] they are abundant [illegible] The specific gravity of the liquid is [illegible] alkaline reaction, and [illegible] with a very small quantity of albumen. [illegible] Goodeve speaks of the patient as almost [illegible] two or three hours, and sometimes voiding [illegible] He remarks that when all that is passed is [illegible] the bile contained in what was first passed [illegible] yellowish colour; or sometimes the tint is pinkish from the admixture of blood. There is often no pain whatever in the bowels, but some patients complain of more or less griping.

After an interval, or occasionally at the very commencement of the attack, *vomiting* sets in; the fluid rejected from the stomach (unless mixed with the food) is pale and watery, being in fact identical with the rice-water liquid; it may even have a still lower specific gravity of 1002 to 1005, due probably to a large admixture of water taken in during the attack. It is often poured out of the mouth suddenly and with great force.

Another early symptom is severe *cramp* in the muscles of the feet, in the calves of the legs, and sometimes in the thighs, hands, chest, or abdomen. This usually comes on at intervals, and lasts for a few minutes at a time. It is an agonising pain, and makes the patient shriek and start up from the bed. Lebert says that cramps were absent in only a third of the cases observed in the epidemic at Zürich in 1855.

The stage of collapse.—The severe symptoms just described are speedily followed by a remarkable condition, known as "cholera collapse," or the "algide stage." It commonly appears within six or seven hours of the commencement of the purging, and often earlier still. Indeed, in some exceptional cases, as mentioned above, the patient dies collapsed before there has been either vomiting or purging, and the rice-water fluid is found accumulated in the bowels after death. The essential sign of this state is a failure of the circulation, beginning at the periphery, but afterwards affecting parts less distant from the heart. The pulse at the wrist becomes more and more feeble and thread-like, until it is no longer perceptible, even in the brachial artery; on auscultation, according to Lebert, the second sound of the heart may be inaudible while the first sound is still heard. The surface of the body becomes cold, livid, and shrunken, and the skin is often covered with a profuse sweat. The hands feel like ice, and look shrivelled as though they had been long soaked in water. The features have a leaden hue, the eyeballs lie deep in their sockets, the nose is pointed, the face meagre, the tongue cold, and even the breath no warmer than the surrounding air.

According to Goodeve, a thermometer placed in the mouth indicates from 79° to 88°. In the axilla, however, the temperature is seldom below 93° or 94°, as was shown by careful observations made at the London Hospital in 1866 by Mr F. M. Mackenzie; and in the rectum, or in the vagina, the temperature rises through the period of collapse, reaching 100° or 102°, or even 104·4°.* Mr Mackenzie observed that a severe attack of cramp raised the axillary temperature as much as 2° F.

Is the elevated temperature in the interior of the body due to a mere accumulation of heat from deficient loss, or is there a positive shifting upwards of the point to which heat regulation is set, as in true pyrexia? A strong argument against the latter view is that during reaction, as we shall presently see, when the peripheral circulation becomes restored, the internal temperature falls and is actually below normal. Moreover the collapse of cholera is essentially different from the initial rigors of acute specific diseases. One distinction on which Hutchinson insisted ('Lond. Hosp. Rep.,' vol. iii) is that in choleraic collapse the pupils are of natural size, whereas in rigor they are widely dilated.

The breathing during collapse is increased in frequency, being at the rate of 24, 30, or even 40 in the minute; and there is often a sensation of dyspnœa. The heart's action, however, usually remains at about 100.

There is great muscular weakness. The voice becomes feeble and whispering, or inaudible. In some cases the patient has been known to sit up, or even walk across the room, very shortly before his death, but as a

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* Mr Simon, in discussing these facts in his 'Ninth Report,' inferred that the choleraic affection of the bowels is a "heat-making" process. But there seems to be no reason for doubting that the heat is generated in the muscles and in the glandular organs, just as it is in health.

CHOLERA

> **Hæc igitur subito clades nova pestilitasque**
> **Aut in aquas cadit aut . . . . .**
> **Lucretius.**

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History.—Like Typhus and like Plague, Cholera is an endemic disease in certain parts of the world, which from time to time has made more or less wide, and more or less severe epidemic incursions. Like them, its terrors have in recent times greatly abated, and its banishment from Western Europe may be regarded as complete, and may be hoped to be final.

The best ascertained home of cholera is Bengal, where it certainly existed during the eighteenth century, and probably from the earliest times. Mr Macnamara cites numerous notices of cholera epidemics both before and after the appearance of the English in India. In the winter of 1817-18 it appeared as a new and fatal disease in the camp of the Marquis of Hastings, then engaged in the Mahratta war on the banks of the Sind. Cholera was recognised the same year at Jessore in Bengal, and there also was regarded as an unheard-of pestilence. During the next few years (to 1823) it spread slowly over a large part of Asia.

The first appearance of Cholera in Europe was in 1830, when it spread from what was apparently its endemic seat in Northern Persia to Russia, and thence to Germany. Its first outbreak at Paris occurred in the midst of the carnival of 1831, and everywhere it came with such suddenness and was so rapidly fatal that it created as much terror as the plague in former times. In October, 1831, it reached Sunderland, and London in the following January. After spreading over the whole of

Europe the new pestilence crossed the Atlantic, and was very fatal in the cities of the United States and of Brazil. This epidemic was not over until 1837.

The second European epidemic was in 1847-8, when the mortality in London was very great. The well-known surgeon, Aston Key, was one of the victims.

The third, like the other invasions, began in the East and slowly spread to England in 1853. It was one cause of mortality during the Crimean war, particularly in the French and Sardinian armies, and was very destructive in America. In London its effects, though severe, were remarkably confined to certain districts.

The fourth epidemic was in 1865-6, and the fifth in 1870-73. The next important invasion of Europe by Cholera was confined to Italy, the south of France, and Spain (1884-6). At Naples and at Marseilles it was very severe.

The last epidemic (1891-3) spread rapidly from India, by Persia, to Central Europe. It was most severe at Hamburg.

Cholera has been endemic in China since 1820, and in 1858 invaded Japan, where there was a severe epidemic in 1879.

Certain regions have hitherto escaped Cholera: Iceland, the Orkneys in Europe, Chili and Peru in the New World, the Cape Colony in Africa, and the whole of the Australian continent, beside many islands.

Name.—The term "cholera" does not seem to occur in the Hippocratic writings, but it is used by Aretæus and by Alexander of Tralles. The description by the former writer ('De causis et signis acutorum morborum,' lib. ii, cap. v) refers to severe diarrhœa with vomiting; the evacuations are said to be first fæcal, then mucous (φλεγματώδεα, *pituitosa*), and lastly bilious (χολώδεα); pains, cramps, scanty urine, feeble and frequent pulse are given as the symptoms which accompany this disease; none of them characteristic of Asiatic Cholera, as distinct from severe diarrhœa.

The word χολέρα is usually and probably rightly derived from χολή, and means bilious diarrhœa. So Celsus understood it: *bilis supra infraque erumpit, primum aquæ similis, deinde ut in ea recens caro lota esse videatur, interdum alba, nonnunquam nigra vel varia. Ergo eo nomine morbum hunc* χολέραν *Græci nominarunt* (lib. iv, cap. xi).*

The name applied to the new disease was extremely inappropriate, for Indian cholera is marked by an absence of bile in the matters vomited or discharged from the bowels. The epithet "Asiatic," or more properly "Indian," was applied to the new malady by way of distinction from so-called "English Cholera," or "choleraic diarrhœa." But the term cholera should be applied to the epidemic disorder alone.

Course.—After exposure to the contagion of the disease there is a short period of *incubation*, always under ten days, most often perhaps three or four days, and sometimes not more than twelve or twenty-four hours. Goodeve cites an instance in which a detachment of sepoys, on their march from one place free from cholera to another, passed through a village where it was raging; one of the sepoys was attacked after forty hours, and fresh cases appeared subsequently.

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* Alexander Trallianus gives another etymology: *Intestina vero* χολάδες *veteres appellabant ut etiam Homerus testatur. . . . Hujus gratia etiam affectum* χολέραν *nuncuparunt* (lib. vii, cap. xvii). Hesychius's Lexicon (quoted by Liddell and Scott) gives a second explanation of the word as an application of χολέρα, "the gutter of a roof down which the rain is discharged:" but this is nonsense.

The invasion of cholera may be either gradual or sudden.

When the disease sets in gradually, the earliest symptom is generally "premonitory" diarrhœa, attended with griping pain and a sense of exhaustion. In some cases depression of spirits, headache, vertigo, and noises in the ears are present during this period. At the London Hospital in 1866 a nurse was doing her work as usual when she was attacked with "singing in her ears," and nausea; so that she lay down hoping that her symptoms would pass off, but they were quickly succeeded by those of cholera. Another patient there complained not only of noise in the head, but of the sound of his own voice; and a third kept shifting his head every few minutes, in the attempt to escape from the ever-pursuing noise. The countenance of a patient during the premonitory stage is often pallid, anxious, and sorrowful. Annesley cites a case in which the approach of cholera was suspected mainly from the aspect of the patient nine hours before the characteristic symptoms appeared. The duration of the premonitory symptoms varies from a few hours to two or three days.

In many instances this stage is altogether absent, and then the disease develops itself with startling suddenness. It very often—according to Lebert, in more than half the cases—begins in the early morning, perhaps waking the patient up from sleep.

Occasionally death occurs with symptoms of collapse, without the premonitory stage, and without vomiting or purging. These cases, called *cholera sicca*, only differ from the rest in severity, for after death the intestines are found full of the characteristic rice-water secretion.

The first symptom of the developed disease is violent *purging;* the contents of the bowels are rapidly swept out in a fluid form, and the discharges soon become almost colourless, like whey, or like water in which rice has been boiled, so that they are commonly spoken of as "rice-water stools." On standing, this liquid deposits a loose whitish-grey material, which consists of mucous flocculi, containing numerous leucocytes and immense numbers of granules, including various bacteria. At one time it was thought that columnar epithelial cells were present, but they appear not to occur in rice-water discharges during life, although they are abundant in the intestinal contents after death. The specific gravity of the liquid is from 1006 to 1013; it has a neutral or slightly alkaline reaction, and chiefly contains chloride of sodium, with a very small quantity of albumen. So profuse is the flow that Goodeve speaks of the patient as almost filling the pan of a night-stool in two or three hours, and sometimes voiding several pints or even quarts. He remarks that when all that is passed is collected in the same vessel, the bile contained in what was first passed may give it a yellowish colour; or sometimes the tint is pinkish from the admixture of blood. There is often no pain whatever in the bowels, but some patients complain of more or less griping.

After an interval, or occasionally at the very commencement of the attack, *vomiting* sets in; the fluid rejected from the stomach (unless mixed with the food) is pale and watery, being in fact identical with the rice-water liquid; it may even have a still lower specific gravity of 1002 to 1005, due probably to a large admixture of water taken in during the attack. It is often poured out of the mouth suddenly and with great force.

Another early symptom is severe *cramp* in the muscles of the feet, in the calves of the legs, and sometimes in the thighs, hands, chest, or abdomen. This usually comes on at intervals, and lasts for a few minutes at a time. It is an agonising pain, and makes the patient shriek and start up from the bed. Lebert says that cramps were absent in only a third of the cases observed in the epidemic at Zürich in 1855.

The stage of collapse.—The severe symptoms just described are speedily followed by a remarkable condition, known as "cholera collapse," or the "algide stage." It commonly appears within six or seven hours of the commencement of the purging, and often earlier still. Indeed, in some exceptional cases, as mentioned above, the patient dies collapsed before there has been either vomiting or purging, and the rice-water fluid is found accumulated in the bowels after death. The essential sign of this state is a failure of the circulation, beginning at the periphery, but afterwards affecting parts less distant from the heart. The pulse at the wrist becomes more and more feeble and thread-like, until it is no longer perceptible, even in the brachial artery; on auscultation, according to Lebert, the second sound of the heart may be inaudible while the first sound is still heard. The surface of the body becomes cold, livid, and shrunken, and the skin is often covered with a profuse sweat. The hands feel like ice, and look shrivelled as though they had been long soaked in water. The features have a leaden hue, the eyeballs lie deep in their sockets, the nose is pointed, the face meagre, the tongue cold, and even the breath no warmer than the surrounding air.

According to Goodeve, a thermometer placed in the mouth indicates from 79° to 88°. In the axilla, however, the temperature is seldom below 93° or 94°, as was shown by careful observations made at the London Hospital in 1866 by Mr F. M. Mackenzie; and in the rectum, or in the vagina, the temperature rises through the period of collapse, reaching 100° or 102°, or even 104·4°.* Mr Mackenzie observed that a severe attack of cramp raised the axillary temperature as much as 2° F.

Is the elevated temperature in the interior of the body due to a mere accumulation of heat from deficient loss, or is there a positive shifting upwards of the point to which heat regulation is set, as in true pyrexia? A strong argument against the latter view is that during reaction, as we shall presently see, when the peripheral circulation becomes restored, the internal temperature falls and is actually below normal. Moreover the collapse of cholera is essentially different from the initial rigors of acute specific diseases. One distinction on which Hutchinson insisted ('Lond. Hosp. Rep.,' vol. iii) is that in choleraic collapse the pupils are of natural size, whereas in rigor they are widely dilated.

The breathing during collapse is increased in frequency, being at the rate of 24, 30, or even 40 in the minute; and there is often a sensation of dyspnœa. The heart's action, however, usually remains at about 100.

There is great muscular weakness. The voice becomes feeble and whispering, or inaudible. In some cases the patient has been known to sit up, or even walk across the room, very shortly before his death, but as a rule he is unable to rise from the recumbent position. There is often, however, great restlessness of the limbs with tossing and jerking.

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* Mr Simon, in discussing these facts in his 'Ninth Report,' inferred that the choleraic affection of the bowels is a "heat-making" process. But there seems to be no reason for doubting that the heat is generated in the muscles and in the glandular organs, just as it is in health.

The mental state is generally, from the first, one of indifference rather than of anxiety; and in some cases it is only when the cramps cause the sufferer to cry out that he seems to be conscious of his condition. But he can easily be roused to understand what is said to him, and may answer rationally. Coma only sets in towards the last, if at all.

When collapse has developed in a marked form, the purging ceases or is much diminished. Probably what is now passed had been poured out into the bowel at an earlier period; Goodeve describes it as often containing gelatinous mucus-like masses. The stomach remains very irritable. There is the most intense thirst; the patient craves for water, and drinks it with eagerness, only to vomit immediately afterwards. Another distressing symptom is a sense of burning heat at the epigastrium, often accompanied by great tenderness, and sometimes by hiccup. The abdomen is commonly retracted as in meningitis.

One of the most marked symptoms of collapse is *suppression of urine*, which seems often to be present from the first. This arrest of the renal secretion in cholera is no doubt a result of the defective flow of blood through the kidneys. Hermann and Cohnheim have independently shown that precisely the same thing occurs in animals when the renal arteries are compressed or ligatured.

Fatal event.—Cholera is often fatal during the stage of collapse, between twelve and twenty-four hours after the commencement of the attack, but sometimes earlier, and sometimes during the second day.

Lebert mentions that before death the eyes may become dry and the cornea slightly opaque. The internal temperature has often been found to rise after life has become extinct; and spasmodic twitchings and quiverings of the muscles have sometimes been observed within the first half-hour after death. Nay, the limbs may actually move; in a case at the London Hospital in 1866 the elbow became raised about three inches above the level of the chest, across which it was lying.

Reaction.—Sometimes, however, even extreme collapse is recovered from. In that case reaction usually begins to develop itself at the end of twenty-four or forty-eight hours. Goodeve remarks that the subsidence of restlessness and jactitation is a favourable sign. The patient dozes quietly with easy respiration; then a flickering pulse at the wrist is detected, which gradually becomes more distinct, the superficial veins on the back of the hands begin to fill, the surface is felt to be less cold, the features look less sunken, and the colour begins to return. As a rule, the improvement occurs slowly and step by step. But the older Indian physicians spoke of such cases as one in which a man, who was in perfect collapse on Monday, was standing at his door on Wednesday.

According to Mr F. M. Mackenzie, the temperature during reaction, when the pulse has fairly recovered, is usually about 97° in the rectum or the vagina, and 96° in the axilla ('London Hosp. Reports,' vol. iii). But in some cases at the London Hospital in 1866 it was observed that certain parts of the surface conveyed to the hand a sensation of burning pungent heat. Another point noticed at the same time was that the pulse often fell to 54, or even to 45, in patients who were doing perfectly well. Lebert, on the other hand, speaks of the pulse as remaining at about 100. It often becomes full and bounding, and may be dicrotic. The patient's aspect is peculiar. The cheeks usually present sharply defined patches of dusky redness; the conjunctivæ are deeply injected, the eyes half closed with the corneæ turned upwards, the expression heavy and vacant.

The period of reaction is by no means free from danger. Sometimes a *relapse* occurs; purging, vomiting, and exhaustion set in again, and end fatally. In other cases the reaction is said to be *imperfect*. The pulse, after improving up to a certain point, remains weak, and the surface of the body continues to be colder than natural; the bowels are still relaxed and the evacuations are watery, although more or less coloured with bile; there is no appetite, and the patient is depressed and drowsy. Sometimes the most prominent symptom is irritability of stomach, attended with thirst and burning sensations along the œsophagus and at the epigastrium. Goodeve also mentions sleeplessness, lasting for two or three days or more, and causing great discomfort to the patient.

Occasional febrile exanthem.—In some cases slight pyrexia occurs at this stage, and is attended with the appearance of a bright scarlet *rash*, a roseola, or an urticaria. Sometimes it nearly resembles the eruption of scarlatina. Mr Mackenzie found in 1866 that it was always accompanied by a rise of temperature, both external and internal. In one instance the former was 101·6°; the latter 102·4°. The backs of the hands and the forearms are its favourite seats, but it may also cover the trunk, and even be seen on the face. In the museum of Guy's Hospital we have excellent models of this choleraic roseola made during the epidemic of 1854 (models 46—50). It seldom comes out until a week or ten days after the commencement of the attack, and in one case recorded by Wilks ('Guy's Hospital Reports,' third series, vol. ix, p. 345) it did not appear until the seventeenth day. It lasts two or three days, and may be followed by desquamation. It is more often observed in young patients than in old, and is particularly well marked in children. As a rule, the cases in which it occurs end in recovery.

Urine.—In the reaction of cholera the most important condition to watch is that of the kidneys. We have seen that during collapse there is generally complete suppression of urine; if any is secreted, it is apt to be, even at that period, albuminous. When reaction sets in, the suppression often continues for several hours, or even for two, three, or four days. According to Buhl, if it lasts up to the sixth day the case is hopeless. Sometimes, however, when the renal function has been re-established, the patient fails to void the urine in the bladder. The hypogastric region must therefore always be carefully examined, and, if necessary, a catheter must be passed. The instrument should be new and aseptic, for cystitis and pyelitis have often been observed in cases fatal at an advanced stage. In a woman who died in Guy's Hospital during the epidemic of 1866 there was suppurative nephritis, which appeared to have arisen by extension from the bladder.

The urine which is first secreted during the reaction stage of cholera is described by Goodeve as high-coloured, acid, and strong-smelling. As to its specific gravity, there are some discrepancies in the recorded observations. At the London Hospital in 1886 it was found to range from 1005 to 1017, the average being 1006. Wyss, however ('Arch. d. Heilk.,' 1868), found that the density of the first urine passed was from 1012 to 1033, the average being 1020. It was only at a later period, when the quantity was more abundant, that it fell to 1010 or lower. The h

amount of urea and salts is much diminished, and albumen is generally present, sometimes with blood and hyaline casts. Lebert says that the occurrence of albuminuria at this stage is constant, but his statement does not accord with the experience of English observers. At the London Hospital in 1866 albumen was found "in about half the cases examined." As a rule, the urine continues to be coagulable during from two to seven days. The albuminuria in cholera was formerly regarded as due to parenchymatous nephritis, and comparable with the acute renal affection that follows scarlet fever or diphtheria. But it is probably the direct result of the ischæmia which exists during the stage of collapse, the view upheld by Bartels and Cohnheim.

In cases fatal before reaction has set in, the kidneys are found small, tough, dry, and of a dark brown or livid colour. At a later stage they are much enlarged—Moxon speaks of them as weighing up to fifteen or sixteen ounces,—soft, greyish white, or yellowish. The epithelium very early becomes cloudy, opaque, or fatty—a change analogous to the "coagulation-necrosis" which Litten has found to occur in the renal epithelium of rabbits after arrest of the arterial blood-current through the kidneys. The Malpighian tufts and their capsules present no morbid appearances in cases of cholera, and the renal affection of cholera appears never to form the starting-point of chronic Bright's disease. This fact accords with the opinion that it is to be distinguished from the various forms of nephritis.

Typhoid condition in reaction.—The stage of reaction often presents a grave complication, known as "cholera-typhoid,"—a bad name, for it has nothing to do with enteric fever. This sets in five or six days after the commencement of the attack. It is attended with great prostration, headache, giddiness, and stupor. The face is at first flushed, but afterwards becomes pale. The temperature does not rise more than 2° or 3°. The tongue becomes dry, and the pulse slow, weak, and small. There is often a remarkable rigidity of the muscles, so that the patient resists any attempt to open his mouth, to separate his eyelids, or to straighten his elbow. Convulsions are seldom observed. Death by coma usually occurs in about forty-eight hours, but sometimes the typhoid state continues for several days, and may even end in recovery.

Frerichs and other writers regarded this complication of cholera as due to uræmia; but this is only occasionally present, and is marked by convulsions and other unmistakable symptoms.[*]

Slighter forms.—There are minor cases of cholera which are not uncommon, although their frequency cannot be positively stated, because they are often allowed to run their course without being brought under medical observation. One of them is often called "choleraic diarrhœa." It is identical with the "premonitory diarrhœa" already described, except that it subsides after a time and does not pass on into cholera. It often begins suddenly, after a chill, or after the patient has eaten unwholesome food. There are three or four or more evacuations of yellow fæcal matter daily, with some pain, and perhaps with slight cramps. A more severe form has been termed "cholerine;" it is attended with vomiting, with cramps, with a transitory disappearance of bile from the stools, and even with some degree of coldness of the limbs. Lebert says that he has seen it followed by pronounced typhoid symptoms. *Cholera nostras*, applied to cases of severe sporadic summer diarrhœa, is a term frequently used. Whatever view is taken of these conditions, the name cholera, with the qualification of mild or slight or abortive, should only be used when from the presence of the characteristic microbe, or on other grounds, the diagnosis of true "Asiatic" cholera has been made. They may be compared with slight cases of sore throat which are frequent in an epidemic of scarlatina, or to the tonsillitis which simulates diphtheria. In the latter case and in that of cholera the only criterion apart from subsequent course and spread of the disease is the presence of the characteristic microbe.

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[*] Urea has been found in the blood in cases of cholera-typhoid by several observers. Buhl estimated the quantity of it in one instance at ·2 per cent. In some instances, too, there has been observed upon the face and neck an efflorescence of urea in a crystalline form, excreted by the sweat-glands. But Goodeve has observed that in some cases a consecutive fever, attended with typhoid symptoms, occurs independently of uræmia, or at least while urine free from albumen is being secreted. Bartels and E. Wagner drew attention to the same fact, and attributed the pyrexia to enteritis or other local cause.

Complications and sequelæ.—Convalescence from cholera may be retarded by complications. One is an affection of the lower segment of the cornea, generally of each eye. This doubtless results from the part having been exposed and irritated during the stage of collapse. Four or five days after reaction the cornea becomes hazy, or covered with opaque lymph, and ulceration presently follows. Sometimes perforation takes place, but generally, if the patient should recover, the eye is preserved.

The *hyperthermal form* of cholera is marked by a rise of temperature in the rectum during the stage of collapse, followed during reaction by pyrexia, and sometimes by hyperpyrexia. These cases are interesting as showing that cholera is a true specific fever, although the increased temperature is usually concealed.

Occasionally, during the second or the third week, the parotid glands swell or suppurate, and this interferes with swallowing food, often with a fatal result. Sometimes bronchitis, pneumonia, or pleurisy sets in; or sloughing inflammation of the fauces, the bladder, or the sexual organs, or actual gangrene of the scrotum or penis, or the toes, ears, or nose. Bedsores, too, are often a source of danger at this period of the disease.

A friend and former pupil, Dr Prall, gives the writer the following account of cholera as it is seen at Sukkin, in Sindh (1893).

"Cholera is always present to a small amount, and about every five years it becomes epidemic. Last year in such an epidemic we had about 400 cases and about 320 deaths (80 per cent.). . . . Even when by assiduous friction, warmth, and mustard to the skin and internally, I had seen patients come through the stage of collapse, I had the mortification to lose my cases from uræmic convulsions a few hours afterwards, much earlier than I expected them to come on.

"Premonitory diarrhœa seems to be always present. . . . The cramps are most distressing and frequent. M— assured me that the pain of them was intolerable. The vomiting occurs without effort, though the fluid is ejected in a stream with great force. . . . The aspect of cholera is most impressive. The drawn face, the sunken eyes, with large black circles round them, the cold breath, the hoarse, anxious whisper, the shrivelled fingers, and the recurrence of the agonising cramps—all combine to make one of the most fearful sights we can witness; and when we see it all in one whom we have seen a few hours before in robust health, we feel our shocking helplessness!"

Morbid anatomy.—Examination after death throws little light on the pathology of cholera. The bowels commonly contain more or less of the rice-water fluid; this microscopically is found to contain an immense quantity of columnar epithelium, not only loose cells, but coherent masses of considerable size, forming complete casts of the villi. If there is no liquid, the cast-off epithelium forms a soft creamy pulp. At one time the opinion was held that shedding of the intestinal epithelium was the fundamental lesion of cholera; but it is now known to be of *post-mortem* occurrence, for the cells are absent or nearly so from the evacuations passed during life. All that is found is a slightly injected, swollen, and œdematous condition of the intestinal coats, with perhaps some ecchymosis of the valvulæ conniventes. The lymph-follicles in the mucous membrane are also in many cases enlarged, and Lebert says that there may be slight ulceration of Peyer's glands. In a case that occurred in Guy's Hospital in 1854 the lining of the rectum showed a patch which was œdematous and sloughing, with suppuration in the submucous tissue. In another, under the late Dr Guy Babington, there was remarkable œdematous swelling of Peyer's patches, figured in a drawing (No. 1840). The mesenteric glands are sometimes swollen. In 1866 Dr Moxon found, on death after the collapse had been unusually protracted, that a tenacious viscid material like spider's web lay between the coils of intestine, and could be drawn out into long filaments by separating one coil from another. Other points on which he insists are the want of fæcal odour and of the blackening produced by sulphuretted hydrogen, and the absence of gas in the intestine, so that the coils lie in a flabby compact mass at the back of the abdomen.

The blood which is found in the heart and in the great vessels is remarkably thick and tarry: whenever venesection has been practised during life the same condition has been observed. This state of the blood, however, is not peculiar to cholera; for it was well marked in a patient who died in Guy's Hospital in 1871 of acute ulceration of the intestine. It is no doubt a consequence of the drain of fluid from the body, like another result, which has been repeatedly noted, the disappearance of dropsical effusion in a few hours when cholera supervenes. There is decided leucocytosis in cholera, as Virchow long ago observed, and the amount of oxygen is diminished. No microbes have ever been detected in the blood.

In all probability the change in the blood is the cause of the formation during life of ecchymoses, which at the autopsy are often found scattered over the outer surface of the heart, upon the pulmonary pleura, in the pia mater, and elsewhere. All the tissues are peculiarly dry. Cohnheim remarks that the serous cavities in the dead body of a cholera patient have a soapy feel, which is not likely to be forgotten by anyone who has ever put his hand in them; and the spleen, the lungs, the liver, and the other organs are tough and leathery.

The peculiar lightness and the shrunken appearance of the lungs is attributed by Dr Moxon, no doubt correctly, to the dry, empty state of the bronchi, which allows the elasticity of the organs to drive the air out of them more completely than usual after the chest is opened. On the other hand, when death has occurred during reaction, the smaller tubes are often found full of pus, and parts of the lungs may be œdematous, or even in a state of broncho-pneumonia.

The spleen is small and shrunken. The kidneys are dry and hard, and often show ecchymoses. Microscopically the tubes are found full of granular material, and some of them denuded of epithelium.

Pathology.—The whole course and natural history of an epidemic of cholera brings it into close relation with the specific contagious fevers of which plague, typhus, smallpox, and scarlet fever are types; it is like them infectious, though not in the same direct and obvious way; its mode of spreading is rather like that of enteric fever, and it has been classed with that malady and with plague as a "miasmatic-contagious" disease.

In all probability the *contagium vivum* has been discovered in the form of a vibrio, to be presently described. But the pyrexia, which we have hitherto found to be a constant attendant upon the invasion of a swarm of microphytes, is either absent or strangely masked, so that it appears in a slight, inconstant, and equivocal form. Possibly something prevents this virus from reaching the thermotaxic centres of the nervous system; or possibly some thermolytic process prevents its effect in raising the temperature from being manifest. Or it may be that the effect of the microbes is chiefly or entirely local, and that the intestinal disturbance is itself the cause of the thickened blood, the low temperature, and the other symptoms.

We cannot consider cholera as a local inflammation, like erysipelas or diphtheria; for, apart from the absence of pyrexia, the characters of the rice-water liquid are so unlike those of any known inflammatory exudation in any part of the body, that we cannot possibly suppose it to be of that nature. An experiment, however, originally made by Moreau, suggested the clue to its real significance. Physiologists had previously learnt from Thiry how to isolate a portion of the intestine, so as to obtain from it an unmixed succus entericus in small quantity. Next Moreau found that when an isolated loop of intestine has all the nerves in its mesentery cut through, it becomes filled, within a few hours at latest, with an abundant secretion of a thin alkaline yellowish fluid, which is of very low specific gravity, which contains numerous mucous flocculi, but in which there is only a very small quantity of albumen, while the chief salts in it are those of soda.* There is, in fact, a paralytic over-secretion of *succus entericus*, which possesses the property of converting starch into sugar. But Kühne showed that the rice-water liquid of cholera likewise contains a ferment having this property, besides corresponding closely with succus entericus in all other respects. The inference, therefore, seems justifiable that the effusion of cholera is nothing else than the secretion of the intestinal glands, enormously increased in amount. This may probably be due to paralysis of the splanchnic nerves.†

The above view with regard to the nature of the rice-water liquid strongly supports the doctrine held on other grounds by many observers, that the primary action of the cholera virus is upon the centres governing the splanchnic area rather than upon the bowel itself.

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* Moreau's results (published in the 'Comptes rendus,' 1858, p. 554, and in the 'Bulletin de l'Académie de Méd.,' 1870, p. 629) have since been confirmed and extended. See the Reports of the Committee appointed by the British Association, published in the 'Transactions' for 1874, 1875, and 1876; also papers by Dr M. Hay in the 'Journal of Anatomy and Physiology' (vol. xvi, p. 243, to vol. xvii, p. 441), and by Dr Brunton and the present writer in the 'Practitioner' for November, 1884, *et seq.*

† Cohnheim explained the so-called "cholera sicca" (in which rice-water liquid is found in the intestines after death, though none had been voided during life) as due to early exhaustion of the excitability of the intestinal walls; and he refers to the same cause the frequent cessation of vomiting and purging as collapse becomes developed. He admits, however, that the muscular coats of the bowel are not always paralysed in cholera, inasmuch as *post-mortem* invaginations are often found which occurred after or shortly before death.

The phenomena of collapse are adequately explained by ascribing them to the drain of fluid from the body, and to the viscid inspissated condition of the blood which we have seen to result from it. Mr Sedgwick has remarked that cholera collapse closely resembles that which follows cases of perforation of the stomach, of intestinal obstruction, and of other abdominal diseases in which no purging occurs. At Guy's Hospital, during one of the London epidemics, a man actually died of internal strangulation of the bowel who was supposed during life to be suffering from the so-called "cholera sicca." It must, therefore, be supposed that collapse is the result of some disturbance of the abdominal regulating nerves. This disturbance, however, is by no means identical with that which causes syncope. Cholera patients have no sensation of faintness; and even when they have no pulse at the wrist they may be able to sit up or to walk about without sudden failure of the heart's action. Again, the collapse of cholera is very different from rigor. The suppression of urine which occurs in collapse is a part of the general state, since it is observed likewise in cases of acute intestinal obstruction, of perforating ulcer of the stomach, and of arsenical poisoning. Probably no pathologists agree with the late Sir George Johnson in ascribing the phenomena of choleraic collapse to obstruction of the pulmonary circulation. The lungs weigh much less than usual in cases of cholera, but this depends on the diminished volume of the blood generally. During the epidemic of 1866 we entirely failed to observe that the branches of the pulmonary artery and the right side of the heart were distended or gorged with blood when death had occurred even at an early stage. It is true that cholera is attended with a lividity which is wanting when collapse is due to other causes. But the altered constitution of the blood may be fairly supposed to interfere with its due aëration, and the amount of carbonic acid exhaled from the lungs has been shown to be much reduced.

During an attack of cholera absorption is retarded. Magendie is said to have found that when camphor was injected into the rectum, five minutes elapsed before its odour could be detected in the breath, instead of only one minute, as in health; and Lebert states that atropine fails to dilate the pupils when administered by the mouth, though not when injected into the blood.

It is, however, a curious fact that in women who are suckling children when attacked by cholera the secretion of milk continues during collapse. The menstrual flow also goes on unchecked; it may actually set in before reaction is established, if the proper time has come round for its appearance. A sanguineous muco-purulent discharge from the vagina also frequently appears, independently of the catamenia. When the disease seizes upon a pregnant woman the fœtus dies. If the period of gestation is early, abortion takes place; if late, the mother often dies undelivered. In this case it appears to be useless to perform the operation of Cæsarean section, even immediately after her death, for the child has already died during the algide stage. The mental and bodily vigour are often maintained throughout the whole of an attack of cholera in such a way as to show that the circulation still remains active in the brain and in the muscles.

The stage of reaction seems to be what its name implies, and analogous to the hot swollen condition observed in a part when its vessels are allowed to fill with blood after having been kept empty, as in experiments on the ears of rabbits.

Ætiology.—It may now be regarded as certain that the diffusion of cholera from India over Europe is the result of human intercourse.*
When in 1823 the first epidemic entered Russia by Astrachan, and afterwards spread in a north-westerly direction, it was ascribed to some mysterious atmospheric or telluric agent. But its progress was far too slow and halting for such a view to be even hypothetically tenable. Prussia was reached by cholera in 1831; in October of that year it passed from Hamburg to Sunderland, and appeared in London in January, 1832. Shortly afterwards it invaded France *viâ* Calais, and it also spread across the Atlantic to Canada and the United States. In 1833 it appeared in Portugal, passed in an easterly direction through South Europe, and became for the time extinct in 1837. Its course from India to the West rather than to the East followed, no doubt, the general course of traffic. The second epidemic, which had begun in India in 1840, extended to Europe in a manner very like the first, reaching London direct from Hamburg in 1848, and continuing to prevail in England during the following summer. The fourth, which occurred among us in 1866, differed from the others in having entered Europe from Alexandria; it had been carried from India to Arabia and thence to Egypt, partly by coast-trading vessels, but also by Mohammedan pilgrims to and from Mecca. A detailed account of this epidemic, by Mr Netten Radcliffe, appeared in a supplement to Mr Simon's 'Report to the Privy Council' for 1874. The next European epidemic started in June, 1884, at the port of Toulon, and entered Italy a month later at the port of Genoa. The only cases seen in England were in sailors from the Mediterranean at Cardiff and Bristol. The last and widespread epidemic of cholera in 1893 passed from the seaport Hamburg to Hull, Grimsby, and Yarmouth on our eastern coast, and, as in 1884, did not travel far inland.

It is remarkable that cholera, although it has spread so widely, seems to be capable of establishing itself permanently in no country except India, and there only in a particular region.

Like enteric fever, cholera seldom passes directly from a sick person to those who nurse or visit him. A few instances have indeed been recorded, which look as though direct infection did sometimes occur.† But instances of this kind are very rare, and probably admit of another explanation.

The contagium of cholera escapes from the body in the rice-water evacuations, but not in an active state, and becomes toxic only after an interval of from two to four or five days; in other words, the contagium acquires its infective properties out of the body. The conveyance of the disease to animals was first attempted by Thiersch in 1854, and repeated by Burdon Sanderson in 1866. They used mice for the purpose, and more recently guinea-pigs, sucking rabbits, and marmots have been fed on choleraic dejecta. Even human beings have taken cultivations of Koch's vibrio, and been ill afterwards, but they have never proved their point by dying.

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* See the full and interesting accounts of the spread of the several historical epidemics of India by Mr Macnamara in his 'Treatise on Asiatic Cholera,' 1870 and 1876. Also Dr Bellew's 'History of the Cholera in India from 1862 to 1881,' published in 1885.

† Thus, in a paper in the 'Edinburgh Medical Journal' for 1838, the late Sir James Simpson related how certain mendicants brought cholera with them to Bathgate on May 27th, 1832, and how one of the nurses who attended upon them was attacked and died on the 30th. He also cites the case of a woman who, having caught the disease at some iron-works where it raged, and being afraid of being sent to the hospital, went on May 12th to a cottage where her mother lived, four or five miles off: on the 14th the mother fell ill, and died in a few hours. Dr John Snow relates that a man who had been working at Chelsea died at Streatham of a bowel complaint which lasted only a day or two: at that time no other deaths from cholera had been registered within two or three miles of Streatham, but the man's mother, who attended him, was taken ill on the very next day and quickly succumbed to the disease.

Microbes.—The stools, even in health, contain various microphytes in large numbers, and it is therefore by no means surprising that the search for a special cholera microbe was long unsuccessful.*

When the epidemic of cholera which prevailed in Southern Europe in 1883 first made its appearance in Egypt, the German Government sent thither Dr Robert Koch, who had lately discovered the bacillus of tubercle. As the result of his examination of the disease at Alexandria, he believed that he had ascertained its contagium vivum to be a minute rod-shaped organism, which he called a bacillus. He was afterwards sent to Bombay, where cholera was prevalent, and there announced that the microphyte characteristic of cholera was of a curved shape, and named it from this character "the comma bacillus of cholera"—an unfortunate name, for it is in form not a bacillus, but a vibrio. Subsequent researches have confirmed Professor Koch in this belief, which is shared by most other competent observers.

It was, however, opposed to the opinions of the majority of Indian surgeons, and met with much adverse criticism. The late Dr Timothy Lewis, the discoverer of the *Filaria sanguinis*, stated that the so-called bacillus is frequently to be met with in the mouth of healthy persons, and it was found also in cases of diarrhœa by Prior and Finkler in Germany. The English Government sent Dr Klein and Dr Heneage Gibbs to India to investigate the subject, and their report was adverse to Dr Koch's theory. In 1885 the Royal Society and Medical Research Association united to send the late Prof. Roy, of Cambridge, to investigate cholera then prevalent in Spain, and in the following year the same Association deputed Dr Sherrington (who had accompanied Dr Roy to Spain) to clear up doubtful points by researches in Italy, where cholera was still partially epidemic in the summer of 1886. Observations had also been made by French physicians at Toulon. As the result of these difficult and prolonged investigations it was long believed that Koch's conclusions were premature.†

Referring to the criteria for determining the causal relation of a microphyte to a contagious disease stated on p. 18, we ask the following questions: (1) *Is the disease itself definite and capable of accurate diagnosis?* Notwithstanding the marked and striking character of the symptoms and course of cholera given in the preceding pages, there is no doubt that slight cases which occur during an epidemic might at other times be put down as only severe diarrhœa, and yet these slight cases may spread the infection. The diagnosis between so-called cholerine, or English cholera (both of them bad names), and true Asiatic cholera, is often difficult or perhaps impossible; and isolated cases occur at seaports which, in the judgment of cautious and experienced men, are indistinguishable from genuine cholera and yet do not spread the disease. Even the *post-mortem* appearances are not always uniform or decisive. Dr Sherrington found that a large number of the supposed cases of cholera in Italy were really diarrhœa or enteric fever, and Dr Roy had observed the same in Spain. Even granting, what is no doubt true, that if sufficient care be taken in selecting well-marked cases, no appreciable risk of mistaken diagnosis is present in the case of men, the difficulty becomes very much greater when we seek to determine whether the clinical features or the morbid anatomy observed in mice and other animals entitle us to regard a disease induced in them to be the true cholera of man.

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* In the 'Journal of Microscopical Science' for 1881 Dr Cunningham states that "monads," belonging either to *Cercomonas* or to *Trichomonas*, are found in excreta of patients suffering from cholera in unusual numbers and in a peculiarly active state; but he adds that they occur to some extent under other conditions of disease, and even in health. He also finds amœbæ to be very abundant in certain cases of cholera; bacteria, he says, make up a very large part even of the normal fæces, at least in India. Dr William Budd published figures of what he supposed to be a "cholera fungus" as far back as 1849; and others made similar statements, but down to 1884 all lacked evidence.

† This was the verdict given by Burdon Sanderson, and also by a Commission appointed by the Crown to examine and report on the evidence, whose report is given in full in the 'Quart. Journ. of Micr. Sci.,' vol. xxvi, p. 303. Since that time, however, evidence has been gradually strengthened in favour of the specific and pathological character of the microbe discovered by Koch.

(2) *Is the microphyte clearly distinguishable from others?* This cardinal point, after many patient and independent investigations, must still be regarded as doubtful. Even Gruber admits that Deneke's cheese-vibrio is sometimes indistinguishable from Koch's. The latter is not properly called a bacillus; it is a vibrio, or rather a fragment of a vibrio, and it is indistinguishable in size, shape, or reaction to staining agents from Lewis's microphyte. The amount of curvature differs much, and it frequently undergoes changes of form, becoming sometimes rod-like, and sometimes rounded like micrococci. The vibrio is motile by means of a permanent flagellum. It grows best between 30° and 35° C., and is readily destroyed by acids and by desiccation.

Pfeiffer proposed another test for the cholera bacillus, based neither on microscopic characters nor on cultivations, but depending on the effect of the serum of (artificially) immune animals on colonies of vibrios. The subsequent researches of Klemperer, Gruber, Bordel, and other bacteriologists have, however, shown that this test also is far from being universally trustworthy. While Penningham and Sanarelli believe that many vibrios may produce cholera, Hankin and Haffkine maintain that all such are only forms, degenerative or other, of the true specific microbe.

(3) *Does the microphyte only occur in cases of cholera?* The answer to this question is involved in the preceding. Very similar or morphologically identical comma bacteria occur in the alimentary canal both in health and disease, but the genuine organism, tested by its growth and development as well as its form and size, has not yet been proved to exist elsewhere than in the intestines or the dejecta of cholera patients.

Is its occurrence in cases of the disease constant? In the great majority of cases, if not in all, there are numerous "comma bacilli" to be found in the intestinal contents, and occasionally their abundance approaches what Koch called a "pure cultivation" in the small intestines. They do not occur, as the spirillum of Relapsing Fever and (as we shall afterwards see) the bacillus of Anthrax, in the blood or tissues; but neither does the specific microbe of tetanus. The commas are found in the dejecta and in the rice-water contents of the bowels, and also in the detached epithelium

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rio is so constantly present in cases
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gium.—Passing on now to consider
gains access to the human body, we
its entrance by means of drinking-
rves special commemoration, not only
also as having devoted infinite labour

ack as 1849, in which local outbreaks were
ination of surface-wells by sewage; one such
ther in Albion Terrace, Wandsworth, a third
incident was very striking. A gentleman
s at Locksbrook, near that town. Cholera
Locksbrook and became very fatal. The
attached to their houses, drainage from the
to the place, said he could smell nothing
and drank a glass of it. This was on
with cholera, and died on the Saturday.

evidence of the communication of
rrefragable. Then occurred the cele-
d Street, Golden Square, which is said
than five hundred persons living within
traced by Dr Snow to the water of a
at the corner between Cambridge Street
eight of the first eighty-three deaths
known to have drunk the water in
that many took it without being aware
public-houses. A lady at Hampstead,
every day for the Broad Street water, was
Her niece, too, being on a visit to her.

Koch's vibrio is great, and some
lieving that several microbes may
Koch observed some very minute
the comma vibrios, and thinks
found straight bacilli in the

drank some of the water, returned afterwards to her own house at Islington, and died there. On the other hand, scarcely any of the inmates of the workhouse in Poland Street, where there was a separate well, were affected; and the disease also spared the men employed at a brewery in Broad Street, close by.

Observations on a still larger scale were made, in 1854, in the south of London, over a district containing a population of at least 300,000 persons, and supplied with water partly by the Lambeth Water Company, conveyed from the Thames at Ditton, partly by the Southwark and Vauxhall Company, whose intake was from the same river at Battersea. These observations were peculiarly conclusive because the mains of these two companies ran side by side, each feeding some houses and not others. Dr Snow went in detail throughout the area in question, from street to street and from house to house, and he found that, during the last four weeks of the epidemic, out of three hundred and thirty-four fatal cases of cholera, there were two hundred and eighty-six in houses supplied by the Southwark and Vauxhall Company, twenty-two among persons who obtained water directly by dipping a pail into the Thames, but only fourteen in houses receiving a supply from the Lambeth Company. Bethlehem Hospital, the Queen's Bench Prison, and Guy's Hospital on the south side of the river had deep wells, and the inmates scarcely suffered at all from cholera.

In 1865 and in 1866 further evidence was collected bearing in the same direction. In 1866 the parts of the metropolis mainly affected were the eastern districts, and Mr Netten Radcliffe, who investigated the matter for the Privy Council, found that there was a great preponderance of cases among persons whose water, supplied by the East London Water Company, had passed through the reservoirs at Old Ford, whereas comparatively few cases occurred among those who received the water of the same Company pumped directly from the filtering beds at Lea Bridge into the mains. The difference was far from being as conspicuous as in South London during 1854, but one must bear in mind that of the poorer classes many eat and drink at a distance from the houses in which they are said to live. Moreover, at an early period of the epidemic a notice was issued, warning people not to drink any water which had not previously been boiled, and from that time the epidemic began to decline.

A point of particular interest is that, assuming the reservoirs at Old Ford to have been in some way concerned in causing the disease, one can account for the entrance of the cholera poison into their water; for part of it was sometimes drawn from two reservoirs which communicated freely with the river Lea by soakage, and this part of the river was, in fact, a canal with locks, and received so large a quantity of sewage, that it was little better than a cesspool. Now, shortly before the epidemic in East London began, a man and his wife, living in Priory Street, Bromley, near the banks of the Lea, had died of cholera, and their evacuations had entered the river about 600 yards below one of the open reservoirs.

In the previous year (1865) there occurred in an Essex village, Theydon Bois, a remarkable local outbreak of cholera. Between July and November the disease was introduced into Southampton by persons who arrived from the Mediterranean ports where it was then prevailing. Whether it was carried from this town during August or September to Weymouth or Portland or Dorchester, or whether it reached one of these places in some other way, was never ascertained. But so much is certain, that a gentleman and his wife, visiting these places (but not Southampton) in September, contracted diarrhœa at one or other of the three, and this, in the case of the lady, developed into fatal cholera after her return to her home at Theydon Bois on September 25th. On the 30th, one of her daughters was attacked and died in a few hours. The same night a serving-lad in the house was seized and barely escaped with his life. Altogether, in a fortnight, eleven persons within a narrow circle—father, mother, grandmother, two daughters, son, doctor, foot-boy, maid, labourer, and countrywoman—fell ill, and only three of them recove Now the drinking-water of the house all came from a well beneath the scullery, and in that well there was habitual soakage from the water-closet.

That cholera is conveyed by drinking-water is confirmed positively by its endemic prevalence in Bengal, where the village ponds and tanks and the soil surrounding them are habitually defiled with ordure; and negatively by such cases as that of Naples, which was until recently notorious for cholera and enteric fever, and now, since an abundant supply of pure water has been obtained, is free from both.

Other modes of conveyance.—Whether the virus of cholera is ever conveyed by the air is doubtful; the comma bacillus dies when dried up, but it is obviously possible for it to be carried into the alimentary canal in food or in anything defiled by contact with sewage. Like plague, the virus may perhaps be conveyed by flies or other insects from soiled linen to food (Haffkine). The following case, recorded by Dr Simpson of York, and cited by Dr Snow, is probably to be thus explained.

> An agricultural labourer was attacked at Monkton Moor on December 28th, 1832, at a time when the disease was not known to be prevailing within thirty miles. His wife and some other persons who visited him were seized on the following day. Presently it was found that his sister had died of cholera a fortnight previously at Leeds, and that her wearing apparel had been sent to Monkton in a box which had been opened by him the evening before he fell ill. During the illness his mother, who lived in a healthy village five miles off, came to attend him, washed his linen, and after two days set off to return home, but was attacked on the road and had to be conveyed to her cottage. Not only did she die, but her husband and her daughter likewise.

Soil-water theory.—The "soil-water" or "ground-water theory" of Pettenkofer is that cholera never prevails epidemically where the soil is impermeable to water, or where the level of the soil-water is not liable to fluctuations. He brought forward several instances in which districts seated upon hard rock escaped, while adjacent towns built upon alluvium suffered. Some apparent exceptions to the rule he accounts for, more or less satisfactorily. Thus Gibraltar, as is well known, shows no immunity, but, in visiting it, he found that the town really lies on a slope of red earth, containin more than 200 surface wells. So, again, Malta, where the disease has prevailed severely, consists of solid rock; but this, a sandstone, is so soft an so permeable by water that the Government Comptroller told Profess Pettenkofer that it was like a sponge, saturated with all kinds of filth. C the other hand, Lyons has, during each of the European epidemics, r mained free or nearly so from cholera, when both Paris and Marseilles we ravaged by it: in 1865 twenty thousand persons are said to have flock thither from Marseilles for safety. But the greater part of Lyons lies on river alluvium, so that on the ground-water theory one would expect inhabitants to be severely attacked.*

After opposing Koch's hypothesis for many years Pettenkofer has a mitted that the presence of the comma vibrio is so constant that it mu be regarded as essential, although he still holds it to be only operative wi the concurrence of favourable conditions in the soil. He still believes that the time when cholera is most apt to prevail is when the level of the ground-water has begun to fall after being high.*

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* The explanation which Pettenkofer gave is that in this town, unlike most others, level of the soil-water is diminished by that of the rivers which flow through it, the bank the Rhone and the Saone being in fact so porous that their streams may almost be said run in part subterraneously beneath Lyons. In other words, the supposition is that Ly escapes cholera either because there is too much surface-water, or because its height subject to scarcely any variation. But, ingenious as these explanations are, one cannot h wondering whether it might not be possible, by similar reasoning, to explain away the v instances on which Pettenkofer relies.—C. H. F.

Secondary ætiology.—Certain conditions seem to influence the diffusion of cholera indirectly. Dr Farr laid down a "law of altitude," at least for London, showing that the incidence of the disease upon the population varied inversely as the height above the river Thames. But Lebert found that throughout the epidemic at Zürich in 1855 the upper parts of the town suffered more than the rest. The fact that in Europe cholera has never been known to prevail at an elevation of more than 600 or 800 metres above the sea is probably accidental, for in other quarters of the world it has raged at altitudes of 2000 to 2500 metres.

In temperate climates the summer and the autumn are the seasons most favourable to cholera. It often dies out as winter approaches, to reappear in the following year; but in Russia, in 1830, it withstood extreme degrees of cold.†

The microbe of cholera does not cause the disease in every person who is infected; some always escape. Thus Macnamara narrates how, of nineteen persons who drank infected water, five only fell ill of cholera; the vibrio had been destroyed by the gastric secretion of fourteen. We can thus admit the mischief done by eating unripe fruit and neglecting ordinary diarrhœa in times of cholera; for the disordered intestine furnishes the most suitable soil for the microphyte.

Diagnosis.—To recognise cholera in its well-marked forms is seldom difficult. Almost the only morbid state which can be mistaken for it is acute irritant poisoning. Louis made this mistake in the case of the Duke of Choiseul, who killed himself with arsenic the day after his arrest for the murder of his wife in 1847. There is therefore good reason for bearing in mind that a crafty person might take advantage of an epidemic to give poison with but little risk. Again, mushrooms and tinned meats have produced effects closely resembling cholera.

To distinguish the slighter effects of the virus of cholera from ordinary diarrhœa is often quite impossible. Under the name *cholera nostras*, or *cholera Europæa*, writers describe an affection of which the symptoms are identical with those of the specific disease (called by them *cholera Asiatica*), but which differs from it in being scarcely ever fatal and in generally occurring sporadically. A typical instance occurred at Guy's Hospital in 1865, and was recorded by Wilks in the 'Med. Times and Gazette' for that year.

A blacksmith's hammerer, aged thirty-three, was at work as usual at 7 a.m. on May 25th, when he was suddenly seized with profuse vomiting and purging. He was admitted collapsed, with cold breath; he had cramps in the flanks and in the legs; the evacuations and the matters vomited had the appearance of rice water, consisting of an alkaline liquid with flocculi floating in it. He recovered in about twenty-four hours. Wilks added that every year he saw one or two such cases, but seldom so early in the summer.*

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* It is, Pettenkofer says, conceivable that, if the yeast-fungus were absent from certain countries, the inhabitants might prepare beverages from grapes, or apples, or malt, and drink them without any liability to intoxication. But if some one were to come from a country where the yeast-fungus was found, and to bring its spores with him upon his clothes or in any other way, the hitherto harmless liquids might speedily produce an "epidemic of drunkenness." Yet the cause would not be the yeast-fungus, but the alcohol of the fermentation set up by it. So Pettenkofer imagines that the "cholera-germ," *x*, acting on the soil under certain conditions, *y*, generates a "cholera poison," *z*.

† The remarkable distribution of cholera in its visitation of England, 1848–9, its spread, ingravescence, and decline in the several towns it attacked, are described, and their bearing on its ætiology as then appreciated impartially discussed by Dr Baly and Sir William Gull in their 'Reports on Epidemic Cholera' to the Royal College of Physicians in 1854.

The mucous stools of catarrhal colitis, sometimes clear and free from bile, should be examined by the microscope and by cultivation for the vibrio when there is doubt as to the nature of the disease.

Prognosis.—This is less favourable in children and in old people suffering from cholera than in adults or in middle-aged persons. The mortality, which over a whole population generally averages about 50 per cent. or a little higher, rises among the very young and the aged to 70 or 80 per cent., while among temperate young adults it may fall to about 40 per cent.

At Dublin, in 1866, of 130 cases at the Meath Hospital, 71 were fatal; of 180 at Sir Patrick Dunn's Hospital, 85; and of 197 at the Mater Misericordiæ Hospital, 106. In 1884, out of 21,519 patients in Italy, 11,563 died. In the following year no less than 338,685 cases of cholera were officially reported in Spain, and of these 119,620 were fatal, or more than a third.

The disease is more than usually dangerous when it affects drunkards or those who are already weakly, especially from renal disease. The degree of collapse is of much greater prognostic importance than the amount of purging. As in other epidemic diseases, the earlier cases are far more often fatal than those which occur when the pestilence is subsiding. At Calcutta, in 1850, the average mortality was 47 per cent., but among the earliest cases it was 75 per cent., and among the latest only 25.

The *protection* afforded by an attack of cholera against a subsequent one appears to be of the slightest. Cases are not infrequent of persons who have suffered in two epidemics, and some have died from the second attack. Moreover, in those parts of the East Indies where cholera is endemic, one illness not only affords no immunity from a second, but seems scarcely to mitigate its severity. All that can be said is that cholera does not predispose to fresh infection, as erysipelas undoubtedly does.

Prophylaxis.—The measures to be adopted when a locality is threatened or actually attacked with an epidemic of cholera were carefully elaborated by Sir John Simon and his colleagues in 1866, and will be found detailed in his ninth 'Report.' They were chiefly directed against the predisposing causes of the disease. All food should be avoided which is apt to set up diarrhœa—beer, meat or game no longer fresh, stale fish or shell-fish, vegetables or fruit long gathered; and looseness of the bowels, however painless and trivial, should be checked at once.* The late Sir George Johnson's theory, that the purging of cholera is an effort of nature to get rid of the *materies morbi*, and should therefore not be checked, but encouraged by administering castor oil, does not appear to be accepted by any other physician.

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* It is to be observed, however, that at the very time when this man was attacked, an epidemic of cholera was approaching England; and, utterly improbable as it may seem that an isolated case should spring up in London several weeks before the commencement of the local epidemics at Southampton and at Theydon Bois, the possibility of such an occurrence cannot be denied. At any rate, no case of so-called "English cholera" (attended with collapse, rice-water stools, and cramps) seems to have been admitted into Guy's Hospital since 1870; and it is certain that when the first epidemic arose in 1831 Sir Thomas Watson, the elder Dr Babington, and many other physicians of experience, declared that they had never "met with the same complaint before." However such sporadic cases are to be regarded, we ought never to call attacks of diarrhœa in infants, or even in adults, "choleraic" merely because the symptoms are severe."—C. H. F.

Prophylaxis should also be directed to maintaining a vigilant inspection of all sources of drinking-water, and providing for the proper disposal of the evacuations which carry the specific vibrio of the disease.

A system of inoculation with a weak cultivation of the vibrio, as a prophylactic, has been elaborated by Dr Haffkine, and tested by him on a large scale in Bengal and Behar, in Oudh and the Punjab, in a gaol at Guya, and in Assam. In 1893 more than 22,000 persons were thus inoculated, and between March, 1894, and July, 1895, nearly 20,000 more. The result was that when cholera visited any of these places the mortality was far less in those previously "vaccinated" than in the rest of the population, including European soldiers as well as natives (official report to the Government of India, Calcutta, 1895, and report of his lecture at the Conjoint Laboratories, December 18th, 1895; see the 'Brit. Med. Journal' of the 21st following, and 'Proc. Royal Soc.,' 1899, p. 254).

Treatment.—Most writers recommend that the initial diarrhœa should be treated with opium in considerable doses, and with astringents, such as acetate of lead, nitrate of silver, catechu, or chalk. In India Dr Goodeve says that it is usual to give from one to five grains of calomel with the first two grain-doses of opium, and to check the diarrhœa as much as possible.†

When *collapse* has developed itself the administration of opium or astringents is useless, or worse than useless. Absorption is nearly arrested; and drugs may, if not vomited, accumulate so as to produce ill effects should reaction afterwards occur. For the same reason alcohol must be used with great caution, if at all.‡

Ice-cold water may be freely allowed to patients with cholera,—not, indeed, in such large quantities as they demand, but by tablespoonfuls or wineglassfuls at a time. Lebert would also give effervescing draughts. In 1866 a "saline lemonade" was employed with apparent advantage. Injections of hot water into the rectum may help in relieving thirst as well as in diluting the blood.

There seems to be no objection to administering a dose of morphia subcutaneously if the cramps are very severe. Or a little chloroform may be given by inhalation. It may also give relief to rub pure chloroform or turpentine or powdered ginger into the painful parts. The burning sensation at the epigastrium may be alleviated by applying a mustard plaster.

The limbs should be kept wrapped in warm flannels, and hot bottles should be placed in the bed. At the London Hospital, in 1866, baths at a temperature of from 98° to 104° were employed in about one hundred and thirty of the worst cases, and with most marked effects. For a few seconds after immersion there was commonly difficulty of breathing, and sometimes an unpleasant sensation of heat. But in less than a minute a favourable action generally became manifest; the pulse returned, or (if it had been perceptible before) became fuller and quicker; the patient grew less distressed, ceased to moan with pain, and sometimes fell into a quiet slumber. In many cases recovery appeared to be the direct consequence of the bath; but in many more the symptoms returned unaltered.

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* Dr Bristowe alone maintained that the diarrhœa which so commonly prevails when cholera is epidemic is neither more nor less likely to pass on into that disease, whether it is left alone or encouraged by purgatives; nor would he allow that, if it is really premonitory of cholera, it can be arrested by any medicine whatever.

† The "cholera pill" consists of opium, black pepper, and assafœtida.

‡ Dr Goodeve says that its admissibility depends upon its effect on the pulse. If a dose of weak brandy-and-water causes the pulse to revive ever so little, there is no harm in continuing to give the stimulant in small quantity. In any case ether or ammonia may be used, properly diluted, unless sickness is produced.—C. H. F.

Experience is in favour of injecting a warm saline solution into a vein even when patients are in an apparently hopeless condition from collapse. Among fifteen cases so treated at the London Hospital by Mr Little there were four recoveries. The immediate effect of an injection is admitted on all hands to be often marvellous; a person speechless, and almost dead, may regain consciousness, sit up, and talk; and the pulse may become distinct and full. But in most cases the improvement has been only temporary; the symptoms soon return and end fatally, even though the operation should be repeated. Still, as Sir Thomas Watson observes, even such a transitory amendment may sometimes be of great importance, allowing, for example, a will to be executed.

Dr Macleod recommended a drachm of sodium chloride and half a drachm of sodium bicarbonate, dissolved in a litre (35 fl. oz.) of boiled water, injected at the temperature of the body. He finds, as others have, that symptoms are remarkably relieved and probably some lives saved, although the numerical proportion of recoveries may not have been increased. A litre should take about ten minutes to flow in by force of gravity; and two or even three may be used if needful to restore the pulse.

When *reaction* begins, the management of the case of cholera continues to require great care and caution. A little beef-tea or chicken broth may now be given at intervals, or such light farinaceous food as gruel or arrowroot. Should vomiting continue, it may often be checked by a dose of opium, or by a blister applied to the epigastrium; but sometimes it is necessary, for a time, to have recourse to nutrient enemata. When suppression of urine continues during reaction, the patient should drink freely; he may take a drachm of liquor ammoniæ acetatis, with five or ten drops of tincture of digitalis, every two or three hours; and mustard plasters or cupping-glasses may be applied to the loins.

YELLOW FEVER

> "I say, messmate, have you ever had the yellow fever, the *vomito prieto*, black vomit, as the Spaniards call it? No? Have you ever had a bad bilious fever, then? No bad bilious fever either? Why, then, you are the most unfortunate creature, for you have never known what it is to be in heaven nor else in the other place. Oh the delight, the blessedness of the languor of recovery!"—*Tom Cringle's Log.*

History—Inculation, course, and events—slight cases—Protection—Anatomy—Ætiology—its contagious nature—its distribution—Its pathology—the specific microbe—Diagnosis—Prognosis—Preventive and curative treatment.

Synonyms.—Typhus icterodes—Febris flava—Bilious Typhoid Fever—Black vomit—Yellow Jack—Specific diffuse Hepatitis—Icterus gravis with parenchymatous hepatitis—Calentura vomito-negro—Vomito prieto—Coup de barre. Mal de Siam is probably a bilious remittent, not Yellow Fever.

Definition.—A specific infective fever, running a short course, with vomiting, jaundice, albuminuria, and hæmorrhage, occurring in epidemics in the hot parts of America.

History.—The Oriental plague has never visited the Western Hemisphere, but it is there replaced by another and not less terrible epidemic disease, which from time to time visits the cities of tropical and subtropical merica. This specific and infectious fever is almost limited to tropical merica, the West Indian islands, and the West Coast of Africa. Although has sometimes been imported into Europe, it has never maintained itself mong us, and in the East it is unknown.

The first epidemic on record was at Porto Rico in 1508. In 1647 Yellow ver appeared at Barbadoes, in Cuba in 1648, Jamaica in 1655, and at uadaloupe in 1635-40-48, where it was called *nova pestis.* A severe pidemic of the same kind visited Philadelphia in 1693, and again in 1762, '93, and 1802. It was at Mauritius in 1815. It first appeared on the razilian seaboard at Rio Janeiro and Bahia in 1849, at Buenos Ayres in 858, and at the port of Callao, in Peru, in 1853. There was a terrible pidemic at New Orleans in 1878, and in Florida in 1888.

Yellow fever is endemic in the island of Hispaniola (Hayti and San Domingo) and in Panama, and more or less frequent throughout the West Indies and the adjacent coasts of Mexico and Guiana.

In Europe yellow fever repeatedly occurred between 1780 and 1820 at Cadiz, at Gibraltar in 1804, 1814, and 1828, and at other Spanish ports; in 821 at Barcelona, and afterwards at Marseilles and Leghorn. There was an epidemic in Lisbon in 1857, and a very small one at Swansea in 1865, introduced from Cuba in the barque "Hecla."

In Senegal yellow fever appeared as an epidemic in 1778, in Senegambia in 1820, and since that year. It was then imported from America, but possibly, as Dr Creighton holds, it was at first carried by the slave trade from Africa to the New World.

Course.—The incubation of yellow fever is short, often only a day or two, rarely above a week.

There are sometimes slight prodromal symptoms of malaise and headache; but the disorder often sets in suddenly with rigors and extreme depression. Sometimes it proves fatal in a few hours with collapse and lividity. More often the temperature rises for two or three days until it reaches 105° Fahr., or, as was once recorded, 107°; the face becomes flushed, the eyes red, ferrety, and staring. Frequently there is one-sided headache, or an agonising pain in the back and sacrum, or there may be pains in all the joints. The stomach becomes irritable, and rejects its contents; the epigastrium is tender to pressure. The palate is reddened and œdematous; the gums swell and bleed; the tongue is furred at first, but afterwards clean, smooth, and raw. Constipation is more frequent than diarrhœa. The urine is very scanty, with deficient urea, and it generally contains albumen.

About the third day, or a little later, the next stage succeeds—the "lull" or *stadium* of the fever. The skin becomes jaundiced, and the urine is bile-stained; but the fæces are seldom clay-coloured. Hæmorrhage now appears; epistaxis is frequent, and sometimes there is cutaneous purpura; the vomited matters, from being "white" and consisting of an acid watery liquid, become "black," containing dark brown flocculi or larger masses made up of altered blood-corpuscles. It is to be observed, however, that this symptom of *black vomit* occurs only in the more severe forms, and chiefly in such as end fatally. Thus, during an epidemic at Gibraltar in 1828, Louis found that of the patients who died, all but one had it, and of those who recovered very few. According to Alvarenga, however, at Lisbon, in 1857, there were no fewer than forty recoveries among cases in which black vomit was present.

Haenisch, from experience of the disease in the West Indies, states that there is usually on the fourth day a remission of the pyrexia, so that the temperature may fall nearly to normal; the patient loses his headache, and experiences so much relief that he fancies himself convalescent. The stomach, however, still remains irritable; and the urine, if not previously albuminous, now becomes so. In some cases the improvement is permanent, and goes on to complete recovery. But more often, after a few hours or within two days at longest, matters change again, and for the worse. The temperature may now rise until it reaches 104°; but sometimes remains normal, as in a patient of Mr Leggatt who died of yellow fever in London in 1878 ('Clin. Trans.,' vol. xi).

The symptoms at this period seem mainly to depend upon disturbance of the renal secretion. The urine is often entirely suppressed for several days together; it always contains albumen, and frequently blood-casts. The patient sometimes falls into an apathetic condition, but is more often restless and talkative, and sometimes violently delirious. The pulse is small and thread-like, sometimes frequent, sometimes slow. Jaundice appears and increases until the skin is of a dark mahogany colour. Hæmorrhage continues, blood being passed from the bowels, from the ears, or from the respiratory mucous membrane. Death is usually preceded by coma.

but sometimes it occurs suddenly, the patient falling backwards upon his bed in syncope, after a state of violent excitement. But recovery may take place even when the case has appeared most threatening —usually by crisis, with a rapid fall of temperature and profuse sweating.

Among the *sequelæ* observed are suppuration of the parotid, sloughing of the scrotum and penis, and the formation of boils. Convalescence is always slow, and several weeks elapse before the patient regains his strength.

During an epidemic of yellow fever cases occur in which the symptoms are but slightly pronounced. The pyrexia is moderate in degree; jaundice is absent or slight, or may appear only when the attack is passing off. In sometimes presenting itself in a very mild form yellow fever resembles enteric fever, scarlatina, and plague.

The *protection* afforded by this disease against second attacks is said to be very efficient, and relapses are rare.

Morbid anatomy.—Rigor mortis is early and well marked; and, according to Nielly, putrefaction is retarded, which is not what one would have anticipated. The body is deeply jaundiced, more deeply sometimes, says Dr Macdonald, than it was during life. The heart is often pale and soft, and the fibres in a state of fatty degeneration. The pleuræ are ecchymosed, the lungs purpuric, and hæmorrhage is found between the muscles and under the serous membranes or the meninges of the brain. There is usually acute catarrh of the whole length of the alimentary canal, and the stomach often shows hæmorrhagic erosions; but in Mr Leggatt's case the stomach was pale, and its mucous membrane was not swollen. According to Macdonald, the stomach appears perfectly normal when the digested black blood is washed off.

The *liver* may present patches of a bright yellow colour, or its tint may be uniformly jaundiced. The gall-bladder and ducts are found empty, or filled with colourless mucus. In Mr Leggatt's case, Dr Greenfield, who made the autopsy, found that the portal canals were crowded with leucocytes, that many of the bile-ducts were filled with swollen epithelium, and that the greater part of the hepatic cells were undergoing disintegration, being swollen and fused together, or broken up into irregular fragments.*

Liebermeister, in his work on 'Hepatic Diseases,' maintains that yellow fever is nothing else than a "parenchymatous hepatitis" dependent upon a specific infection. This view is supported by Dr Wickham Legg in his work on the 'Bile and Jaundice,' where he cites the description of microscopical appearances by Dr Alonzo Clark, of New York, and several other observers. Acute fatty degeneration with atrophy is constant, and all observers agree that the hepatic cells are loaded with fat globules.

The *spleen* is not enlarged; its tissue is often firm and healthy-looking, and is not infrequently shrunken—probably as the result of gastric hæmorrhage.

The *kidneys* are swollen, and may show points of suppuration; in the case already referred to, Dr Greenfield found the epithelium of the convoluted tubes swollen and proliferating, and the straight tubes contained hyaline casts; but the kidneys were in this instance granular, so that these changes may have been unconnected with the yellow fever. According to Dr Legg, however, a condition of cloudy swelling like that found in the early stage of parenchymatous nephritis is the rule.

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* It may, however, be a question whether in this case the state of the portal canals was not an accidental cirrhotic ch[illegible] due to the known habits of the patient.—C. H. F.

The *blood*, according to Dr Joseph Jones, of New Orleans ('Journ. of Amer. Med. Assoc.,' March 16th, 1895), shows little alteration of the red discs in number or tint, but great deficiency in coagulation, the clot being soft and bulky. The serum is of a deep yellow colour from the presence of bilirubin.

Ætiology.—One of the most striking features of the disease is that its contagion is often transported by ships. Instances of this have become historical. In 1823 H.M.S. "Bann" carried yellow fever from Sierra Leone to the Island of Ascension. In 1845 the "Eclair" steamer brought it from the African coast to Boà Vista, one of the Cape de Verde Islands. In 1861 it was conveyed by a wooden sailing ship, the "Anne Marie," from Havannah to St Nazaire, in France; and in 1865 by a similar vessel, the "Hecla," from Cuba to Swansea. Moreover the disease frequently passes from one ship to another, not only when they have been lying side by side in the same port, but also on the high seas. The "Anne Marie," for instance, infected seven other vessels which happened to be brought near her; and during the local epidemic at Swansea a smack, the "Elinor," which took in cargo near the "Hecla," had her crew attacked after they had left that port and had reached Llanelly. So, in 1795, the "Hussar" frigate captured a French ship, the "Raison," on which yellow fever was prevailing; only those prisoners who were believed to be in perfect health were transferred to the English vessel, but notwithstanding this precaution the disease soon broke out among her crew.

These facts are best explained by supposing that yellow fever, like typhus, measles, and scarlatina, is directly propagated from the bodies of the sick. The infection, like that of plague, has also been found to cling to the hull, or perhaps to the cargo, of a particular vessel, after the crew have been paid off. It is believed in the West Indies that a cargo of hides or of sugar is favourable, and one of salt unfavourable, to the development of yellow fever on board or its transfer from one port to another.

In the 'Med.-Chir. Review' for 1848 and subsequent years there appeared a series of able articles, which are now known to have been written by the late Dr Parkes, in which evidence was offered that yellow fever is contagious. He relates in full detail the circumstances which attended its diffusion in Boà Vista from two soldiers, belonging to the fort, who were lodged while ill in the chief town of the island, Porto del Rey, as well as from a labourer, who brought it direct from the "Eclair" into another town, Robil; and in each case he shows that the next persons to be attacked were those who lived close to the patients and visited them.

Nevertheless many of those who have had the largest acquaintance with yellow fever have disbelieved in its contagiousness, in the ordinary sense of that term, and many facts have been adduced in support of their view. One is that, as Griesinger remarked, the disease often remains localised upon the sea-shore, or in close proximity to the banks of a navigable river; it may even confine itself to a small part of a seaport town in the immediate neighbourhood of the harbour. Thus, when it prevailed at Lisbon in 1857, one hundred and eighty-two persons are said to have left the city for different places in Portugal, carrying with them the disease, and eighty-six died, but in no instance was it communicated to other persons in the places whither they went. In 1865 the late Sir George Buchanan, having investigated with great care the local epidemic at Swansea, came to the conclusion that "the evidence tending to negative personal contagion was about as strong as such evidence can by its nature ever be."

The evidence of contagion is very strong, however, in many cases (see Dr Davidson's cases in Allbutt's 'System,' vol. ii, p. 391); and the contagion seems to hold to ships as that of plague to houses.

Climate.—Yellow fever appears to be endemic in some parts of Central America. Probably in the gulf States the cold weather in winter prevents this, and hence epidemics occur there only at intervals and among a largely unprotected population. The disease only flourishes in hot climates—in Mexico and Guatemala, New Orleans, the West Indies, and Brazil. The regions in which the disease commonly prevails are all situated near the equator, and the occurrence of a local epidemic within the temperate zone seems constantly to be associated with unusually sultry weather at the time. This was the case at St Nazaire, in France, where it appeared in 1861, and also at Swansea in 1865. The latitude of Swansea is 51° 37′, which is beyond the geographical limit usually laid down for the disease. So, again, upon low coasts and near the mouths of rivers the worst months are generally July, August, and September; although in small rocky islands within the tropics the period from October to February is stated to be the most dangerous. Some exceptions to the rule that the contagion of yellow fever cannot resist cold have been observed. Thus Dr Archibald Smith recorded, in the first volume of the 'Transactions of the Epidemiological Society,' the fact that in 1855 it prevailed at Cuzco, in the Peruvian Andes, where (as he was informed) the temperature of summer rarely, if ever, reaches 65° Fahr. in the shade; and also at Cerro Pasco, with a mean temperature of 44° by day. So, again, Mr Leggatt's patient, who died in London, was taken ill on March 21st, 1878, the mean temperature of the four previous days having been 54°. He had arrived at Southampton from Rio Janeiro (where the disease was epidemic) on March 17th, and there had been three other cases of yellow fever on board the ship.

The geographical limits of yellow fever are remarkable, not only in being almost confined to tropical America, but in its preference for the owns on the coasts and along the course of the great rivers.

Its original seat seems certainly to be the Antilles, whence it has within istorical times invaded Central and South America, its northern limit being uebec, and its southern Monte Video; and good observers in the French est Indies, like Nielly, believe that it is endemic in Guadaloupe and artinique in a comparatively mild form. This would agree with the atural history of plague, of cholera, and of the sweating sickness.

Race and age.—It is remarkable that yellow fever seldom affects egroes, and is said to be less common among mulattos or quadroons han in those of pure European blood. According to Humboldt, the ndians of South America are also exempt. The Algerian Turcos in the rench army escaped during the campaign at Vera Cruz in 1866, and hinese coolies are said not to suffer in an epidemic. This, however, is not a mplete exemption, for Dr James Thorington reported several cases in egroes and mulattos *at Colon, in Panama, in the* year 1882, and Rush at Philadelphia in 1793

Men are more often attacked than women, and adults than children and old people—facts which probably depend on the usual spread of the disease in epidemics being by commercial intercourse.

Pathology.—Yellow fever is certainly a specific disease in its origin as in its course, and the above ætiological facts point to its being miasmatic-contagious (cf. p. 17), although of this there is at present no proof.

It is now universally admitted that yellow fever is distinct from any form of intermittent or remittent fever of malarial origin; for its geographical range is quite different, it is epidemic, it is transmitted from place to place, it consists of a single attack and protects against future invasion, albuminuria is constant, the spleen is not enlarged, and quinine, so far from being a specific remedy, is believed to be injurious.

A question remains as to the relation of yellow fever to other forms of malignant jaundice (*icterus gravis*). Grisolle, Garnier, Liebermeister, and Wickham Legg maintain that the two are identical, and that the cases of acute yellow atrophy of the liver which occur in Europe are sporadic cases of yellow fever. This can scarcely be. The absence of fever in the former, and its presence and height in the latter; the atrophy as well as softening and degeneration of the liver, the swollen spleen and the non-contagious character of the former, to say nothing of the absence of hæmorrhagic vomiting and the presence of leucin and tyrosin in the urine—all these facts prevent our identifying the two diseases. But the icterus, the hæmorrhages, the rapid course, and the histological change in the liver, with the affection of the kidneys, show some relation between them.

The discovery of the pathogenic microbe of Yellow Fever has been repeatedly announced; but Sternberg, of Washington, showed that none of them had sufficient evidence to support its claim. None was found, though carefully searched for with all the modern methods of detection, during the epidemic in Florida and Cuba in the year 1888. In 1897, however, Dr Sanarelli, of Monte Video, formerly of the Pasteur Institute, discovered what appears to be the pathogenic microbe of yellow fever ('Nature,' July 15th, 1897, p. 249). He believes that he has succeeded in separating from the crowd of other organisms, and particularly from the *Bacterium coli commune*, streptococci and staphylococci, a rod-like microbe, constantly found in the liver, spleen, and kidneys of those who have died of Yellow Fever. He makes a pure culture of this *Bacillus icteroides* (as he somewhat oddly names it), and reproduces the microbes and the anatomical changes of the disease by injecting it into dogs, goats, and horses. The same symptoms follow injection of the toxines, after filtration of the culture through a Chamberland filter. Corroborative evidence from other observers seems to be all that is needed to establish the specific character of the microbe.

Diagnosis.—This is not always easy except when yellow fever is known to be prevalent. The affection most apt to be mistaken for it is the bilious form of remittent fever. Other diseases that must be borne in mind are relapsing fever, the various forms of jaundice attended with pyrexia, acute yellow atrophy of the liver, and poisoning by phosphorus. The distinction from remittent malarial fever has already been indicated.

The "bilious typhoid" described by Griesinger in Egypt and the

Siamese fever of French writers were both probably remittent fevers, and not, as has been supposed, yellow fever.

Prognosis.—Yellow fever is a dangerous pestilence—very different in this as in other particulars from malarial fevers. The mortality seems to vary widely in different epidemics, being sometimes as low as 15 per cent., sometimes as high as 75 per cent. Even in the comparatively mild visitation of New Orleans in 1878 no less than 4056 people died, and there were about the same number of deaths at Barcelona in the year 1821.

The general indications are that the greatest danger is in the case of drunkards, and of those who go about while the fever is already upon them. In children the prognosis is favourable.

In a given case the severity of the initial pyrexia is not of bad omen; but much vomiting, and particularly hæmatemesis, in the second stage, and the presence of petechiæ, suppression of urine, severe cerebral symptoms, convulsions, and stupor with dilated pupils, are all unfavourable and often fatal symptoms.

The symptom of most value as regards prognosis at an advanced stage is said to be albuminuria; if the amount of albumen in the urine diminishes as the case goes on, the patient is likely to do well; if it increases, a fatal termination is probable.

Prophylaxis.—The method of quarantine (Fr. *quarantaine*, a period of forty days) was introduced by the republic of Venice in 1348, to prevent the invasion of the Oriental Plague, or Black Death as it was then called. In England it was enforced by law under Queen Anne (1710) in order to prevent the return of the plague, and a fresh Act was passed in 1727 by the advice of Mead. No plague appeared, but the quarantine laws were in force until the beginning of the present century, when their uselessness was seen. Since 1831 the same precautions have been in force in many parts of Europe against Cholera. In the New World, and also in Spain, quarantine regulations have also been adopted to prevent the invasion of Yellow Fever.

If yellow fever is a contagious-miasmatic disease, the precautions which should be adopted in order to prevent its transport from one country to another may be greatly simplified, in comparison with the regulations laid down until recently by the best authorities,—as, for instance, by M. Mélier in France after the St Nazaire epidemic in 1861. As Sir John Simon remarks in his 'Eighth Report,' the segregation of persons arriving from an infected town, and their confinement in a lazaretto for a definite period of time, is, on this view, superfluous, and in a trading country like England all but impracticable. The points to which the whole energies of a port sanitary officer should be directed are the isolation and disinfection of the vessel which is known or suspected to contain the virus of the disease. It should be compelled to anchor at a distance from all other vessels; and every part of the hold should be thoroughly cleansed. It would now be advisable to use carbolic acid for this purpose, rather than the chloride of lime, which M. Mélier recommended. The cargo, and the clothes and other effects of the sailors and passengers, should be disinfected at the same time.

The necessity for stringent precautionary measures against the development of yellow fever in England and in other temperate climates is very much diminished by the fact that a high external temperature is an important, if not essential condition of its spreading.

Treatment.—The first point, as in other fevers, is to get the patient to bed at once, and, if possible, without being moved far. At the beginning some experienced physicians give a purge of calomel, jalap, and ginger. The cold pack has proved useful in many cases, but mustard applied to the legs, or a hot foot-bath, appears to be preferable at the onset. For the relief of the sacral pains dry cupping may be used. The irritability of the stomach may be checked by the administration of creasote or of cocaine, of hydrocyanic acid, of chloroform, or of chlorodyne. The patient should be fed with thin arrowroot, barley-water, or chicken broth. Ice is used, if it can be obtained; and it seems to be the usual practice to allow the patient champagne or weak brandy and water during the second stage. In one case recorded by Dr Macdonald, the cook of H.M.S. Icarus very nearly succumbed in the second stage, "but he rallied immediately on the administration of a stout glass of rum and water, and recovered steadily." Notwithstanding the almost constant presence of albuminuria and the not infrequent supervention of uræmic symptoms, physicians experienced in the treatment of this disorder use subcutaneous injections of morphia to relieve vomiting.

In a report to the U.S. Consul at Colon, Drs Thorington and Jennings, who saw much of yellow fever among the workmen of the Panama Railway in 1883-8, strongly recommend the administration of cocaine (ten minims of a 4 per cent. solution of the hydrochlorate) to combat vomiting, and believe that if this can be checked, and the black vomit prevented, the disease is seldom fatal. Dr Thorington insists on darkness, quiet, and sponging as the best helps to sleep, mustard to the nape of the neck and to the epigastrium to relieve headache and gastric irritation, and chloral (gr. xx—xxx) administered *per rectum* when needful as a sedative. Quinine and opium he regards as worse than useless. For suppression of urine he advises mustard to the loins, spirits of juniper, digitalis, and gin and water; and recommends frequently passing the catheter, so as to draw off any urine that is secreted. Surgeon-General Sternberg recommends six grains of sodium bicarbonate and $\frac{1}{80}$ grain of mercuric chloride in an ounce and a half of iced water every hour.

If Dr Sanarelli's bacillus proves to be the true pathogenic cause of Yellow Fever, we may hope that in the serum of horses or other animals which have been rendered immune may be found a direct antidote to this disease, like that which we now employ against diphtheria.

DENGUE

> "Nova febrium
> Terris incubuit cohors."
> HORACE.

History and geography—Incubation—Onset, course, and symptoms—The fever and the interval—The exanthem—Sequelæ—Pathology—Contagion—Diagnosis—Prognosis—Treatment.
Mediterranean fever—Beri-beri.

Synonyms.—Dandy Fever—Breakbone Fever—Three-day Fever—Scarlatina rheumatica—Febris exanthematica articulosa. Other absurd names are "broken-wing fever," and "giraffe," the latter because the neck is held in a stiff position. The word *Denguis* has been coined as a Latin name of the disease, but *Dengue* is the form used by French and German as well as English writers.

Definition.—A specific infective exotic fever, epidemic, running a short course, with severe myalgia and characteristic exanthem, and usually with a favourable result.

History.—Among the new epidemic fevers of the last hundred years is one which, like yellow fever, is unknown in England; but it is common in India and some of the colonies. It clearly belongs to the group of specific fevers, though no microbe has yet been discovered. Among these it resembles influenza in its wide prevalence when it invades a community, in the acuteness of its onset, the pain it occasions, and its usually favourable event.

In 1824 this disease, hitherto unknown to Indian surgeons, broke out at Rangoon, in Burma, and quickly spread to Calcutta, Guzerat, Benares, and afterwards to Aden and Zanzibar. In 1827 the same malady appeared in the West Indies in the island of St Thomas, and a few weeks later in the adjacent island of Santa Cruz; in 1828 it was seen at Savannah and Charleston.

Dr Rush, of Philadelphia, described it as prevailing in that city in 1780. It is remarkable that this epidemic was nearly coincident with one at Batavia and one at Cairo.

During the last fifty years it has from time to time attracted attention both in the Eastern and the Western tropics. There was an outbreak in India in 1871-2, which was said to have come from Zanzibar, passing to Bombay by way of Aden; in 1873 it spread to China and to Cochin China, and also to the islands of Mauritius and Réunion. In the

West Indies it was last seen at Martinique in 1874 and in 1875. The only spot in Europe in which this malady has hitherto appeared is Cadiz, where it was observed by Poggio in 1867; and an earlier epidemic is said to have occurred in the same town as far back as 1764. During 1853 it was common in the United States, and in 1849 in Brazil. It attacked China in 1872. Recent accounts tell of the same disorder as an epidemic in the Levant (1889), and again in Egypt in 1896.

In St Thomas the negroes called the new disorder "dandy fever," apparently in ridicule of the attitude and gait of the patient; and it is now universally known as Dengue—a Spanish word, meaning "prudery."

Course.—The period of incubation is short: four days according to Cotholendy; one to three or four, as Dr Manson observed it at Amoy.

Dengue sometimes sets in with lassitude, drowsiness, vertigo, a sensation of chilliness down the back, and other febrile symptoms; but more often it begins suddenly with pain in some particular part of the body, coming on while the patient is walking about, or waking him from sleep in the night. Dr Stedman, who, in the 'Edinburgh Med. Journ.' for 1828, gave an excellent account of the disease in St Thomas, says that the first thing noticed was often a stiffness in one finger; this would increase and be accompanied by intense pain, which spread over the whole hand and up to the shoulder; and in a few hours the fingers of both hands would be swollen, stiff, and painful. It does not appear that effusion can be detected in the joints, as in rheumatic fever; but Hirsch says that, in the rare cases of death, serous infiltration has been found in the connective tissue round certain joints. Cotholendy, in describing dengue in the Isle of Réunion, supposes there may be effusion into the sheaths of the extensor tendons, which renders movement of the joints so exquisitely painful; and he also speaks of "a slight fulness, a sort of œdema," of the hands and feet. There is often violent pain in the eyeballs, which feel too large for their sockets. Before long every part of the body becomes the seat of extreme suffering, aggravated by restlessness, which compels the patient to be constantly changing his position. He is also extremely prostrate and sleepless. Children are sometimes delirious.

The temperature rises until it reaches 102°, 103°, or 104°, seldom higher; remissions occur three or four times in the twenty-four hours. Most writers describe the pulse as very rapid, 120 or 140 in the minute; but a writer in the 'Arch. de Méd. Navale' for 1874 says that he often found it not much over 80, far lower than might have been expected from the temperature.

Twining long ago noticed that the face was flushed and scarlet, and recent observers describe a fugacious *initial rash*, consisting of bright red patches upon the face, the chest, the palms of the hands, and elsewhere, which subsides after the lapse of a few hours. The whole body is red and swollen, but the forehead, eyes, and cheeks are most affected. There is anorexia; the tongue is thickly furred, with bright red edges. The stomach is extremely irritable, and often rejects everything that is swallowed. The bowels are constipated.

The condition of the patient at this period might well cause considerable anxiety to anyone unacquainted with the disease; but at the end of twenty-four or thirty-six hours the pyrexia begins steadily to abate, without any marked critical sweat, and the temperature soon falls to normal or slightly below it. At the same time the pains in the limbs cease, and soon there is nothing for the patient to complain of except a sense of general weakness, loss of appetite, and backache. At this time it is said that the glands in the neck, the axillæ, and the groins may be felt to be slightly enlarged. The parotid is often swollen and salivation is frequent.

After an interval of about three days an *exanthem*—the *second rash*, or "terminal eruption"—appears. It is first seen on the palms of the hands, next on the feet and the knees, and may in exceptional cases spread all over the body. In appearance it is intermediate between the rash of scarlet fever and that of measles, and it has also been compared with the erythema which sometimes accompanies rheumatic fever. In some cases it is attended with the formation of wheals like those of urticaria, or occasionally of bullæ. It gives rise to a distressing sensation of tingling, which presently passes into still more intolerable itching. Its development is sometimes associated with a return of pyrexia, but recent observations seem to have shown that, as a rule, the temperature remains normal at this period of the disease. Consequently the fact that an eruption has made its appearance is not seldom overlooked; whether it is ever really absent is doubtful. After a few hours, or two or three days, it subsides and disappears, and with it the fever, if any. Afterwards the cuticle begins to desquamate, usually as a brawny powder, but sometimes in large flakes, and this may be attended with considerable discomfort and soreness, especially of the feet. The second eruption, or true exanthem, is often associated with a repetition of the articular pains, which are, however, less severe than at first.

Sometimes after a respite of three or four weeks there is a relapse. The pains return, and compel the patient to take to his bed again and be fed like a child. Dr Stedman speaks of the pains as most severe in the morning and wearing off toward evening. They are felt chiefly in the fingers and toes, in the wrists, the ankles, and the knees. The affected joints may be so stiff and swollen as to produce deformity. After a few days the pains begin to subside, and one limb after another becomes free. A second relapse has occasionally been observed.

Bronchitis may occur as a complication, and Nielly has seen pericarditis in severe cases. Months may elapse before the patient is entirely exempt from return of pain, and the weakness is long-continued and distressing.

Protection from future attacks is, with few exceptions, complete.

Ætiology.—From an epidemic of dengue scarcely anyone escapes, even among the inhabitants of a large city,—as, for example, among the half-million of residents in Calcutta in 1824. It attacks persons of all ages, including infants a few days old, and the coloured races are as liable to it as whites. "When it has lasted for a little time, one might think that there were none but cripples in the place, so many are seen limping about the streets on crutches, with bodies half bent or with arms in slings." The duration of an epidemic is, according to Hirsch, from two to seven months.

The disease is said by Dr Aitken to prevail chiefly with sultry, cloudy weather, or at the time of heavy rains. Dr Manson denies this. In temperate climates it occurs only during summer and autumn, and disappears when frosts set in; but in the West Indies vicissitudes of weather do not seem to have interfered with its course. Its diffusion from one place to another seems plainly to be effected by human intercourse; but whether it is contagious in the restricted sense, passing from the sick directly to the healthy, is uncertain. Dr Stedman says that in 1827 it was introduced from St Thomas into Frederickstadt, a town of Santa Cruz, by some young ladies who went to stay at a house where all the members of his family were attacked, and a few days later it appeared in the next house and affected every one there. Cotholendy relates that an infant, which had taken the disease while with the family of its nurse, was brought home to its mother; four days afterwards she fell ill; the grandmother and the aunt did not see the child until the day after its arrival, and they were attacked a day later than the mother.

No distinctive morbid anatomy is known, and nothing has yet been ascertained as to the nature of the infective virus.

Diagnosis.—The epidemic character and the peculiar rash, with the immunity of the heart, distinguish this singular disease from rheumatism, the articular pains and absence of sore throat from scarlatina or measles. Dr Sandwith writes of an epidemic in Cairo that it is more like Rubeola than any other disease. It is not malarial, for there is no splenic enlargement, and quinine is useless.

Prognosis.—Dengue is very seldom fatal, but infants sometimes die with convulsions during the primary pyrexia, and old people may succumb to exhaustion towards the end of the disease, or to secondary bronchitis. During convalescence the patient is prone to attacks of malaria, dysentery, or other tropical disorders.

Among the natives in India, Dr Mouat had convincing proof that dengue is severe and protracted when left to run its own course.

Treatment.—The practice recommended by this physician, and by Twining before and Aitken after him, was to abstain from bleeding, and trust to emetics and purgatives. The more modern practice, represented by Dr Manson, is to avoid purgatives and emetics, as aggravating rather than relieving the distress of the patient. He advises antipyrin or antifebrin, belladonna, or subcutaneous injection of morphia to meet the myalgia and arthralgia. Change of air and "tonics" are recommended during convalescence.

Liniments of chloroform, belladonna, or cajeput oil are often useful, and Dr Stedman found that the application of mustard plasters or blisters to the neck or to the loins gave great relief. When the joints remain stiff and painful after the subsidence of the disease, sulphur baths are said to be useful. After the bowels have been freely opened, a dose of laudanum, or Dover's powder, completes the cure.*

MEDITERRANEAN FEVER.—There is another exotic fever, which used to be known as Malta fever, but it is now ascertained to be identical with that known at Gibraltar as "Rock fever," and it has been met with in Sicily, Naples, on the Danube, in Constantinople, and at Massowah. Hence the term Mediterranean has been applied to the fever in the official nomenclature of the Registrar-General of the Navy and Army, drawn up by the College of Physicians (1896). It was formerly called gastric remittent and bilious remittent fever; and a new title, "Undulant Fever," was proposed by the late Dr Hughes.

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* For valuable observations on the clinical characters and the treatment of dengue, as well as of yellow fever, the reader is referred to Dr Shattuck's notes to the American translation of Strumpell's 'Handbook of Medicine.'

As in the case of relapsing fever and plague, the writer thinks it proper to mention that he has never seen a case of yellow fever or dengue, nor of the two other exotic affections described in this chapter.

This fever is endemic, not as a rule epidemic, and "miasmatic," not contagious in origin. It begins sharply, but continues a very lingering course of several weeks or several months, and is liable to repeated relapses. There is no diarrhœa, no exanthem, and a very irregular course of temperature. Muscular pains, profuse sweats, a swollen spleen, appear to be constant, together with acute pain and moderate swelling of various joints, particularly the sacro-iliac, and occasional acute inflammation of the epididymis or testicle.

Dr Bruce, who carefully studied the disease in 1887, has described a specific microbe, under the title *Micrococcus melitensis*, as constantly present in the spleen ('Practitioner,' vol. xxxiv, p. 101). It appears not to occur in the blood.

The course of the disease is very tedious, and it leads to marked anæmia, but its mortality is very small indeed—due to hyperpyrexia, to pneumonia, or to endocarditis.

After death the only constant condition is enlargement of the spleen. None of the lesions of enterica are present.

No efficient treatment is known. Quinine and salicylates have no effect on Malta fever. Antipyrin relieves the myalgia, arthralgia, and neuralgia, which are the most distressing symptoms.

A full account of this disorder, by Dr J. L. Notter, will be found in the second volume of Allbutt's 'System,' from which the above summary has been compiled. See also papers by Capt.-Surg. M. L. Hughes * ('Med.-Chir. Trans.,' 1896, vol. lxxix, p. 255), Prof. A. E. Wright, of Netley ('Brit. Med. Journ.,' April 15th, 22nd, and 29th, 1896), and Dr H. E. Durham ('J. of Path.,' 1898, vol. v, p. 377).

Beri-beri.—Another exotic specific disease, which has recently shown itself in the British islands, is known by the above name in Singhalese. It is endemic in Japan, where it is known as *Kakke*, in Corea, Shanghai and Hong Kong, Manila, Sumatra, Java, Singapore, and the whole of the Malay archipelago. It is common in Ceylon, and not unknown in India and Further India. In Africa it is met with in Madagascar, Mauritius, and Zanzibar, and on the river Congo; in America among the West India islands, in Brazil, and at Monte Video.

Lastly, Beri-beri has sometimes been imported in ships from China, Japan, or Singapore, into the port of London; and a severe outbreak of peripheral neuritis in the Richmond Asylum of Dublin, which was more or less constantly present from 1894 to 1897, has been explained to the satisfaction of competent observers as nothing else but Beri-beri.

The symptoms begin with remarkable depression, bodily and mental.

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* This distinguished officer was killed in the engagement near Colenso, in Natal, while doing his duty on Sir Redvers Buller's staff, 1899.

like that of influenza, but without the fever or acute symptoms. After some days or weeks, loss of power in the limbs appears; often suddenly, with numbness, tenderness, loss of knee-jerks, and the other symptoms of peripheral neuritis. At the same time there is anasarca, beginning in the face and feet, and gradually becoming general. The hands are also affected. and the patient suffers, beside the pain in his limbs, from palpitation and dyspepsia. After a variable time the œdema disappears with free diuresis, and then the atrophy of the paralysed muscles becomes apparent. They very slowly recover their volume and power, and convalescence may not be completed for many months, or even for a year.

These symptoms are not equally pronounced in every patient. In some anæmia is the most striking feature of the case, which would be taken for one of renal or cardiac dropsy until the urine and the heart were examined. In other cases there is only slight œdema, and the case presents itself as one of paraplegia or of muscular atrophy. In others again dyspnœa and palpitation are the predominant symptoms, and these are of the gravest import.

It is remarkable that pyrexia is often absent, certainly after the earlier period of the disease, and possibly throughout. There is no evidence of affection of the cerebral or spinal centres. The nerves are affected with a toxic inflammation like that produced by alcohol, or by the poison of diphtheria; and the effects—loss of power, anæsthesia, numbness, tickling, burning, formication, tenderness, with loss of knee-jerk and early appearance of the reaction of degeneration—are just those observed in other forms of multiple parenchymatous neuritis. The distribution is constantly in the legs, frequently in the arms, sometimes also in the trunk and face, and very rarely in the muscles of the eyes or neck.

The mortality varies greatly—from only 1 per cent. to more than half the cases. It is most often due either to syncope or to gradual cardiac dyspnœa; less frequently to dropsy—pleural or pericardial effusion, or œdema of the lungs. After death the nerves are found wasted and degenerated, and also the muscles they supply, and the heart is dilated, with vitreous degeneration of its fibres.*

The pathology of Beri-beri is uncertain. It is certainly not a form of malaria, nor of scurvy, nor is it caused by any poisonous food, nor by anæmia, which is only seldom a marked symptom. It does not depend on the presence of animal parasites. Numerous investigations have been undertaken by Pekelharing and other pathologists at the instance of the Government in the Dutch East Indies; but the micrococcus discovered is not yet generally acknowledged as either constant or pathogenic. Beri-beri does not appear to be directly contagious, and it does not protect against recurrence.

No specific treatment is known, and Dr Manson chiefly advises change of locality, and improved dietary. He treats the dropsy with laxatives and diuretics, followed by steel, the neuritis by strychnia, and the cardiac symptoms by nitrate of amyl or nitro-glycerine, by a brisk purge, and, in an urgent case, by venesection.

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* Manson, 'Tropical Diseases,' 1890, pp. 221—247.

ERYSIPELAS

> Ἐρυσίπελας ἔξωθεν μεν εἴσω τρέπεσθαι οὐκ ἀγαθόν, ἔσωθεν δὲ ἔξω ἀγαθόν.
>
> HIPPOCRATES, Aph. vi, 25.*

er uses of the term—A contagious disease—The specific microbe—Relation of the phlegmonous form to facial idiopathic erysipelas—Incubation and onset—the eruption of the face and fauces—its histology—the symptoms and course—Recurrent erysipelas—Occasional useful effects—Complications—Sequelæ—Diagnosis—Prognosis—Treatment.

ynonyms.—Febris erysipelatosa, Ignis sacer, Rosa, the Rose, St ony's fire—*Fr.* Érysipèle.—*Germ.* Rothlauf.

efinition.—A specific infective disease, characterised by an exanthem fever; of short course, often complicated by suppuration and septi ia, and always the result of invasion by a special micrococcus.

itherto all the diseases discussed have agreed in the essential features e group of diseases called Specific, and each has been readily defined eparated from any other malady. But now we come to less easily d categories.

rysipelas and Diphtheria have both been the subjects of much contro as to the extent to which each name should apply, and as to the ific" character of many cases. The difficulty arises in each case the prominence of the local lesion, and its likeness to non-specific mation.

ysipelas is frequently mentioned by Hippocrates as a form of acute mation of the skin, and the term has never since been lost in medical ure. There have, however, been great differences of opinion as to ffections should be included under this name.

me physicians, especially in France, have separated a "medical elas" of the face and head from the cases seen in surgical practice, sipelas attacking the limbs and the body after injuries. On the other many surgeons used to regard as "erysipelatous" almost every form ading inflammation of connective tissue, even including diffuse suppu of the post-peritoneal structures after operations on the rectum. iew is strongly objected to by Volkmann and other German writers. ill they allow the term "phlegmonous erysipelas" to cases of suppu or sloughing attended with intense redness of the skin, if the sub ous and intermuscular textures are involved. Again, the epithet

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r erysipelas to spread from the surface inwards is a bad sign, but for it to spread thin outwards is a good one.

"erysipelatous" has been applied to the septic peritonitis of puerperal women, and to the ascending suppuration of the urinary tract which follows necrosis of the vesical mucous membrane. Lastly, the relation between erysipelas and erythema was at one time much discussed. The latter term is applied to various forms of transient superficial dermatitis, which will be discussed in the last section of this work: but without doubt it has sometimes been confounded with slight cases of erysipelas.

Idiopathic facial erysipelas: its pathology.—All these questions must ultimately be settled by ætiological considerations. Erysipelas is a contagious disease. Of its propagation by contagion examples were recorded many years ago by Dr Wells, of St Thomas's Hospital, the author of the celebrated 'Essay on Dew.' For a long time Continental observers disputed the possibility of such an occurrence: but Velpeau recognised the fact, Volkmann cited more than a dozen cases in proof, and it is now universally admitted.

A striking series of recorded cases was brought before the Académie de Médecine in 1864 by Dr Blin. One of the surgeons at the Lariboisière Hospital had under his care two patients suffering from erysipelas, when he was himself seized with it. A medical friend from Guise visited him and fell ill after returning to that place, where no other case of the disease then existed. That gentleman's servant was attacked, and also a relative who came to see him. The latter gave erysipelas to his wife, and three members of another family who were repeatedly in contact with them during their illness suffered in their turn. From this family the disease spread to two Sisters of Mercy, and they conveyed it to the medical man who attended them. Lastly it passed from him to his daughter.

Erysipelas is not only a contagious, but also a specific disease, in the sense of being always dependent upon the entrance into the body of a definite *virus* from without. It is a mistake to suppose that the disease, like pyæmia, constantly becomes prevalent wherever surgical patients are crowded together under unfavourable conditions. In the Crimean war of 1853-4, in the Austro-Prussian war of 1866, and in the Franco-German war of 1870, repeated examples presented themselves of hospitals which it was impossible to keep healthy, and yet erysipelas never appeared. On the other hand, there have been many instances in which it has prevailed in a single ward of a hospital. Whether it ever spreads as an epidemic over an entire district, like typhus or scarlatina, is more doubtful. Between 1841 and 1854 this is said to have been the case in certain parts of the United States, but both Volkmann and Hirsch are of opinion that the supposed erysipelas was more probably diphtheria.

In a hospital the poison of erysipelas often clings to particular wards, and even to particular beds, with extreme obstinacy. Mr Savory, in 1873, recorded that during a small epidemic which occurred at St Bartholomew's, the disease almost always, in passing from one to another, attacked the nearest patient who had an open wound. The late Mr de Morgan, in Holmes' 'System of Surgery,' cites, on the authority of Dr Goodfellow, an extraordinary instance in which erysipelas spread in regular order throughout a ward of thirteen beds to almost every patient in turn, going down one side of the ward and then up the other side. The following case was observed by Mr de Morgan himself. It having been found at the Middlesex Hospital that patients occupying two adjacent beds with a window between them were particularly apt to be attacked, the suspicion arose that this might be due to the presence of a dustbin in the area below. It was cleaned out, and there were no further cases. Two years later the disease reappeared, and it was found that the dustbin had again become foul, but the adoption of the same measures as before rendered the beds again healthy. In this instance it might perhaps be doubted whether the impure air actually conveyed a poison to the patients, or whether it merely predisposed them to be attacked. But no such doubt attaches to another set of cases, recorded by Dr König, of Rostock, in the 'Arch. d. Heilkunde' for 1870. In the hospital of that town a small epidemic of erysipelas was clearly traced to infection from the cushion of the operating table. This cushion was deeply discoloured with blood, and from the day it was removed no fresh case occurred.

The contagion is undoubtedly conveyed not only by direct contact with a patient, but also by fomites, probably by the air, and there is reason to believe by the dead body.

So long ago as 1873, Dr J. Orth, of Bonn, recorded in the 'Arch. für experim. Path.' a series of experiments in which he infected rabbits by the subcutaneous injection of fluid taken from an erysipelatous bulla in man; he also transmitted the disease from rabbit to rabbit by inoculating with liquid from inflamed and œdematous parts of the skin, and even with blood from animals already infected. That erysipelas can be conveyed in a similar manner from one human being to another seems to be established by an old observation accidentally made by Doepp: he vaccinated nine children with lymph from a child who on the following day fell ill with erysipelas; and all were attacked by the latter disease.

Orth found micrococci in the infecting fluid with which he made his experiments on rabbits, and he succeeded in producing erysipelas by inoculating animals with micrococci that had undergone artificial cultivation. The presence of such organisms in erysipelas had, indeed, been previously demonstrated by Lukomsky and by Hüter, not only in tissues affected with the disease and in the œdematous fluid which satruates them, but even—in small numbers—in the blood. Subsequent investigations by Fehleisen and Koch in 1883 confirmed the constant presence of a special micrococcus occurring in chains, now named *Streptococcus erysipelatosus*. Cancerous tumours have been injected with a pure cultivation of this microbe, and erysipelas has followed as was intended. The streptococcus is often found in the thin serum of a bulla and pus of an abscess, and in the lymph channels; but is absent or only sparingly present in the blood. It is excessively minute, smaller than the granules observed in the lymph of vaccinia. Cultivation and inoculation seem to establish its specific distinction from *Streptococcus pyogenes*, although some competent critics still doubt it.

Thus all the tests enumerated in p. 18 are satisfied, and we may conclude that erysipelas is a "specific disease."

Phlegmonous erysipelas.—The question remains of the relations between the various forms of disease that have been included under the name of erysipelas. Volkmann admits that acute diffused suppuration, diphtheria, or puerperal fever, when introduced into a hospital free from erysipelas, is often followed by erysipelas of the face. He cites a case in which a man who had a severe gunshot wound of the foot died of "pseudo-erysipelas," which in several places had passed on to gangrene. A brother who attended him was attacked with erysipelas of the face, and so was a nurse; and several other persons became affected with phlegmonous inflammations. Such occurrences are the more striking because contagiousness is a far less marked feature of erysipelas than of many specific maladies. But just as in cultivation experiments there is great difficulty in keeping fluids free from accidental contamination with foreign bacteria, so diseased surfaces in living patients may afford a favourable nidus for the growth of a specific microbe, and thus erysipelas be grafted on suppuration. In phlegmonous erysipelas it is quite possible that *Streptococcus pyogenes* may be mingled with *Streptococcus erysipelatosus*, and hence the disease be due to a mixed infection.

It was formerly thought that erysipelas of the face and head, as physicians see it, differed from the surgical affection in appearing upon the unbroken skin, without any wound or abrasion. Trousseau, however, pointed out that it often starts from some slight breach of surface, as from a suppurating pimple at the angle of the eye, which the patient may have scratched, or from a trifling eczematous eruption on the nose, or from a fissure at the corner of the mouth, or even from a sore gum due to a decayed tooth. This view has since been adopted both by Volkmann and by Zülzer. A most careful record of observations for the purpose of testing its correctness was made by König. Among twenty-nine cases of erysipelas of the face or scalp which occurred in the inmates of a prison at Ziegenhain, fifteen were traceable to previous injury of the affected parts. The supposition is that the contagious microbe of the disease settles upon the spot which afterwards becomes the seat of erysipelatous inflammation, and that infection of the blood is secondary. The complete want of symmetry in the distribution of the rash furnishes a strong argument in favour of this view. Nevertheless one often fails, after careful search, to find a primary lesion, and is forced to admit that though a breach of the skin is the most frequent, it is not the only inlet for the virus. The outlet is probably the cast-off epithelium of the affected skin or mucous membrane.

Baumgarten found the streptococcus of erysipelas in the wards of a hospital, so that it may be in some cases a "miasmatic contagious" disease, like plague.

Predisposing causes.—The presence of a wound, combined with neglect of antiseptic precautions, favours the occurrence of the disease. Erysipelas is no doubt always derived from another case of the same disease; but it does not, as a rule, spread to healthy persons, and surgeons have often conveyed the contagion by hands, bedding, and vestments without suffering themselves.

Women are believed to be somewhat more liable to facial erysipelas than men, and it is supposed to be most common "in spring and fall." Drunkards are particularly exposed to it if they receive any injury, and so are patients suffering from Bright's disease.

Incubation.—The incubation of erysipelas appears to last from one to three or four days. In Fehleisen's experimental inoculations the period of incubation was from fifteen to sixty hours.

There is reason to believe that during this time local changes are actively going on, although there is no obvious inflammation of the skin. For it had long ago been noticed by Frank and by Chomel that the development of erysipelas of the face is often preceded by pain, tenderness, and swelling of the cervical glands; and the late Mr Busk, after close observation of a large number of cases, was convinced that this was an invariable occurrence. Perhaps inflammation is already going on, but the lymphatics carry off the exudation as fast as it appears. Doepp's cases of erysipelas following vaccination seem to show that the infective microzymes of the disease must be multiplying locally during incubation, for the lymph which conveyed erysipelas was taken from the arm of a child in whom the disease did not appear until the following day. Nor is there any difficulty in imagining that the blood may even at this early period contain the specific streptococci, whether by direct absorption through the veins or by transmission through the lymph-glands, and onwards through the thoracic duct. Before the outbreak of erysipelas the patient often feels ill, shivery, languid, or drowsy; his pulse is frequent and his temperature raised; he may suffer from nausea, vomiting, or diarrhœa, and sore throat very frequently accompanies the complaint.

On the other hand, Volkmann states that in many cases he has watched the development of erysipelas in patients, already in hospital, whose temperatures had been systematically observed for some time previously, and has never seen any prodromal symptoms whatever. In his opinion, whenever they seem to be present, the fact really is that the local affection has already begun, but in some deeply seated part, so that it remains undiscovered. Trousseau maintained that erysipelas of the face often starts from the mucous membrane of the palate and fauces, and reaches the skin by passing outwards through the nostrils. Such a course might well account for the early swelling of lymph-glands.

Dr Fagge observed a case in point in 1882. A man who had had jaundice for some time was admitted under his care on June 24th. His temperature was then 105°. He said that his febrile symptoms had begun with a slight rigor on the evening of the 22nd. There was no evidence of pylephlebitis or other local affection of the liver to account for the pyrexia. On the 26th the end of the nose was found to be red, and a blush of erysipelas soon spread over the face. It was then remembered that when he first came into the hospital he complained of sore throat, and that on the 25th the clinical clerk had noticed the submaxillary glands to be swollen.

Onset and exanthem.—The onset of erysipelas is usually sudden, and it is often marked by rigors. The late Dr Woodman (in his translation of Wunderlich's work) remarks that even in adults epileptiform convulsions are not uncommon. The temperature may rise in twelve hours to 104°, and usually reaches its *fastigium*, which may be at 105° or even 106°, within the first two or three days.

In some cases severe febrile symptoms may set in and last for a day or two before the rash appears. This "prodromal" period between that of incubation and the appearance of the exanthem is exactly what we notice in cases of scarlatina, measles, and smallpox. The writer has seen it last three days, with high temperature, severe headache, and thickly furred tongue.

Usually, however, within a few hours of the rise of temperature some part of the skin, usually near the angle of the eye or the ala of the nose, begins to burn and tingle. It soon becomes red and swollen, and the redness rapidly deepens into a crimson blush, which fades under pressure, but returns as soon as the pressure is removed. It spreads until in a day or two it may cover the whole of the face, and where it is spreading, the finger. On the other hand, where it is stationary, its edge fades off gradually into the healthy skin beyond. There is extreme swelling, especially where the skin is loose, as on the eyelids; these become converted into smooth, rounded, translucent tumours, and it is impossible to separate them so as to get a view of the eyeballs. The features are so altered that the patient cannot be recognised. The surface is tense and shining, though it may be made to pit by keeping up gentle pressure on it. There are often a few scattered vesicles, or blebs, which may reach a large size; and Volkmann confirms a statement originally made by Sanson that minute vesicles can always be seen with a lens.

For three or four days the disease may go on spreading, until, if it began upon the face or the head, it may cover the surface down to the root of the neck. Volkmann, however, remarks that the chin always remains untouched. The conditions which determine its advance in one line rather than another were carefully studied by Pfleger, whose views are endorsed by Zülzer. It would seem that this depends mainly upon the arrangement of the subcutaneous connective-tissue bundles; they everywhere interlace, so as to form rhomboidal meshes, but these are usually horizontal or oblique, whereas on the chin their direction is vertical. Pfleger also maintains that wherever the skin is tied down to the deeper parts the spread of erysipelas is retarded or arrested,—as, for example, along the crest of the ilium and Poupart's ligament. Erysipelas of the face and head seldom extends far upon the chest; but when the disease begins upon the trunk or upon a limb, it may spread until it has covered the whole body. If it goes on advancing, however, it subsides in the parts first attacked while it is springing up elsewhere; hence it is never a universal or a symmetrical exanthem. Beyond an affected area small islets of redness may not infrequently be seen, but these are always connected with it subcutaneously. Volkmann declares that erysipelas never gives rise to two or more patches at a distance from one another: it may, indeed, happen that in a case of double amputation both stumps are affected, or that in a case of erysipelas of the head the disease breaks out a few days later round a pimple on the leg; but such cases are probably due to multiple infection.

Erysipelas faucium.—During an epidemic of erysipelas in a hospital it is no uncommon thing for cases of sore throat to occur, which are evidently of the same nature, but in which the skin remains unaffected. An account of this form of the disease was given by Cornil, based upon a study of eighteen cases. He describes a shining, purple-red, œdematous swelling of the fauces, sometimes accompanied with the formation of bullæ. The tonsils often take no part in it. The lymph-glands below the jaws and in the neck are much enlarged. There is considerable pain in swallowing, and sometimes a profuse flow of saliva.

Erysipelas may begin at the nostrils and spread to the nares and pharynx, or in the throat and spread to the face, or at the eyes and spread to the nostrils.

Histology.—The minute anatomy of erysipelas was first studied by Biesiadecki, and more recently by Volkmann and Steudener. In the dead body the disease is so little marked, from the redness and swelling having almost completely disappeared, that Volkmann was surprised at finding the deeper strata of the cutis, as well as the subcutaneous tissue, infiltrated with enormous numbers of granular leucocytes. He gives a drawing of the microscopical appearances, in which the cells are seen packed closely side by side. In the more superficial strata, they are, however, scattered very sparingly. The structure of the bullæ was investigated at Vienna in 1868 by Dr Haight, of New York. He found them to be divided into loculi by irregular septa which are made up of cells of the rete, drawn out into long spindle-shaped and branching processes. The fluid of the bullæ contains numerous leucocytes, and is often converted into pus. The specific streptococci of the contagion are found in the fluid that fills the vesicles or blebs, and in greater numbers in the lymph-spaces of the affected cutis.

Symptoms.—While the local process is thus running its course the pyrexia continues, the temperature rising and falling irregularly, or remaining at nearly the same level. According to Reynolds it is frequently lower in the evening than in the morning. The pulse is quick, and it is generally soft and feeble; it may be dicrotic or intermittent. There is usually more or less delirium at night, and sometimes violent maniacal excitement. The patient often complains of headache, sleeplessness, and irritability of sight and hearing. He has no appetite, and is thirsty; the tongue is thickly coated, and there is often diarrhœa.

The urine is scanty, albuminous, and may often contain casts and blood. The writer looked for this in a series of cases when medical registrar at Guy's Hospital, and found it more often present than in any other fever.

The duration of erysipelas is variously stated by different writers; it is, in fact, uncertain. Billroth says it seldom reaches fourteen days; Volkmann puts it at about six or eight. When the disease wanders over the body and limbs it may run on for weeks, or even months. The final defervescence is usually sudden, the temperature falling to the normal point in a few hours.

The subsidence of the exanthem is also rapid, and then the skin becomes pale and flaccid, and shrivels; at the same time other parts of the surface may be at the height or the beginning of the morbid process. Volkmann and Steudener have investigated the histology of this stage of the process; they find that in the subcutaneous tissue the leucocytes disappear with extraordinary rapidity, breaking down in a few hours into a granular *débris:* in the superficial layers of the cutis they remain visible a little longer, but within a day or two all signs of tissue changes vanish. In the meantime the vesicles or bullæ have dried up into yellowish crusts. The cuticle subsequently desquamates, and is detached either in flakes or as a branny powder. When the scalp has been the seat of the disease, the hair falls out for a time but is soon reproduced.

But it does not always happen, even when a case of erysipelas ends in recovery, that the local affection subsides thus favourably. Delicate parts, as the eyelids, the prepuce, or the labia, sometimes slough. In other cases, when the swelling has gone down, abscesses form beneath the skin, and need lancing. Suppuration of the swollen lymph-glands is very rare.

Recurrence.—Erysipelas does not protect against its own recurrence. In surgical wards it used to be no uncommon thing for a patient to have two or even three successive attacks while a large wound was healing. Women sometimes have the disease once or oftener every year.

In course of time this "recurrent" or "habitual" erysipelas leads to a persistent thickening and induration of the nose, ears, or eyelids, curred again and again for several years. The same pathological process is seen in the production of elephantiasis.

Effect on other forms of inflammation.—A remarkable result of an attack of erysipelas is that it is sometimes followed by the disappearance of other long-standing affections of the skin. This seems to have been first noticed by Cazenave in cases of chronic eczema and lupus. But even sarcomatous growths may vanish in a similar way. Volkmann gives copies of photographs taken from a woman under the care of Dr Busch, who had several tumours on the face, varying in size from a hazel-nut to a pigeon's egg: a portion of one of them was excised, whereupon she was attacked with erysipelas, and this led to the absorption of all the rest. In two other patients enormous tumours of the cervical glands underwent marked decrease of size under similar circumstances: symptoms of collapse, however, set in, which in one case proved fatal, while in the other case, as recovery took place, the growth rapidly regained its former dimensions. This patient had been intentionally exposed to erysipelatous infection, in the hope that it might act beneficially upon her disease. The tumour from the former patient, who died, was examined histologically by Rindfleisch, who found that almost the whole of it had undergone fatty degeneration, so that only in certain portions could the structure of a round-cell sarcoma still be recognised.

Complications.—When there have been severe cerebral symptoms it was formerly supposed that inflammation had extended from the scalp or face to the membranes of the brain. Examination after death, however, has very rarely verified this suspicion. When the disease has spread inwards from the orbit through the sphenoidal fissure, the meningitis that followed has been probably the result of a mixed infection by other streptococci.

It is not uncommon for the disease, when it affects the fauces, to extend onwards to the larynx, and so to destroy life, unless tracheotomy obviates the danger. The folds at the entrance of the larynx are intensely œdematous, or infiltrated with pus. This was the immediate cause of death in the case of John Stuart Mill, who died at Avignon of faucial erysipelas.

In other cases the immediate cause of death is pneumonia or pleurisy. In the 'Guy's Hospital Reports' for 1861 Wilks recorded two cases in which erysipelas of the abdomen appeared to have set up fatal peritonitis.

Sometimes erysipelas of the fauces spreads by the Eustachian tube to the tympanum, and there excites suppuration with rupture of the membrane (Cheyne). A curious complication, twice observed in France by Larcher ('Arch. Génér.,' 1864), is ulcer of the duodenum, perhaps of the same pathology as that which sometimes follows burns and scalds. In a third example, reported by Malherbe ('Arch. Génér.,' 1865), there were ulcers in the lower part of the ileum. Bayer has related in the 'Arch. d. Heilkunde' for 1870 a case in which severe hæmorrhage from the bowels preceded death; the only lesion found at the autopsy was intense congestion of the ileum.

It has been questioned whether pyæmia is frequently associated with erysipelas in the same patient. Zülzer says that this is only the case in the phlegmonous form attended with diffuse suppuration of the connective tissue: and our observations at Guy's Hospital seem to bear out this statement. Volkmann, however, observed metastatic abscesses in more than one third of his fatal cases. There may have been an embolic and septicæmic process going on independently of the specific contagion.

Sequelæ.—Scars only follow "phlegmonous erysipelas;" but when repeated attacks affect the same part a chronic hypertrophy of the skin, with œdema durum of the subcutaneous tissues and lymphatic engorgement, sometimes follows; and thus the condition known as elephantiasis of the legs and the scrotum is often the result of recurrent erysipelas.

Various affections of the eyes may follow an attack of erysipelas—opacity of the cornea and, it has been said, optic neuritis.*

It is very rare for the febrile albuminuria which is so very frequent a symptom of this malady to persist, so that Bright's disease is scarcely ever to be traced to this origin. The writer has only met with a single undoubted instance of this result.

Diagnosis.—The recognition of erysipelas is easy apart from questions as to its relation, on the one hand, to phlegmonous dermatitis and diffused suppuration, and, on the other hand, to certain forms of erythema. Herpes zoster of the first division of the fifth nerve is sometimes mistaken for erysipelas,† and so is traumatic inflammation or acute eczema with œdema of the eyes or scrotum or prepuce.

Prognosis.—This is generally favourable for the cases of facial erysipelas that come under the care of physicians, except in old people and infants: in the latter it not infrequently appears at the umbilicus, and proves rapidly fatal. Even in surgical practice recovery so generally takes place that it is difficult, as Dr Wilks has remarked, to understand how it came to pass that the common form of certificate submitted to a magistrate in cases of slight wounds used to be that there was no danger, "unless erysipelas should ensue." It may be, as he suggests, that what really was pyæmia was often set down to erysipelas. There can be no doubt, however, that erysipelas itself was once far more fatal, at least in certain institutions, than it is at present; thus Volkmann says that in the old Hôtel Dieu at Paris it would often happen that the majority of surgical cases attacked by it ended in death. Probably the difference depends, first, on the segregation and antiseptic treatment of cases; and secondly, on the fact that patients suffering from erysipelas are now supplied with beef-tea and wine or brandy, instead of being bled or leeched and kept on low diet.

That when erysipelas spreads from the skin to mucous membranes it is more dangerous than when it spreads from within outwards is the aphorism of ancient medicine placed at the head of this chapter, and it is supported by modern experience.

In adults a fatal termination is likely to occur only when the patient has chronic disease of the kidneys, or has been intemperate, or is otherwise broken down in health. Before death the temperature usually rises to a great height, and sometimes it goes on rising for a short time afterwards.

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* See on this point, however, an abstract of numerous observations made in Russia which appeared in the 'London Medical Record' for November, 1888, p. 473.

† I once found lying in my ward a youth whose face was covered with flour, through which a diffused redness was visible, while the eyelids were enormously swollen. The first glance, however, showed that the affection scarcely, if at all, passed the middle line of the forehead; and, according to the account which the patient himself gave, it had begun with an eruption of vesicles. I therefore made a confident diagnosis that the case was one of zoster, and dropped atropine into the *eye*, on account of the danger of iritis. At my next *visit the redness and swelling had disappeared* without desquamation, and the vesicles had [illegible] —C. H. F.

Treatment.—In this country we believe that the tincture of steel has a marked influence in checking erysipelas. Mr de Morgan, from his experience at the Middlesex Hospital, spoke most positively of its efficacy in reducing the duration of the disease, so that it subsides in from two to four days, instead of lasting a week or ten days. He gave at least a fluid drachm or a drachm and a half daily, and sometimes as much as an ounce and a half. On the Continent it is more usual to give quinine.

When a case comes under observation at the very commencement of the disease an emetic or a mercurial purge is believed to be useful, and Dr Ringer thinks that aconite administered at this period may cut short the attack. Given after the inflammation has appeared it usually brings down the temperature, but has no other useful effect, and possibly may do harm.

Many years ago Mr Higginbottom introduced the practice of applying nitrate of silver round the circumference of a patch of erysipelas, in the belief that its spread might in this way be arrested. This was termed the "ectrotic" method, and there are still some who have faith in it, including Volkmann, who prefers the silver salt to the tincture of iodine, which has since been used with a similar object. The part must be first carefully washed with soap and water, or with a solution of soda or potash, so as to remove oily matter from its surface. It is then brushed over with a solution of the nitrate in distilled water (one to eight or ten parts) for a distance of some inches round the reddened area. Sometimes the disease ceases to spread, and on the following morning defervescence occurs, as is shown in charts given by Volkmann.

The application of collodion all over the affected surface is said to be useful as a palliative. But the traditional treatment is to dust flour over the affected skin; and this is certainly a valuable means of relieving the local smarting and irritation, probably by excluding the air. Eichhorst strongly recommends the local application of carbolic acid in oil of turpentine (1 to 15) painted on the affected skin every hour. Carbolic oil (1 part of phenol to 20 of olive oil) has been used with the same object of providing protection from the air and antiseptic action at the same time. Lead lotion used to be a favourite and useful application; now hot boracic solution is more used.

When violent delirium accompanies erysipelas of the head, an ice-bag is said to give great relief. If the swelling is so great that gangrene threatens, a series of minute incisions is the most likely means of preventing it.

It is not often necessary to employ cold baths in the treatment of erysipelas; for the pyrexia, although reaching a great height, usually subsides too quickly to be in itself a source of danger. But if a high temperature is maintained, one must adopt measures to reduce it. Volkmann gives a chart of a case in which a cold bath was given on the ninth day, when the thermometer indicated 105·8°; in the course of the following week this treatment was repeated thirteen times, and the patient recovered.

In severe cases wine and brandy are required, and should be given freely. Opium is also a valuable sedative, and does not appear to be countermanded by the albuminuria, if it be febrile and not due to previous Bright's disease. In the latter case the prognosis is bad, whatever treatment be adopted.

When the larynx is affected, tracheotomy is probably the safest and most effectual treatment.

DIPHTHERIA

> "Inde ubi per fauces pectus complerat et ipsum
> Morbida vis in cor mœstum confluxerit ægris,
> Omnia tum vero vitaï claustra lababant,
> Spiritus ore foras tetrum volebat odorem."
> LUCRETIUS.

History of the disease—Its various aspects—Its anatomy and pathology—Relation to croup and to "diphtheritic inflammation"—Ætiology—the specific bacillus—transference of the contagion—Course and clinical symptoms—Varieties—Complications and sequelæ—albuminuria—paralysis—Prognosis—Treatment by drugs, by tracheotomy, and by antitoxic serum.

Synonyms.—Ulcera Syriaca ? (Aretæus, *c.* 70 A.D.), Ulcera pestifera in tonsillis ? (Paulus Ægineta, *c.* 680 A.D.), Epidemica gutturis lues, Angina puerorum epidemica (Bartholinus, 1646), Putrid sore throat attended with ulcers (Fothergill, 1748).—*Fr.* Mal de gorge gangréneux (Chomel), Angine couenneuse (Louis), Diphthérie (Bretonneau, 1821).—*Germ.* Diphtheria, Cynanche contagiosa (Senator), Häutige Bräune, Bösartige Rachenbräune.—*Ital.* Morbus strangulatorius (Cletus, 1636).—*Sp.* Angina maligna (Heredia, 1673), Garrodillo.

Definition.—A specific infectious fever, with membranous inflammation of the throat and respiratory passages due to a specific microbe.

History.—In the years 1855-6 a severe form of angina became prevalent in England, and in some places was epidemic. It was known as the Boulogne sore throat, and resembled scarlet fever in being generally attended with an affection of the fauces, but differed from it in the exudation being membranous, and in the absence of a rash. Many experienced physicians declared that they had never seen this disease before; and although a search into medical literature brought to light several instances of its occurrence, in both a sporadic and an epidemic form, yet within the present century it had never before appeared in this country as an epidemic disorder. In France it had been well known since Bretonneau, of Tours, described it in 1821 as *diphthérite* (διφθέρα, leather or membrane).

It is impossible to distinguish, in the accounts of the ancients, between the sore throat of scarlatina and that of diphtheria, or even of syphilis. But undoubted epidemics of diphtheria occurred in Spain in the seventeenth century, and were described in 1614 by Mercatus, physician to King Philip III, and in 1670 by Heredia, physician to Philip IV. It spread to the Spanish provinces in Italy, and was epidemic at Naples in 1618, where it was described by Cortesius (1625), and by Ætius Cletus (1636). Soon after it was recognised by the celebrated Danish anatomist Bartholinus (1646). It probably appeared in Edinburgh in 1733, according to Fothergill, from whose treatise on 'The Putrid Sore Throat attended with demic at Bromley, in 1747 one at Greenwich, and in 1749 one in Cornwall, which was described in the 'Philosophical Transactions' by Dr Starr. It reappeared as an epidemic in South Wales in 1849.

There is reason to believe that the *cynanche maligna* of the older writers included not only cases of scarlatina anginosa, but also some which would now be regarded as diphtherial.

Since 1855 this disorder has constantly prevailed with more or less severity in different parts of England, as well as in Europe and America. There is no doubt, writes the Medical Officer to the Privy Council, that apart from diagnosis and nomenclature the mortality from and the prevalence of Diphtheria have been increasing between 1870 and 1895, and this increase is most marked since 1890.

Doubts as to its nature and mode of propagation, and particularly as to its relation to certain other diseases, long prevailed; but since the first edition of the present work was published, they have for the most part been decided, so that a more certain and connected account of Diphtheria can now be given than was possible in 1883.

Views of its nature.—As the term diphtheria implies, its most striking character is a sore throat with a membranous exudation spread over the tonsils, the larynx, and other mucous membranes.

By Bretonneau and his followers great stress was laid upon the fact that this false membrane could be detached from the fauces, leaving the mucosa reddened and ecchymosed, or at the utmost slightly excoriated. They admitted that the ash-grey membrane often simulated sloughing of the parts it covered; and, indeed, that the disease had long been known as gangrenous angina, or malignant sore throat. But they declared that such appearances were misleading, and that no considerable loss of substance occurred. Finding, moreover, that in many cases the false membrane spread down into the larynx, they recognised that the complaint previously known as "membranous croup" was only a form of diphtheria.

By German writers the subject was developed in a different direction. Virchow, in the first volume of his well-known 'Archiv' (published in 1847), distinguished between a "croupous" form of inflammation in general and a "diphtheritic" one. In the former, he said, the exudation lay free upon the surface of the mucous membrane; but in the latter its seat was within the epithelium, and generally caused sloughing as the result of its presence. These definitions have led to great confusion. A good description of the characters of "diphtheritic inflammation," in the sense attributed to it by the great leader of the Berlin school, was given by Rindfleisch in his 'Pathological Histology.' He made it consist of an infiltration of new-formed cells into the subepithelial corium (the *mucosa*); and, following an idea originally suggested by Buhl, he taught that this infiltration compresses the blood-vessels, and so arrests the circulation through the affected parts, brings their nutrition to a standstill, and deprives them of life. As examples of such an affection, he cites the putrid inflammation of the urinary bladder that is set up by decomposition of stagnant urine, the more severe forms of dysentery, and the dangerous inflammation of the uterus and vagina which may occur immediately after parturition; and adds that a similar condition may be met with upon other free surfaces: upon the skin, as in the more destructive kinds of variolous eruption, which lead to permanent pitting; and upon wounds, as in hospital gangrene. It must be admitted that all these forms of inflammation have characters in common, and deserve to be known by a special name (cf. p. 53). But the objection to calling this process "diphtheritic" is that the disease "diphtheria" must be excluded. Accordingly, Rindfleisch describes diphtheria as "pharyngeal croup." He gives a full and accurate account of the histological characters of the pellicle or false membrane of what we call diphtheria. On the palate and tonsils he says they consist, not of fibrin, but entirely of hyaline cells which have become fused together. In the air-passages, on the other hand, he describes the pellicle as laminated, and consisting of layers of cells which alternate at intervals with layers of fibrin.

This histological account is perfectly accurate; but the fact that in the very same case of diphtheria the membrane on the tonsils and that in the larynx differ in their histology shows that we cannot define the disease by its anatomy alone.

In the same patient we often find, after death, upon the fauces a thin, grey, ragged and decomposing membrane, which bled and stank during life—and the same characters mark the disease when it spreads from the palate and tonsils to the nares and the pharynx; on the posterior surface of the epiglottis, in the larynx and the trachea, we find a thick, white, firm, and continuous layer with abundance of fibrin, along with leucocytes, but with no blood-discs; and lastly, in the bronchial tubes we find only scanty shreds of membrane with abundance of mucus and pus.

Moreover, in the fauces themselves, the morbid changes present wide variations in different cases of the disease. There is, in the first place, "*diphtheria sine diphtheriâ*;" in which the tonsils and uvula are merely reddened and affected with catarrhal inflammation without any "false membrane." This is recognised as diphtheria, because it occurs in the same family simultaneously with the more severe forms. Even when membranes are present, they differ in extent and thickness. Sometimes they are thick and white, sometimes thin, grey, and ragged, with hæmorrhage and apparent necrosis. It is quite true that in some cases which presented during life the appearance of sphacelus—the uvula and tonsils in a state of "putrid dissolution"—the fauces are found after all to be intact at the autopsy, or when the patient recovers; but all recent writers admit that Bretonneau went too far in denying that gangrene ever occurs.

The definition of diphtheria as a disease must rest, not on its histological characters, but on its pathology and symptoms—its natural history.

The epidemic course, the infective power, the kind of fever, the effect on the kidneys, and the sequent paralysis—these are the distinctive characters which mark the Boulogne sore throat or Cynanche contagiosa or Diphtheria, whether in the fauces or the larynx; and the conclusion thus led up to by clinical observation is now confirmed by the discovery of the specific microbe to be presently described.

It is certainly quite distinct from what is called diphtheritic inflammation by most German pathologists, and, we shall see reason to believe, includes all that is called "croupous" by them, or "croup" by physicians in England and France.

*Relation of diphtheria to membranous croup.**—In 1765 Dr Francis

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* *Synonyms.*—Cynanche trachealis—Cynanche stridula—Angina suffocatoria—Morbus strangulatorius.—*Fr.* Angine couenneuse trachéale ou membraneuse; le croup.—*Germ.* Häutige Bräune; der Croup.

Home, of Edinburgh, published a tract of sixty pages, an 'Inquiry into the Nature, Causes, and Cure of the Croup.' This disease, as he said, was then unrecognised by medical writers, although it was known to the common people of Scotland by several distinct names, of which "croup" is one. In each of the *post-mortem* examinations which he made he found the trachea lined by a more or less complete membranous layer. Thirty-six years later, in 1801, another Scotch physician, Dr John Cheyne, of Dublin, wrote on the same subject a work which has become classic; his views upon the pathology of croup were the same as those of Home.

The word Croup is of English origin, and applies to the peculiar sound of the breathing in this disorder. It is properly a clinical term, but has unfortunately been associated with the pathological condition of membranous laryngitis. The question really started by Bretonneau's observation was whether the stridor and dyspnœa in children, which was known as "croup," was due to diphtheria invading the trachea. This was what Bretonneau's memoirs on diphtheria (1821-6) maintained, and the view was adopted by his pupil Trousseau, by Guersent, Barthez, and other French physicians. In England it was for a long time repudiated, but it has now proved to be correct.

Both Home and Cheyne were perfectly acquainted with the fact that the disease which they described was liable to be confounded with one which affected the larynx secondarily, having its original seat in the fauces. Home, in quoting Dr Hare's graphic account of the "morbus strangulatorius" in Cornwall (which was epidemic diphtheria in its most typical form), says that that complaint "appears more nearly allied to the malignant sore throat, although it sometimes attacked the trachea." And Cheyne begins his section on diagnosis by remarking that he had seen several children, whom he would have supposed to be in the second stage of croup had he not discovered sloughs upon the tonsils and uvula.

It was long believed that the false membranes in croup and in diphtheria presented constant differences, microscopical and chemical; but we now know that in diphtheria itself they vary in their appearance, in the relation which they bear to subjacent parts, and even in their histological characters, according to the part of the mucous tract upon which they are developed. This fact overthrows all the histological distinctions between the two diseases.

Another supposed distinction between diphtheria and croup was that a dry, brown tongue, sordes on the lips, petechiæ on the skin, internal hæmorrhages, and, above all, albuminuria are only present in the former disease. In many cases of diphtheria, however, no such symptoms show themselves: in others not until several days have elapsed; and since "croup," *i.e.* laryngeal diphtheria, is quickly fatal by suffocation, there is less time for development of septicæmia. Moreover absorption is far more rapid from the fauces than from the larynx.

Notwithstanding Dr Fagge's arguments in the 'Medico-Chir. Trans.' for 1879, and in the first edition of the present work, there is now general agreement that the disease as we see it in this country is one—faucial diphtheria or laryngeal diphtheria or both. Traumatic cases of membranous laryngitis may occur,* and possibly idiopathic cases of the same kind; but

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* In the 'Guy's Hospital Reports' for 1877 (p. 384) seven cases were recorded by Dr Fagge of "false membrane" on the fauces or air-passages as the result of scalds of the

"croup" expresses a certain combination of symptoms, and though these may depend on diphtherial laryngitis, they may equally depend on acute laryngeal catarrh or on spasm. It is therefore most undesirable to use "croup" as a pathological term in, for instance, "croupous" pneumonia.

Diphtheria, then, we conclude to be a general or "constitutional" disorder, producing, like scarlatina, a specific inflammation of the fauces and larynx. The local nidus of the contagion is the source of secondary septicæmia. The so-called membrane is also a specific product, and is not comparable to the fibrinous exudation on the surface of a serous membrane.

Ætiology.—Diphtheria is always acquired by contagion. When a patient suffering from diphtheria is admitted into a hospital, house surgeons, nurses, or patients in the same ward are often attacked by the same disease. Sir William Jenner relates several instances in which patients sent into the country infected those with whom they came into contact. The epidemic in East Kent in 1856 was ushered in by a striking instance. "No case of diphtheria had ever been seen in Folkestone during my time," says Mr Eastes, "until Isabella W—, aged 4¾, arrived from Boulogne on the evening of July 2nd, being then in an advanced stage of the disease. She died on the following day. On the 6th her sister, aged ten, was attacked, who had always resided on the East Cliff; another case occurred in the same house three days after, and they all terminated fatally." But the most conclusive argument is furnished by the unhappily frequent instances in which a medical man has been attacked by diphtheria, immediately after having had phlegm coughed into his mouth or nose by a patient whose fauces he was examining; or after having used his lips to inflate his patient's lungs, or blow through a tracheotomy tube. Oertel mentions by name five physicians whose lives were thus sacrificed, among whom was Valleix, the writer on neuralgia. Lamentable cases of death from such self-devotion are not rare in hospitals. It was believed that the late Princess Alice of Hesse contracted the disease by kissing her child who was ill of diphtheria.

In the 'Guy's Hospital Reports' for 1877 Dr Fagge recorded a series of fifty cases of what would be regarded as diphtheria in the common acceptation of the term; and whereas eleven of fifteen cases in which the larynx was free could be brought into connection with other cases (by either ascent or descent), only eight out of thirty-five cases in which the air-passages were involved could be so brought; moreover these eight cases were cases in which the fauces were severely affected. Other facts show

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throat by boiling water, which the children had sucked from the spout of a teapot or kettle; one case of a boy who got a bean into his right bronchus, and whose larynx and trachea were coated with lymph; two cases of children whose fauces were irritated, one by a piece of hot potato lodging in the throat, the other by a burning stick, and in whom membrane formed; one case of a man aged twenty-four who was admitted for a cut throat, and who died of a plastic inflammation of the larynx, trachea, and bronchial tubes; three cases in adults in which membranous laryngitis was secondary to cancer of the pharynx, tubercular ulceration of the vocal cords, and syphilitic disease, for which tracheotomy had been performed; and lastly, two cases, both in adults, in which a similar affection was associated with pneumonia. Moreover both Rietz and Oertel found it easy to set up a plastic inflammation of the trachea in dogs and rabbits by dropping a few minims of Liquor Ammoniæ into it through an external wound. Oertel performed this experiment on seventeen animals, and succeeded in every instance in producing artificial "croup"—*i. e.* a traumatic membranous laryngitis.

that when diphtheria attacks the air-passages it is less contagious than when it attacks the fauces.

How soon a patient ceases to be infectious is not yet known. The disease has been communicated by a child who had apparently been well for three weeks, and there is unfortunately no doubt that the duration of infection is sometimes longer still.

It appears that in about only half the cases have all the specific bacteria disappeared within three days of the patient being well and the fauces clear; in the great majority of the remainder they are all gone by the end of three weeks, but in a small residue they are still to be found after three, four, or even five weeks. (See Goodall and Washbourn's 'Infectious Diseases,' p. 134.)

The specific microphyte.—Since modern methods have been applied to the study of infectious diseases, the whole subject of diphtheria has assumed a new shape. Buhl in 1867, and Hueter and Oertel independently in 1868, discovered in the "false membrane" of diphtheria micrococci,* either separate or joined in dumb-bells, in chains, or as *zooglœa*; and mixed with these were some rod-shaped bacilli. It is the bacilli which are now proved by their constant presence and by inoculation on animals to be the specific origin of diphtheria. They are found not only in the mucus of diphtheria (both within the epithelial cells and in the leucocytes), but also in the adjacent structures; they extend beyond the limits of the disease as apparent to the naked eye, and they block up the lymphatic channels. Löffler finds that the characteristic bacillus of diphtheria is only present in the affected mucous membranes, not in the blood or in the kidneys, where previous observers had reported its presence. Oertel passed on the infection from one animal to another, choosing sometimes the trachea, and sometimes the muscles of the neck or chest, as the seat of inoculation; and after six transmissions he obtained a product capable of giving rise to the formation of a false membrane in the air-passages of the last animal experimented on.†

The true bacillus of diphtheria was probably seen by Professor Klebs in 1883, and his name is therefore conjoined with that of Löffler by many pathologists in naming the specific microbe. But it is to the latter investigator that is due the demonstration of the characters by which it can be identified, its size and shape, its reactions to dyes, and its appearance when cultivated. Löffler's observations were amply confirmed by Klein in England, by Roux and Yersin in France, and many pathologists since.

The microbe of diphtheria is a rather long (4—5 μ), blunt-ended, often somewhat curved rod. It frequently occurs as two united laterally or end to end. A shorter variety has been described (1·5—3 μ), and has been supposed to be less virulent in its action: but the two may exist together, and there seems to be no reason to suppose that they differ in their effects.

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* It is important to remember that the first observations of Hueter were made, not upon cases of "diphtheria," as we define the disease, but upon those of hospital gangrene. In other words, his facts apply rather to so-called "diphtheritic inflammation" than to true "diphtheria."

† "Experimentelle Untersuchungen ü. Diphtherie," 'Deutsches Arch. f. klin. Med.,' 1871, Bd. viii; Roux ('Annales de l'Institut Pasteur,' 1888, Nos. 11 and 12); Cohn ('Beiträge zur Phys. der Pflanzen,' 2tes Heft, pp. 164 *et seq.*); Eberth ('Zur Kenntniss bact. Mycosen,' 1872); Klein ('Micro-organisms and Disease'); Crookshank ('Bacteriology,' pp. 117, 136, 177); Kanthack ('Allbutt's System,' vol. i, p. 718).

The bacillus is not motile; it stains readily, and is readily cultivated on blood-serum and other media. It must, however, be admitted that the bacteriological diagnosis, though practically most useful, is occasionally difficult, even to the most practised observers. Bacilli of "pseudo-diphtheria" have been described, particularly "Hoffman's bacillus," which closely resembles the short form, and the "xerosis bacillus," which is found in diphtherial and other forms of inflammation of the conjunctiva. Those most experienced in microscopical and cultivational observation admit that the distinction between genuine and false is sometimes almost impossible.

Diphtheria, however, though a specific bacterial disease, is clinically the result not only of the invasion and local multiplication of the microbe on the fauces or other parts of mucous membranes, but also of the chemical products of the growths. These toxines (p. 22), secreted by the bacilli, and not the organisms themselves, are the cause of the disturbance in the heart, kidneys, and nerves, which make up the clinical composite of diphtheria. This part of the pathology has been worked out by Dr Sidney Martin, who confirms the results of Roux and Yersin, that the inoculation with the chemical products of Löffler's bacillus will produce diphtherial paralysis in rabbits and guinea-pigs. Dr Martin finds that the efficient toxine is not a ptomaine, but an albumose.

Transference of the contagion.—Probably in most cases this takes place by direct contact, as to the mouth and nostrils of a house surgeon or nurse, from the throat of a diphtherial child; or in the case of a mother or sister nursing and kissing an infant, or by using infected handkerchiefs, or cups or spoons, or tracheotomy tubes. Bedding and clothes may thus convey the contagion: to name one instance only, the late Sir Richard Thorne knew the despatch of a pillow from an infected house to a place free from any affection of the kind to be followed within a few days by fatal diphtheria.

Another medium of infection was ascertained by Dr Power in 1878 to be milk, which Klein has shown to be an excellent medium for cultivating Löffler's bacillus at from 64° to 68° Fahr. Probably the origin of the disease in these cases (as in the parallel instance of tuberculosis) is usually in the cows, which are liable to diphtheria. Another animal in which it appears (apart from experimental inoculation) is the cat, and Dr Klein has shown this to be another source of possible infection. Dr George Turner several years ago described tracheal diphtheria in pigeons, and more recently infection has with some probability been traced to canaries and other cage birds.

As above stated, the specific bacilli of diphtheria sometimes continue to infect the fauces long after all toxic symptoms have disappeared, and thus a patient convalescent or restored to health may yet be the means of conveying the disease to others. Moreover there is no doubt that persons may contract the infection and yet resist its effects, so that apparently healthy companions may convey the disease to others more susceptible.

Auxiliary causes.—It must be admitted that diphtheria often springs up in isolated houses, but the same thing is likewise true of all the exanthemata. It is generally believed that diphtheria is favoured by the effluvium from foul sewers or cesspools. Scarlet fever, however, is also associated with bad drains and foul smells, and probably in the same way—namely, by these conditions producing a sore throat, on which the germs of scarlatina or of diphtheria alight and find a favourable nidus; or perhaps we should rather say, on which they are not opposed by the antagonism of healthy tissues (cf. p. 51).

It is possible that when diphtheria is epidemic, and when the specific contagion has perhaps already been introduced into the system, exposure to cold may act as an exciting cause in bringing out the lesion on the fauces.

Dr Yeats (ed. 'Med. Journ.,' 1876) relates how several persons at Auchtergaven, in Perthshire, were attacked between March and June, 1875, who had been working late in their gardens, or playing croquet on a damp lawn, or driving after sunset; while others, who had been confined to bed for a considerable time from other causes, were infected on their first appearance in the open air.

The poor and the rich are probably, in proportion to their numbers, equally liable to diphtheria. It is notorious that the disease often carries off very healthy and robust-looking children. There are more cases in girls than in boys, probably, as Sir R. Thorne says, because they nurse and kiss the babies. Diphtheria is far more apt to attack *children* under twelve years of age than older persons, and is most common as well as most fatal between two and five.

It is doubtful whether one *season* of the year rather than another is favourable to the spread of diphtheria. Fothergill found epidemic sore throat far more frequent from September to December inclusive: and this agrees with the experience of the Registrar-General, that most deaths take place in the last three months of the year. According to Dr W. H. Power, cases are most frequent in October and November. The number rises in the latter weeks of September, and falls again by the middle of December. Cold winters have not hindered its epidemic diffusion in London; and it has often prevailed during the summer. Dr Newsholme has shown that epidemics of diphtheria are more common in dry than in wet seasons, and are not related to damp clay soils ('Epidemic Diphtheria,' 1898).

Diphtheria is of much less frequent occurrence in the tropics than in the temperate zone. Geological conditions of the soil seem to have no part in its causation. In England it is most prevalent in London and the eastern counties, and in North Wales; while the mortality from it is below the average in the counties north of the Humber and in the central Midlands—including the manufacturing districts of Tyneside, Lancashire, and the West Riding, Birmingham, and the Black Country, and also South Wales.*

The importance of contact in schools as a means of spreading diphtheria has long been recognised, since Dr Power drew attention to it with examples at Pirbright, in Surrey, and at Coggeshall, in Essex.

Diphtheria as a complication of other specific fevers.—In the chapters on measles and on scarlet fever it was mentioned that each of those complaints is now and then complicated by the appearance of membrane on the fauces, which is probably true diphtheria.† The writer saw a case of rubella in a young man of twenty, followed by diphtheria; and it has occasionally followed enteric fever.

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* See Dr E. Barnes' paper, 'Brit. Med. Journ.,' July 28th, 1888, and also Dr Longstaff's results ('Studies in Statistics,' Stanford, 1891).

† The following fact related by Dr Dewes, of Coventry ('Lancet,' 1875), tends to show that both the specific contagia may be present in some cases of this kind. Two brothers had been in succession attacked by scarlet fever, and had been separated from one another throughout the course of the disease. The elder had remained at his school to be nursed; the younger had been sent to a cottage in the country. The former passed through a mild form of the disease; the latter had it severely, and also had his

Protection.—Does the fact that a person has had diphtheria imply subsequent immunity from the disease? In Simon's 'Report' for 1859 several cases are recorded of patients who suffered twice, at intervals of two or three months or longer; and it is to be noted that the second attacks were more severe than the first ones. Second attacks are not uncommon in children, according to Dr Eustace Smith. Apart from second attacks, relapses before complete recovery are occasionally seen (about 1·5 per cent. in more than 1000 cases observed by Dr Goodall at the East London Hospital).

Incubation.—The incubation of diphtheria is short. It was said by Trousseau to be from two to seven days, Oertel put it at from two to five days, and Goodall and Washbourn at two to six. One instance is related by Jenner of a girl who sickened eight days after having been sent into the country from a house where her brother and her sister had been attacked. Where there has been a direct transference of the poison from one person's fauces to another's the period seems to be much shorter: Valleix had a pellicular deposit on the tonsil the day after he became infected with the disease, and died in forty-eight hours.

Clinical varieties.—Diphtheria may occur under two different forms, but they are connected by still more numerous intermediate cases. In one it has the characters of an infective fever, epidemic in course, contagious in origin, with febrile albuminuria, and "typhoid," *i. e.* septicæmic, symptoms; it affects the fauces, and the membrane is grey and bleeding when cast off. This answers to the putrid sore throat of early writers.

In the other clinical form there is less constitutional disturbance, the cases are less epidemic, though very contagious; the affection begins in the larynx, and it forms more fibrinous membranes. These cases answer to membranous "croup."

In the cases which combine both characters the membrane begins in the fauces, and afterwards descends to the larynx, trachea, and bronchi.

Beside these well-marked and severe cases, many occur in which the symptoms are very mild, sometimes so mild that the occurrence of slight paralysis is the first characteristic symptom noticed.

Faucial diphtheria.—The disease sometimes begins insidiously, with anorexia, headache, lassitude, and loss of energy; but occasionally with sudden access of fever and rigors, nausea or vomiting, and acceleration of pulse. There may from the first be difficulty of swallowing, the sensation varying from a slight pricking up to darting pain, which may prevent the patient from taking food, but this is rarely so severe as in quinsy. On looking at the fauces one finds that the uvula, the palate, and the back of the pharynx are more or less swollen and of a red or violet colour. After a few hours, or at latest two days, one or more whitish-grey spots begin to appear on some of those parts; they are at first small, and they may remain of the same size for several days. From an early period the lymph-

fauces covered with diphtheritic exudation. After a time the boy at the cottage sickened with scarlet fever, and he too had a diphtheritic throat. When both brothers had been convalescent for a fortnight they were allowed to be together, and even to lie in the same bed. Presently the elder one fell ill of diphtheria with laryngeal complications, and died on the third day.

glands of the neck, especially those near the angles of the jaws, become swollen and painful.

In mild cases of diphtheria the febrile disturbance is slight and of short duration. The false membrane is quickly cast off, and the patient recovers in two or three days.

But, as a rule, the disease takes a less favourable course. The fever continues, the temperature remaining at 102°, or mounting higher; if it should have fallen, it rises again on the fourth, fifth, or sixth day. Death from hyperpyrexia is almost unknown in diphtheria; and in severe cases, particularly in young children, the temperature is sometimes so low that it must be raised by artificial means. The urine is moderately febrile in character, and decidedly albuminous.

The diphtherial spots on the fauces run together, and extend in every direction; so that the tonsils, the uvula, and all the visible parts of the fauces may in a few hours be covered with membrane. The cervical and submaxillary lymph-glands swell still more, particularly those at the angle of the jaw, just opposite the tonsil. In severe cases the tissues are infiltrated with inflammatory œdema, so that the hollow of the neck is obliterated, and the whole space from the chin to the sternum is occupied by a uniform brawny mass, with the skin red and shining as in erysipelas. Before long the membrane begins to soften and decompose, becoming darker in colour, and separating here and there in shreds. The patient's breath becomes horribly foul, and all the symptoms of septicæmia develop. Under treatment gradual improvement and ultimate recovery may ensue, but the case is long an anxious one, and convalescence is slow and uncertain, as after enteric fever.

In the worst cases the throat bleeds as the membranes separate, and the fœtor becomes unbearable. A thin discharge runs from the corners of the mouth, excoriating the skin, and causing ulcers, which become covered with a greyish-white pellicle; it is not uncommon for sore places behind the ears, or in the creases of the skin, in the groin or elsewhere, to become diphtherial. If a blister should unfortunately have been applied, the raw surface becomes membranous; and leech-bites are apt to pass into unhealthy spreading ulcers.

Along with these grave local symptoms, no less grave constitutional disturbance marks the progress of the case. The pulse rises to 120 or more in the minute, and becomes daily smaller and weaker. The countenance rapidly acquires a waxy pallor, and the muscular power is more diminished than in any other febrile disorder. Purpuric spots sometimes appear upon the skin. The tongue becomes dry and brown, and sordes collect on the teeth and lips.

Occasionally there is delirium, but as a rule the mind remains perfectly clear. At length the temperature falls below normal, to 97° or 96° F., and the pulse becomes irregular and intermittent, falling perhaps to 50 in the minute. Death may either occur very gradually by asthenia, or more often it takes place suddenly while the patient is in the act of sitting up in bed or making some other slight movement. Such cases generally terminate between the tenth and the fourteenth days. The condition is one of septicæmia. Pneumonia, or œdema of the lungs, is often the immediate cause of death.

More latent cases.—Even when characteristic diphtheritic patches exist on the mucous membrane of the fauces the real nature of the disease may be overlooked, because the patient does not complain of difficulty in swallowing or of pain in the throat. Children have been brought to the out-patient room suffering from febrile symptoms for which no cause could be found until, as a matter of routine, the fauces were examined.

Some years ago a chlorotic girl, who had been for a few days in Guy's Hospital, died without anyone suspecting the real nature of her disease. The chief symptom, beside extreme pallor, was a weakness so extreme that she could not sit up even when supported. After death the fauces were found covered with false membrane. The weakness was no doubt diphtherial paralysis.—C. H. F.

Another obscure form of diphtheria is limited to the *nasal mucous membrane*. The chief local symptom is the escape of a thin blood-stained fluid from the nostrils, which become more or less reddened and excoriated. Epistaxis is not infrequent, and it may be so profuse as to account for the bloodless appearance of the patient, apart from the fact that anæmia is so constantly present in diphtheria. With a speculum we may sometimes see the turbinated bones covered with membrane, or casts may be discharged from the nostrils. After the first day or two the secretion from the affected parts is dark and very offensive. The disease not infrequently extends along the canal duct to the conjunctiva, which becomes coated with a perfect diphtherial membrane. Or it may pass through the Eustachian tube to the tympanum, causing singing or buzzing in the ears and deafness; perforation may then take place, and pus be discharged through the external meatus.

Another direction in which diphtheria sometimes spreads is from the pharynx down the *œsophagus*. In one fatal case at Guy's Hospital Dr Fagge found a number of small ulcers in the stomach close to the cardiac orifice, some of which were coated with a distinct layer of false membrane; and instances have been recorded in which the whole œsophageal and gastric mucous membranes have taken part in the morbid process. This condition seems to have given rise to no special symptoms.

Laryngeal and bronchial diphtheria.—The continuity of the false membrane is often traceable directly over the epiglottis and the arytæno-epiglottidean folds; but sometimes this is not the case, and possibly the spread of the disease to the larynx and trachea is the result of "auto-infection," an inoculation of the mucous membrane by particles of secretion drawn downwards with the inspired air. Upon the epiglottis and the vocal cords the false membrane is firmly adherent, and it may pass completely over the space between the true and the false cords. Below the glottis it is more loosely attached to the mucous surface. It becomes thinner as it descends, and in the trachea, at a variable distance down, it commonly ceases, and becomes continuous with a muco-purulent layer which bathes the rest of the air-passages. But in some cases even the bronchial tubes within the lungs present a well-formed tubular membrane.

It is seldom practicable to employ the laryngoscope to determine the presence of a false membrane in the air-passages in a case of diphtheria, and only by rare skill and fortune would a satisfactory view of the larynx be obtained in a child. The diagnosis of laryngeal diphtheria rests mainly upon the fact that the entrance of air into the lungs is impeded, and that fragments of membrane are coughed up: this, however, we do not see in young children, and often must wait until tracheotomy has been performed before the membrane is seen. This is shown not only by the rapidity and stridor of the respiration, but also by the way in which at each breath the soft parts above the clavicles and sternum are sucked in, as well as the lower intercostal spaces, and in young children even the sternum and ribs. Every time that the patient inspires a loud crowing or croupy noise may be audible, and the cough is often hard and brassy. In other words, the symptoms are as those of Croup. The dyspnœa is apt to become greatly aggravated from time to time, a circumstance probably due either to spasm or to impaction of membrane in the narrow chink of the glottis.

When diphtheria affects the larynx it does so commonly from three to six days after the onset of the disease. Jenner, in 1861, had never known it delayed beyond the end of the first week; but Oertel says that it is not infrequent on the eighth or tenth day, and may be as late as the thirteenth. In Jenner's cases when death occurred it was in five days from the setting in of laryngeal symptoms; and he says that out of twenty-six fatal cases of Bretonneau's there were only five in which life was prolonged after the third day, and but one after the sixth day, except as the result of tracheotomy.

The presence of false membrane in the air-passages is so dangerous to life, from mechanical interference with respiration, that laryngeal diphtheria is always a grave disorder, even when symptoms of muscular weakness, anæmia, and depression of the heart's action are less marked.

In certain cases diphtheria seems to begin in the air-passages, without any primary affection of the fauces. In the epidemic form of the disease, however, this is rare. Bretonneau met with but two instances; of one of these he gives details (Case 45 in his fourth 'Memoir'). The patient was an infant, a year old, in charge of a nurse at Tours, where no case of diphtheria had been seen for months; but in a hamlet some miles distant the disease was prevailing, and a nephew of hers had died of it a few days before the infant fell ill. Bretonneau put the frequency of primary laryngeal diphtheria at one in thirty cases; Guersant at one in twenty cases. In the epidemic which occurred at Auchtergaven (cf. p. 322), Dr Yeats observed, among one hundred and eighty-three cases, fifteen in which laryngeal symptoms were present from the first, but in which there was no visible affection of the fauces when they were first seen; and in six of these the pharynx remained free throughout the disease.

In 1877 Dr Fagge found among fifty cases of diphtheria at Guy's Hospital thirty-five in which the air-passages were affected. The present writer's experience is that in children extension from the fauces to the larynx is the rule, and that in adults it is the exception. Primary laryngeal diphtheria without membranous tonsillitis he has only seen in children.

Diphtheria of the skin.—This is a somewhat rare and very interesting form of the disease. It is usually secondary to the ordinary faucial lesion, but sometimes primary, as in the case of a little girl under the writer's care, with empyema (June, 1897), in whom, after operation, a false membrane appeared on the wound. The bacillus of Löffler was found in the pus, and the temperature rose, but the fauces were unaffected; there was no albuminuria, and no paralytic symptoms. The wound was treated locally and the child recovered well.

The writer has also seen a membrane appear after a blister in a case of ordinary diphtheria; and it seems probable that the skin is only affected when the protecting epidermis has been removed; so that this form may be called traumatic diphtheria.

Diphtheria sometimes appears on mucous membranes other than those of respiration: on the prepuce or vulva: but this, like diphtheria of the skin, appears to be always secondary to the disease in its ordinary seat. Mr Walter Pakes told the writer of this occurring in a child about two years old, suffering from faucial diphtheria, in whom was found swelling of the vulva with a glistening white membrane, and on staining and examining this membrane microscopically Löffler's bacillus was found.

Complications and sequelæ.—Albuminuria.—It is a question whether this should be regarded as a constant or a very frequent symptom. Its frequency was pointed out by Dr Wade, of Birmingham, in 1858, and is of practical importance as aiding in the diagnosis of obscure cases. Hyaline casts are often present, and, much more rarely, blood. Eberth met with albuminuria in two cases out of three; the present writer has not found it absent in one out of ten. In most patients it is observed within a day or two from the commencement of the disease; in a few not until convalescence. It is sometimes very transitory, and may be detected only once or twice, even in cases in which the urine is repeatedly examined; but it more often lasts for a week or two. It is generally supposed to be devoid of prognostic significance; Oertel, however, although he admits that the urine of some patients who die rapidly contains very little albumen, yet says that he has made out a close relation between the quantity excreted in twenty-four hours and the severity of the disease. From one to three drachms was the amount usually passed in cases so severe as to threaten life; and the albuminuria persisted, in those who recovered, for six or eight weeks after the subsidence of the diphtherial affection of the throat.

There are probably two kinds of albuminuria dependent on diphtheria as upon scarlatina. One is the ordinary febrile albuminuria, dependent on temporary "cloudy swelling" of the renal epithelium, and without sequelæ or grave significance. This is present in the great majority of cases, more often than in pneumonia, scarlatina, or any other specific fever except erysipelas. The other depends on acute nephritis, and is much less common than in scarlatina.

In some cases, particularly when blood and casts have been present, the kidneys are found after death to be large and congested, the epithelial cells swollen, opaque, granular, and filling the tubes. Minute extravasations of blood are also present in some cases; and, according to Oertel, masses of lymph-corpuscles often surround the Malpighian capsules. At Guy's Hospital the affection of the kidneys observed in cases of diphtheria is, as a rule, only slight, much less than scarlatinal nephritis; and this accords with the well-known fact that dropsy very rarely occurs after diphtheria. A few instances of general anasarca have, indeed, been recorded by different observers—one, for example, by Oertel in the 'Deutsches Archiv' for 1871. But when this writer speaks (in Ziemssen's 'Handbuch') of fifty fatal cases, mostly attended with suppression of urine and dropsy, as having occurred in Kiel and the neighbouring villages, the doubt arises whether the epidemic was not really one of scarlet fever with diphtherial complications. In the writer's experience, while albuminuria is nearly constant, it does not with rare exceptions last during convalescence, and once only has he seen it end in chronic albuminuria with dropsy.

Washbourn and Goodall describe cases of diphtheria in which the urine becomes very scanty and at last is suppressed, while extreme pallor and severe vomiting mark the progress to death.[*]

Paralysis.—Convalescence is sometimes attended by a remarkable sequela—*diphtherial paralysis.* This was observed by the Spanish physicians in the seventeenth century. It commonly begins during the second or third week after the subsidence of the throat affection, often earlier, and is said to be occasionally postponed until the lapse of a month or six weeks. Paralysis is rare after laryngeal diphtheria; this may be because when the larynx is affected death is often too early for sequelæ to develop, but also the absorption of toxines from the fauces is much more active than from the tracheal mucous membrane.

The soft palate is first affected; it hangs flaccid, the uvula cannot be drawn up, and its sensibility is lost. One consequence is that the patient speaks indistinctly, or "through the nose;" another, that when he attempts to swallow liquid, it returns through the nostrils. The pharyngeal muscles are occasionally involved: deglutition is then difficult, and some of the food is apt to find its way into the air-passages.

Next, generally after an interval of a few days, one or more of the ocular muscles may be attacked; the patient then sees double and squints: or paralysis of accommodation sets in, affecting both eyes, so that he is not able to distinguish near objects properly, and cannot read small print with comfort.

The limbs are affected somewhat later still, but sometimes they are the parts in which a loss of power is first observed. Sensations of numbness or pain in the feet are complained of; and presently the legs grow weak and tremble, the gait is shuffling and uncertain, or the patient may be unable to stand without support. The arms are much more rarely involved, but sometimes the patient cannot dress himself or hold anything in his fingers. He can neither bend the feet on the ankles, nor extend the hands on the wrists.

According to Oertel, the faradic contractility of the affected muscles is greatly impaired, and their substance undergoes rapid wasting. Duchenne, however, denied the loss of faradic contractility, and Buzzard agrees with him. The degeneration-reaction is certainly not constant, but it is present in almost all severe cases. Atrophy is usually moderate, but the muscles have a characteristic flabby feel. There is more or less anæsthesia, but no pain. The knee-jerk is often abolished, sometimes without other sign of paralysis.

The muscles of the larynx may be affected, in which case the vocal cords may be seen in the laryngoscopic mirror to lie motionless in a position midway between that of respiration and that of phonation—the position which they always occupy in the dead body, but which is never seen in health. Wilks regards this affection as a frequent cause of suffocative dyspnœa, when the tracheal tube is removed, after being worn for a few days. The inspired current of air draws the paralysed cords inwards, until they meet and close the glottis.

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[*] It was maintained by MM Bouchard and Labadie-Lagrave that *endocarditis* is of frequent occurrence in diphtheria. I have repeatedly searched for such an affection in making autopsies in children who had died of the disease; but the valves have always appeared to be perfectly healthy. And Sanné (who has had good opportunities of testing the value of the statements in question) declares that the slight irregularities that are commonly found under normal conditions on the upper margins of the mitral and tricuspid *valves have been* mistaken for vegetations.—C. H. F.

The muscles of the neck and of the trunk may be affected, so that the patient is unable to keep his head supported, or to raise his body from the recumbent position, or to turn over in bed. In some instances the bladder and rectum take part in the paralysis, and there is often loss of virility in adults. Lastly, the diaphragm or the intercostal muscles may be paralysed, and lead to death by suffocation, and the vagus nerve may be affected and cause death by syncope.

In March, 1888, a girl of fifteen was under the writer's care in Mary Ward. She went through an ordinary attack well, and after a week's convalescence was attacked by paralysis. It began as usual in the soft palate; also the velum never completely lost power of movement; then the pharynx was affected, and she became unable to swallow, so that for nearly a fortnight nutrition was carried on first by the rectum, and then by an œsophageal tube. The arms and legs were paralysed, but the ocular and facial muscles escaped. The muscles of the neck and trunk were so weak that she could not turn or move in bed or lift her head from the pillow. Happily the heart and lungs were unaffected, and, as usual, "no pelvic symptoms" were present. The reflex movements and knee-jerks were abolished, and there was considerable, though not complete anæsthesia of the paralysed parts. There was no tenderness on pressure over the paralysed muscles; no reaction of degeneration, but loss of faradic contractility. Recovery was very slow. First she regained power of swallowing; then the arms recovered; then the legs; and before she could walk the muscles of the back and neck had completely regained their power. Ultimately convalescence was established.

The pathology of this sequel of diphtheria is no longer doubtful.* The ordinary diphtherial paralysis is certainly peripheral in its seat, and toxic in its origin. The muscles are found atrophied, and the nerves in the condition of parenchymatous neuritis.

The frequent tenderness in the course of the affected nerves, the loss of knee-jerk, and anatomical observations by Charcot, Vulpian, and Lépine in France, and by Leyden and Mendel in Germany, have established the theory of diphtheritic paralysis depending on peripheral neuritis (cf. Dr Buzzard's 'Harveian Lectures' for 1885, p. 108).†

Syncope.—This serious and sometimes fatal complication is not limited to cases of severe diphtheria; it sometimes happens when all the symptoms have been slight. The first of the following instances is related by Jenner.

A boy aged ten, when convalescent from a very mild attack of diphtheria, began to vomit, and the pulse (which had been becoming less frequent for two days) fell to 36 in the minute. There was nothing in the patient's appearance to suggest that he was in imminent danger; but notwithstanding the free use of stimulants the pulse continued to fall. By the next afternoon its beats were only 24, and soon afterwards they ceased altogether.

A little girl, in whom there were all along well-marked diphtheritic patches in the fauces, seemed to have so little the matter with her that she was allowed to play with other children in the garden in front of the house. Early one morning she was being brought downstairs from the nursery as usual before being dressed, when it was noticed that she looked very pale. On being hastily sent for, I found her pulseless with her extremities perfectly cold, and in spite of all that could be done she died about eighteen hours afterwards, without having rallied in the least from her collapsed condition.—C. H. F.

A boy eight years old, under the writer's care, after going through an ordinary attack of diphtheria, showed symptoms of paraplegia, and, in spite of free exhibition of alcohol and strychnia, died of increasing cardiac weakness without dropsy. At the autopsy the heart, particularly the left ventricle, was found greatly dilated; but the muscle, though pale, was not degenerated.

In this last case the cause of the acute cardiac dilatation and syncope was probably "nervous" or "paralytic." In others there is distinct evidence of myocarditis or fatty degeneration.

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* In one case Oertel found extensive changes in the spinal cord, hæmorrhages surrounding the roots of the nerves, proliferation of nuclei in the grey matter, and exudation in the central canal. In another case Buhl observed hæmorrhages in the membranes and substance of the brain, and a red, swollen, softened state of the roots of the spinal nerves. See also Dr *Percy Kidd's paper in the 'Medico-Chirurgical* Transactions' for 1884.

† *Similar* [illegible] enteric, scarlatina, and smallpox.

Diagnosis.—The distinction between diphtheria and scarlatina is comparatively easy. The absence of the high fever, rapid pulse, and rash of the latter disease, with the presence of membrane on the fauces, is usually sufficient; but when diphtheria complicates scarlatina, or when a mild attack of diphtheria is without any visible membrane, only a skilled bacteriological investigation can decide the question.

Traumatic inflammation of the fauces may produce a membrane in the case of children. Cases have come before the writer which prove this. One was a child eating a hot potato; another a case of drinking from the spout of a kettle; another of poisoning with corrosive sublimate. In some such cases it is possible that the accident had nothing to do with the membrane, which was really the result of previous infection; but this will not apply to all. The course of the affection can alone decide the point clinically, and the presence or absence of the bacillus pathologically.

Tonsillitis—particularly some cases of follicular tonsillitis—is often difficult to distinguish from slight forms of diphtheria. The temperature is usually higher, the spots are more yellow, less grey, less raised, and do not coalesce; there is no albumen, and none of the prostration of diphtheria.

Thrush is of a more opaque white, is cleaner in appearance, and unattended with hæmorrhage, ulceration, or sloughing. A stained specimen shows the *Oidium albicans*, not the bacillus of Löffler.

Albuminuria as a symptom and paralysis as a sequel are each important helps in the diagnosis of diphtheria: but after all it must be confessed that the nature of some appearances of the throat can only be determined by consideration of the circumstances, by the presence of other cases in the house, and by the subsequent development of symptoms; while, on the other hand, some cases, where no membrane is discernible, are proved to be diphtherial, not only by the microscopic test and cultivation of the mucus of the tonsils, but also by the unanswerable fact of their producing fresh cases by infection of the disease.

The practical deduction to be made from the last fact is that no patient convalescent from diphtheria should be allowed to mingle with other children until the mucus from the fauces ceases to contain Löffler's bacillus, and this may be for a fortnight, a month, or even longer, after he is apparently perfectly well.

Prognosis.—No case of diphtheria, however mild, is free from peril. The average mortality has varied in different epidemics, but according to Oertel it generally used to range between 30 and 40 per cent.

Age is the most important element of prognosis. The fatality among children under two years old used to be terrible, and is still considerable. The larger the proportion of children among those who are attacked, the more fatal the disease; for in adults it comparatively seldom assumes the fatal laryngeal form. However, according to Trousseau, the nasal variety is almost equally dangerous.

It was observed by the Spanish physicians in the seventeenth century that "at its first coming it was most severe, but by degrees became less violent—as is usual with other epidemical disorders" (Fothergill, 1769).

In faucial diphtheria the chief dangers are inability to swallow, and *septicæmia*: in laryngeal diphtheria, asphyxia before tracheotomy, and *afterwards* bronchitis, often with lobular pneumonia and collapse, which more slowly but not less surely stifles the patient: in both, failure of the heart, either gradual or sudden.

Unfavourable symptoms are hæmorrhage, extreme pallor, vomiting, a low temperature, and a very frequent pulse. Apart from the danger of its attacking the heart or diaphragm, diphtherial paralysis is, as a rule, recovered from even in severe cases.

Hæmorrhage is very rarely fatal; it may be from a branch of the external carotid, or possibly from the trunk of the internal carotid artery.

In 86 consecutive cases of diphtheria under the writer's care (ending December, 1894) the mortality was 19 in 23 under three years old, 21 in 40 between three and ten, 3 in 10 between ten and seventeen, and only 2 in 12 adults between twenty and fifty. Of purely faucial cases only 5 out of 26 were fatal, of faucial and laryngeal 24 out of 33, and of purely laryngeal all. In 51 cases tracheotomy was performed, with 34 deaths. (Compare these figures with the result of the antitoxic treatment since introduced.)

Treatment.—A patient with undoubted or with doubtful diphtheria should at once be separated from other children, and given an injection of the antitoxin to be presently described.

As soon as the heart begins to flag, brandy or port wine should be prescribed freely, and nourishing soups should be given in small quantities at short intervals night and day. Jenner mentions the case of a child, three years old, who took from three to five ounces of brandy in twenty-four hours with apparent advantage. When swallowing is refused or impossible, food must be introduced into the stomach by a soft flexible nasal tube, or the patient must be fed by rectal suppositories. The tincture of perchloride of iron is most usually given as a medicine, while by others quinine, or bark with ammonia, is preferred.*

As to the *local treatment* of the throat affection, there used to be uncertainty of opinion, but observers are now agreed that the membrane on the tonsils and palate should never be forcibly removed.

When the fact became recognised that the local affection within reach is not the really dangerous part of the disease, disinfectants were employed. Diluted chlorine water was found to be the most efficient agent by Oertel, who added various disinfectants to liquids in which diphtherial membranes had been repeatedly washed, and afterwards tested their powers of setting up a putrefactive process in "Pasteur's fluid." Other useful applications are alcohol, solution of permanganate of potash (gr. iss—gr. iiss ad ℥j), corrosive sublimate (1 in 4000), and solution of carbolic acid (gr. iiss ad ℥j). These antiseptics may be used as a gargle or spray for adults,† but with children the thorough and frequent use of them is almost impossible. Sucking small pieces of ice often gives great relief and helps deglutition.

The local application of the weaker or of the stronger solution of perchloride of iron was at one time freely used; also lime water as a gargle, first suggested by Küchenmeister, on account of its remarkable power of dissolving diphtheritic membranes. The present writer has used neurin (the alkaloid of lecithin) locally in diphtheria of the fauces, following some trials made at Vienna;* it clears away the membrane without pain or further injury, and leaves a clear surface. Another preparation introduced from the physiological laboratory is papain, from the Brazilian papaw-fruit. This powerful digestive agent dissolves false membrane with great readiness, and we have used it at Guy's Hospital, to that extent with success. But it is doubtful whether the result is worth the distress caused by any local application. The writer, having gone through an attack of faucial diphtheria himself, thinks that it is not.

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* Dr Hermann Weber has drawn attention to the fact that the practice, universal in England, of giving abundance of nourishment in diphtheria, is far from having obviated the liability to the occurrence of the sudden collapse which we have seen to be one of the principal modes in which the disease proves fatal; but whatever may be said with regard to this complication, I do not think there can be any doubt that the general tendency of such treatment must be good.—C. H. F.

† A gargle which is both useful and harmless in the case of adults and older children is the *Lotio boracis alkalina* of the Guy's pharmacopœia, made by dissolving three grains and a half of sodium bicarbonate, borax, and common salt with seven of powdered sugar *is an ounce of warm water. Siegel's spray* apparatus is a convenient way of applying

In the case of children, syringing the fauces with equal parts of lime water and milk, or with some antiseptic solution, is probably the most efficient and least injurious procedure. Experience shows that there is no ground for the fear expressed by the elder Heberden that syringing the fauces would poison the patient by introducing putrid matter into the stomach; for the danger is not in digestion, but in absorption of this matter unchanged. That admirable author concludes the chapter in his 'Commentaries' which deals with the malignant sore throat (under which he probably included some cases of scarlatinal as well as of diphtheritic angina) as follows:—"The gargle may be injected with a syringe into the throats of children, but this should by no means be done so often as to tease or fatigue them. Similar reasons would forbid us still more strongly to take great pains in rubbing off the sloughs from these ulcers or in scarifying them. . . . . My only reason for suspecting that I ought to lay more stress upon applications made immediately to the throat than I have here done, is that several physicians of deservedly great authority have judged them to be of more importance than they have appeared to me."†

When the disease has its seat in the *nasal cavities*, these parts should be frequently cleansed with disinfectants (dilute solutions of the permanganate of potash or carbolic acid, or lime water), which may be injected with a syringe every two hours, or even oftener.

For diphtheria affecting the *skin*, the local application of calomel was recommended by Trousseau; we now use iodoform. On account of the liability to development of false membrane wherever blisters are applied, they should never be ordered in cases of diphtheria.

When diphtheria attacks the *larynx*, an emetic of ipecacuanha or of sulphate of zinc should be given, and if a good result is obtained it may be repeated after two or three hours.

But in most cases of laryngeal diphtheria the operation of *tracheotomy* is necessary. The indications for tracheotomy are rapid respiration with laryngeal stridor, and with deficient breath-sounds at the base of the lungs; pallor combined with a purplish tinge of the lips; and sucking in with each inspiration of the soft parts at the root of neck and at the epigastrium, as well as of the lower ribs and ensiform cartilage. It is better not to delay the operation after vomiting has failed to relieve.

After tracheotomy it is probably best to abstain from all treatment except cleansing the tube and removing membrane and muco-pus from the trachea with a soft feather. In cold weather with dry air the child's cot or the patient's bed should be kept moist with steam from a kettle, but not when the patient is sweating and exhausted.

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* "Ueber die Wirkung des Neurins bei Diphtheritis," von Prof. E. Ludwig in Wien ('Centralbl. f. d. med. Wissensch.,' 1877, No. 12).

† 'Commentaries,' chap. vii. Compare the excellent remarks by Senator on the useless and mischievous attempt to apply local remedies to the fauces of young children in an efficient manner ('German Clinical Lectures,' 2nd series, p. 447).

Children under a year old used generally to die; the small calibre of the trachea, the yielding nature of the chest walls, and the difficulty of feeding them are the causes of this high mortality. Even when the air-passages are freed from the obstruction which threatens the patient's life, there is always the fear that the disease may, as it often does, spread downwards and obstruct the bronchial tubes. We have tried intubation instead of opening the trachea, but so far the results have not been encouraging. When a child refuses to swallow, liquid food must be poured down one nostril while the other is closed,—not through a catheter, but by a funnel inserted into the orifice. Nutrient enemata are rarely successful for more than a short time with children: suppositories sometimes succeed better. Brandy in frequent doses is of great service.

Serum treatment.—For the last few years we have all been treating diphtheria by means of the antitoxic serum, and the writer agrees with Dr Church and other physicians at the great London hospitals, and with those who have direction of the still more extensive field afforded by the sick asylums, that in this method we have by far the most effectual means of treating diphtheria. The credit of conceiving and carrying it out is due to Behring of Berlin. The animal inoculated with the pure cultivation of Löffler's bacillus is the horse. When by successive injections of varying strength he is rendered immune to the poison, blood is drawn from his jugular vein, and after coagulation the serum is sterilised and kept in stoppered bottles until needed. A large manufactory is at work under Government inspection near Frankfort, another in Paris, and the serum is now provided by the conjoint laboratories of the Royal Colleges and by the Jenner Institute of Preventive Medicine, as well as by private manufacturers.

Of the good effects of this treatment there is no reasonable doubt. Baginsky in Germany, Welch in America, Washbourn and Goodall in England, are only some of those who have published numerous and well-recorded cases. The present writer has seen it entirely alter the prognosis of diphtheria in children under five, and rescue older children and one grown man in whom the disease showed every sign of fatal ending. The remedy should be tried, however late in the disease one may be summoned, but the best results are where it is used at the beginning.

The chief drawback to the serum treatment of diphtheria is the large amount needful to inject under the skin: 20 c.c., though trifling in an adult, is a large subcutaneous injection in an infant, particularly when its repetition is necessary. The efforts, however, of Behring himself, of Roux, and in this country of Dr Klein, Dr Bullock, Dr Woodhead, and Dr Cartwright Wood, have succeeded in intensifying the activity of the serum, so that 10 c.c. or 5 c.c. contain the requisite number of "units," and no ill effects are found to attend the use of the smaller and stronger dose.

The only unpleasant consequence, and that an occasional one, is a roseolous rash. The writer has only seen six or seven examples of this, but others have observed it more frequently, and reported its occasional combination with slight pain, moderate fever, and swelling of the joints. It is probable that this erythema is not caused by the same constituent of the serum which acts on the toxines, and Dr Brodie, aided by a grant from the Grocers' Company, has been trying to separate the constituents which produce the rash.*

Treatment of complications.—Local diphtherial inflammation, whether of the nostrils, the conjunctiva, or the genitals, or of an accidental injury, must be treated by the sedulous application of the stronger antiseptic solutions above mentioned. After death from diphtheria, the lungs are found in young children collapsed in several lobules; and in all cases the tubes are filled with pus and mucus. Ammonia and senega, sweetened with treacle or syrup of tolu, is the best medicine, and brandy the best form of stimulant to prevent this condition.

Another danger is *failure of the heart.* Steel and small doses of digitalis or strophanthus are probably the best means of guarding against this catastrophe. Feebleness of the pulse, and the first sound of the heart resembling the second, are indications of the danger; when they appear, alcohol and strong meat extracts should be given, and, if necessary, strychnine injected under the skin.

Treatment of sequelæ.—Diphtheritic paralysis is the most important of these. It generally subsides spontaneously within three or four months, and sometimes much earlier. A house physician of the writer, who suffered from partial paraplegia after diphtheria, was nearly a year before he entirely regained his powers of walking. This paralysis is rarely fatal; but sometimes the patient is left permanently with slightly impaired power of certain muscles. For instance, some years ago a boy came to Guy's Hospital as an out-patient who had been in our wards four years previously; he was still unable to swallow perfectly, and fluids sometimes returned through his nose if he tried to drink rapidly.

Iron is indicated in cases of this kind, and often proves very useful. But the most useful drug seems to be strychnine, particularly used subcutaneously; and slowly interrupted galvanism is also of considerable service.

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* G. E. C. Wood, 'Proc. Roy. Soc.,' Feb. 20th, 1896.

SYPHILIS

> "Consumptions sow
> In hollow bones of man, strike their sharp shins,
> And mar men's spurring. Crack the lawyer's voice,
> That he may never more false title plead,
> Nor sound his quillets shrilly: hoar the flamen,
> That scolds against the quality of flesh,
> And not believes himself: down with the nose,
> Down with it flat, take the bridge quite away
> Of him that his particular to foresee,
> Smells from the general weal: make curled-pate ruffians bald."
>
> *Timon of Athens.*

History of the disease—Its present extent—Its nomenclature—Its place among specific exanthems—Origin and incubation—Primary lesion—Infective and soft sores—Premonitory stage—Secondary lesions of skin, throat and mouth, eyes, &c.—Tertiary lesions of skin, tongue, palate, bones, testes and other viscera—Diagnosis—Conditions of infection—Special questions in syphilitic pathology—Prognosis—Protection—Treatment.

Congenital syphilis—Its transmission to the child—Syphilitic placenta—Local manifestations—Its further transmission—Its treatment—Later effects.

Synonyms.—Lues venerea—Lues—Morbus gallicus—The pocks or great pox.—*Fr.* La grosse vérole, Mal de Naples.—*Germ.* Lustseuche.

Definition.—A specific contagious disease running a protracted course, with many local forms and numerous sequelæ.

History.—The disease which was first recognised three hundred years ago and received the name of Syphilis, was then regarded as a pestilence, an epidemic disorder—spreading like the plague, or putrid fevers, by "ill conditions of the air," and conveyed by infection at a distance. It was only gradually discovered to be closely connected with foul local disorders of the genitals, and to be as a rule communicated by sexual congress. In the seventeenth and eighteenth centuries "constitutional" syphilis was known as the venereal disease (*lues venerea*), and was supposed to spring from any form of local disease acquired in impure connection. Its constitutional effects were until lately compared with those of Gout and of Tubercle, and thus a syphilitic "diathesis" was placed side by side with assumed "arthritic," "scrofulous," and "malignant" diatheses. Inherited syphilis was unrecognised after infancy, or was confounded with what was called "scrofula;" and "scrofula" itself was often supposed to be the expression of a distant syphilitic taint. Owing to the labours of Ricord, Bassereau, Lancereaux, Hutchinson, Wilks, and many other observers, the accidental connection of this disease with others of venereal origin no longer obscures its distinctive characters; it is completely separated from tuberculosis in both its acquired and its hereditary form, and it now resumes its original historical place among specific contagious diseases, not always venereal in origin, and not more different from measles, or smallpox, than are diphtheria or cholera.

There is no clear account of syphilis in the writings of antiquity, or in those of the Middle Ages. The disease was first recognised by its epidemic prevalence in Italy at the end of the fifteenth century, when Charles VIII of France was besieging Naples. It was then supposed to be a new malady; either generated by the filth of a camp and the strange wickedness of mercenary troops, or introduced from the newly discovered Western World by the sailors of Columbus, who arrived in Europe about the time when it broke out. However, there are grounds for the belief that the disease had been observed in France, in Germany, and in Italy, as well as in Spain, before the French invasion of Naples (1494-5), and even before Colombus had reached the port of Palos (March, 1493), on his return from his first voyage to the West Indies. Critical inquiries seem to show that in all probability syphilitic affections were by no means unknown during previous centuries, although they were confounded with other maladies, particularly with leprosy. Moreover some of the allusions of Martial must almost certainly be referred to secondary syphilis.

The Italian epidemic of 1494 and the subsequent years was severe and wide-spread. Lancereaux supposes that almost a twentieth part of the population were attacked, and although few died, fewer still were cured. The disease soon lost its epidemic pestilential character, and by the middle of the sixteenth century its type resembled that with which we are now familiar. The passage from 'Timon of Athens' (iv, 3), which stands at the head of this chapter, shows how familiar the symptoms of secondary syphilis had become by the beginning of the seventeenth century. As above stated, its diffusion was at first ascribed to climatic or telluric influences, in ignorance of its real mode of conveyance.

More limited epidemics of syphilis have since been observed, which have sometimes been misunderstood, and described under various special names; and some affections, long regarded as peculiar to certain regions, are now recognised as syphilitic. A disease which attacked one hundred and eighty persons at Brünn, in Moravia, in 1578, one which raged in Canada in 1780, and the "Scherlievo" of Fiume at the beginning of this century, were almost certainly local epidemics of syphilis; while endemic syphilis was called "the Sibbens" in the west of Scotland, and "Radesyge" in Norway.

Syphilis is now found in every part of the world, but more or less widespread according as the conditions are favourable or otherwise for its usual mode of propagation. Among the inhabitants of Iceland it is said never to have established itself, although it has repeatedly been introduced by sailors; according to Livingstone, among the negroes of Central Africa it is mild and transient in its effects; and in China and Japan it would appear to be seldom severe. Possibly in China its virulence has gradually become attenuated by its wide diffusion through the closely packed population in successive generations. Lancereaux states that lues acquires unusual intensity when it passes from a Chinese to a European. Similar assertions have been made with regard to its transmission from one race to another elsewhere,—in the case, for instance, of the British army in Portugal during the Peninsular War.* The worst forms of syphilis appear in seaport towns, where vice and intemperance prevail together.

Names.—The term "Syphilis" was invented by Fracastorius, a learned physician of Verona, who in 1521 published a poem under that title, in which he related how *Syphilus*, a shepherd, was stricken by Apollo with the new disease—even then not recognised as venereal. It is a great pity that we have not for every specific disorder a name as distinguishing, as short and flexible, and as free from meaning.

The *locus classicus* is as follows:

> ". . . . primus
> *Syphilus* ostendit turpes per corpus achores:
> Insomnes primus noctes convulsaque membra
> Sensit, et a primo traxit cognomina morbus
> *Syphilidem* que ab eo tabem dixere coloni."
> (Hieron. Fracastorii, 'Syphilis sive de Morbo Gallico,' lib. iii, v, 329.)

The French contrasted *la vérole* with *la petite vérole* (i. e. *variola*), just as in England the word "smallpox" formerly conveyed a similar distinction.† Many other names, employed in various countries, indicated a belief that the disease was of foreign origin. Thus, while it was *mal de Naples* to the French, it was to the Italians *mal francese*;‡ in Egypt and Turkey it is called *el Frangy*, the disease of the Franks or Europeans; and, unhappily, the Sandwich Islanders first knew it as the "English disease." At one time the most common designation was *lues venerea*, which dates back to Fernelius (1556). But the venereal disorder was held to include both gonorrhœa, proved to be distinct by Ricord in 1831, and the soft chancre, which nearly all pathologists now regard as an independent affection. Indeed, the conception of syphilis as a general malady, comparable with the exanthemata, had no existence from the time of Fracastorius until about forty years ago. It is true that John Hunter described a constitutional form of the venereal disease, but he expressly taught that the action of the poison was different from that which occurred in any kind of fever. Since the modern view has been accepted, the name of *Syphilis* has superseded all others, but *Lues* and its adjective "luetic" are still used abroad, and are sometimes convenient from their decent obscurity.

Pathology.—Like other results of purely scientific and speculative inquiry, the recognition of the true nature of the disease has proved of the utmost practical importance. The physician must almost forget the local primary disease, and put aside the idea that the diagnosis of syphilis carries with it the stigma of impurity. There are many ways in which a person may fall a victim to syphilis without illicit intercourse; and in exceptional instances one must be prepared to recognise its manifold varieties in patients of either sex, at any age, and in every position of life.

In the second edition of this book the writer accordingly included Syphilis among the specific febrile diseases; for it arises by contagion alone, and breeds true; inoculation is followed by a latent period of incubation; the onset is febrile, with an exanthem and a local lesion in the throat; lastly, it has definite sequelæ, and it protects against a second attack.

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* On this point, however, see Dr Geo. Ogilvie's able and interesting criticism of the statements made by the army surgeons of the time (1806–12).—'Brit. Journ. of Dermat.,' 1898, vol. x, p. 232.

† My learned friend Dr Norman Moore informs me that the term "pocks" frequently occurs in Irish and other mediæval MSS., and probably refers to cutaneous syphilis.

‡ It was called the French disease also in England:

> "News have I that my Nell is dead i' the spital
> Of malady of France."—*K. Henry V*, v, 1.

The contagium has not yet been certainly identified, although Lustgarten long ago described a bacillus, resembling those of tubercle, lupus, and leprosy, in the cells of the diseased tissues.*

The chief peculiarities of syphilis are the slightness of its pyrexia, the remarkably slow evolution of its stages, the importance and frequency of its sequelæ, its hereditary transmission, and, we may add, its reaction to remedies. As regards its being hereditary, Mr Hutchinson argues that smallpox and even typhus are, like syphilis, occasionally transmitted from mother to fœtus, but the brevity and severity of these and other specific fevers make it almost impossible for them to be inherited.

Course of the disease.—Incubation.—After inoculation with the syphilitic virus there elapses a considerable time before any symptom is observed. This is the period of incubation. It is remarkable that this fact was unknown until, between 1856 and 1862, certain experimenters inoculated syphilis upon healthy individuals.† Professor Bäumler, in 'Ziemssen's Handbuch,' brings together thirty-one observations of this kind, in most of which the incubation was from fifteen to twenty-five days. Once it was only ten days; four times between thirty-five and forty-four days. In 1865 Fournier recorded a series of cases in which no treatment was adopted, so that the disorder developed itself naturally. The incubation was more often over than under three weeks; it not infrequently reached a month or six weeks, and once was prolonged to ten weeks. In one patient of Bäumler's, in whom the exact date of exposure to the poison was known, the incubation was twenty-five days; in another twenty-nine days.

Primary stage.—The earliest symptom of syphilis is manifested at the seat of infection; it is spoken of as the *primary* lesion, while the later symptoms are termed *secondary*. It is often called a hard or Hunterian "chancre," but upon the skin it is first seen as a small flat red papule. This appears three or four weeks after infection, in the prodromal period, according to the usual reckoning, although really the appearance of the papule and its induration at the seat of inoculation must be regarded as the first symptom of the constitutional infection. As it grows larger it becomes indurated, so that to the touch it feels like a piece of cartilage let into the part. After a week or ten days it may desquamate slightly; or a little moisture may ooze from its surface, and presently dry up into a thin scab; or it may continue to look shining and glazed; or, lastly, it may become excoriated, and slightly depressed in its centre. Upon a mucous membrane, the primary affection seems to begin as a very small itching vesicle with a reddened base, which soon breaks, forming an erosion, and afterwards a shallow ulcer; this, too, acquires an indurated floor as it enlarges.

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* 'Med. Jahrb. der k. k. Ges. der Aerzte,' 1885; 'Brit. Med. Journ.,' Oct. 17th, 1885, p. 757.

† Clinically incubation is as a rule prolonged to six weeks or more. The idea that no such period occurred was based partly upon a natural tendency to ascribe the disease to the last impure intercourse preceding its appearance, partly upon the circumstance that another virus (that of the soft chancre), which produces an effect almost at once, is often transmitted in association with that of syphilis proper, and formerly was not recognised as distinct.—C. H. F.

The histology of these lesions has been investigated by several observers. Biesiadecki found an abundant infiltration of nucleated cells, not only between the bundles of connective tissue in the cutis or mucous membrane, but also in the adventitia of the blood-vessels, which are thereby narrowed. He states that there is an actual development of connective-tissue fibres towards the periphery of the indurated mass, and that this is the cause of its hardness, which Auspitz had attributed to the presence of amorphous exudation between the cells and the spaces in which they lie.

The course of the primary lesion of syphilis varies in different cases. Sometimes it passes quickly away, leaving no trace of its presence; but when the sore is large it may take months in subsiding, and its site remains marked by a brown pigmented patch, with more or less superficial scarring in its centre. On the other hand, mucous membranes never show pigmentation. Mr Hutchinson has drawn attention to the fact that in some patients a fresh induration appears again and again during a period of several years exactly where a former primary syphilitic lesion was situated, without any fresh infection. Ultimately even the pigment disappears, so that no evidence remains that the patient has ever suffered from primary syphilis.

Hard and soft sores.—The first syphilitic lesion is called a Hunterian or indurated chancre; but if (as is often the case) it has the characters of a chancre, *i. e.* of ulcer, or "sore," they are accidental rather than essential. Bäumler calls the primary lesion of syphilis, not a chancre, but an "ulcerating sclerosis."

In 1852 Bassereau took the pains to trace to their origin in the opposite sex a number of venereal cases; and this method of "confrontation" (as he terms it) showed that, whereas sores which were followed by secondary symptoms had been derived from persons who themselves suffered under similar effects, other sores which remained simply local, or which at most were attended with suppurating buboes, came from individuals in whom the disease had likewise failed to produce any constitutional effects. His views were soon afterwards adopted by Ricord.

The one lesion was thus distinguished as the "indurated," "Hunterian," or "infecting" chancre, and the other as the "soft" or "non-infecting" chancre. The former cannot be inoculated upon the patient, nor upon any one else who has already had syphilis; the latter is readily reproduced by contact with other parts of the patient's body, or with other persons, whether syphilitic or not. The former is a specific infective lesion of syphilis, the other is a local contagious suppuration and non-syphilitic.*

The latter begins, without any incubation, in a pustule, which in two or three days breaks, and forms a deep, punched-out ulcer, with irregular and slightly undermined edges, a grey surface, and a soft base; it secretes pus freely, and is inoculable again and again. Hence there is seldom more than one indurated chancre, and often several soft chancres together. A soft chancre is scarcely ever seen except on the genital organs, and the cicatrices it leaves are puckered and more conspicuous than the macule which follows a Hunterian sore.

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* Bidenkap has, indeed, since found that there are occasional exceptions, it being ometimes "auto-inoculable" during an early stage of the disease, when there have as yet seen no constitutional symptoms. But such observations are strictly parallel with the fact hat vaccination can be successfully performed before the eruption of smallpox appears, *i. e.* efore the infection has become generalised throughout the body.

There is no doubt of the truth of Bassereau's observations, nor of the validity of Ricord's distinctions between two kinds of venereal sore.* But subsequent experience has shown that the practical application of those distinctions in diagnosis is liable to certain sources of error, particularly to the possibility of a mixed infection. The theoretical question still remains under discussion, whether the soft chancre is or is not in its origin independent of syphilis; but in a medical treatise the only lesion we have to deal with is the true syphilitic chancre.

In practice, although "hard" sores are almost sure to be followed by secondary symptoms, it is not safe to assert that any sore, however "soft," will *not* be so followed.

Symptoms of syphilis are sometimes seen without any apparent primary affection. One source of fallacy in these cases is that a primary lesion sometimes assumes the appearance of a secondary one. Thus a parchment-like glazed papule on the glans penis may be undistinguishable amid a general papular eruption, while on the labium the affection may be so modified as to simulate a "broad condyloma" or "mucous tubercle." In other instances, perhaps, it is so inconspicuous that the patient never notices its presence, and it quickly subsides, leaving no mark. Or the chancre may be in the urethra. Very few still admit cases of true "*syphilis d'emblée*," *i. e.* the invasion of the malady without a local lesion.

Prodromal period.—This latent stage of syphilis, after the appearance of an infective chancre and before the exanthem comes out, is marked not only by the induration of the primary lesion, but also by swelling and induration of the corresponding *lymph-glands*. So constant is this symptom that Fournier failed to detect it in only three out of 265 cases in men, and in three out of 223 cases in women. The glands which correspond with the usual seat of the chancre are those in the groins above Poupart's ligament. An indurated sore upon the finger leads to enlargement of glands in the axilla, and of the small lymph-gland which lies just above the internal condyle. One upon the lip affects those under the jaw. These "amygdaloid" or "bullet" glands are the size of almonds, or a little larger, and never reach the dimensions of the suppurating bubo which accompanies a soft chancre. They feel hard, are freely moveable, and are painless. There is no reddening of the skin over them, and they scarcely ever suppurate.

This "indolent bubo" of syphilis, as it used to be termed, appears a few days later than the primary lesion. A chain of glands is commonly affected, and often those in both groins at the same time. The swelling runs an exceedingly slow course for six months or more, and hence is most useful in diagnosis.† The spleen sometimes partakes in this swelling.

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* Much confusion at first prevailed as to the point at issue from the unfortunate nomenclature adopted by Ricord and his followers. Instead of speaking of the "duality" or "unity" of the chancre, they insisted on the *duality of the syphilitic virus*. This, however, is scarcely more than a verbal difficulty. The question which divided the partisans of the unity or duality of syphilis was, as Mr Hutchinson justly remarked, wrongly put. Syphilis is and can only be one and the same disease. What is not syphilis may be psoriasis or a soft chancre or smallpox. Syphilis is not always "venereal" in origin, and there are many other maladies of more or less frequent venereal origin, not only soft sores and gonorrhœa virulenta, but phthiriasis pubis and scabies.

† Syphilis is not the only cause of enlarged inguinal glands. Mr Cooper Forster insisted on the frequency of the occurrence in rowing men; and although the firm, painless, separate bullet-glands are usually characteristic, one may occasionally feel something very similar in cases of irritative enlargement, and in Hodgkin's disease.—C. H. F.

During the rest of the prodromal period we may suppose that the virus is undergoing multiplication in the primary induration and the adjacent glands. The interval between the appearance of the primary sore and that of the exanthem is generally *six or seven weeks;* but it is liable to wide variations. When the disease has been inoculated experimentally it has been found to range from twelve days to twenty weeks. During its course most patients look and feel well.

Secondary stage.—The general or *constitutional* symptoms of syphilis begin differently in different cases as well as at different times. In some there is well-marked fever, the temperature rising suddenly or gradually, until within a few days it may reach 104°. With the appearance of an eruption it sometimes declines, but it may run on for several weeks, assuming, as Bäumler has shown, an intermittent type resembling that due to malaria. The proportion of cases attended with febrile disturbance is stated by Güntz at 20 per cent.; but Bäumler thinks that it is really higher.

A more frequently noticed and characteristic symptom of this period is what French writers term *bitemporal neuralgia,* the occurrence of pains which shoot upwards along each temple. They are not usually felt during the day, but come on, often with surprising regularity, in the evening or at night.[*]

Pains in the back and limbs may be present, and occasionally swelling of the joints. Dr Fagge once saw a distinct, though slight and painless, enlargement of some of the articulations of the fingers; and Bäumler speaks of patients seeking advice for pain and swelling of the thumb in the early stage of the disease.

Syphilis of the skin.—Like other specific poisons, the virus of syphilis produces an exanthem, but this is followed by developments far more varied and more difficult to describe than those of measles or variola. They are known as *syphilides,* or collectively as syphiloderma (more properly syphilodermia).[†]

They are entirely distinct in pathology, in prognosis, and in treatment from the non-syphilitic eruptions which may simulate them. Since a name given should indicate a diagnosis, the terms psoriasis and roseola become useless if liable to be contradicted by the far more important qualification "syphilitic." A non-syphilitic disease, as psoriasis, is not more "modified" by syphilis than is scabies or a typhus rash; any of the three may be seen as a coincidence accompanying cutaneous syphilis, but unaffected by it.

The following are the common characters which belong to all the syphilides. (1) A peculiar *colour,* said to resemble raw ham, or to be "coppery."

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[*] Ricord used to ascribe it to the warmth of the bed, and said that in persons whose occupations compelled them to sleep during the day, the time at which the pains returned was reversed. But, whether or not this is the fact, there can be no doubt that his explanation of it is incorrect. For the hour at which the bitemporal neuralgia sets in is often early in the evening, while the patient is still up; and Bäumler is probably right in thinking that it coincides with an increase of fever, and is in some way dependent upon augmented vascular excitement.—C. H. F.

[†] With regard to the causes which lead to the evolution of one of these eruptions rather than another, all that we at present know is that a patient who is in a bad state of health is most likely to have those forms which suppurate and ulcerate. The differences between them are by no means attributable to tendencies towards particular non-syphilitic cutaneous diseases; for instance, a person liable to ordinary psoriasis is not specially apt to be affected with a squamous syphilide, nor one who has lichen with a papular one. We should, therefore, avoid using such names as "syphilitic lichen," "syphilitic ecthyma," or "syphilitic pso[illegible]."

According to Bäumler, the former comparison has been traced to Fallopius, who wrote about three hundred years ago; the latter only to Swediaur, early in the present century. The tint is but slightly marked at first; and a similar colour may be seen in non-specific eruptions, of chronic course, especially in the legs. What we can say is only that syphilides cause pigmentation more quickly than any other hyperæmias of the skin, and that the tint is less purple and more orange. (2) *Multiformity* or "polymorphism" in the same patient and at the same time. Sometimes macules, papules, pustules, scaly patches, are so intermingled that we cannot say which of them preponderates. (3) *Absence of itching* or pain. The indolence of the primary chancre and the secondary adenopathy above described, extends to all the syphilides, even the latest. (4) They often form *circles* or semicircles, "horseshoes," or less completely annular forms. (5) *Asymmetry.* The earliest rash, or any universal syphilitic eruption, may be symmetrical, just as the exanthem of measles or smallpox is symmetrical, because it affects the symmetrical human body. But syphilides do not show the characteristic symmetry of psoriasis, eczema, and some other cutaneous diseases, in picking out corresponding parts of the trunk or limbs. They are irregular in distribution. Characteristic localities are the forehead, the soles of the feet, and the palms of the hands, and they often affect regions rarely visited by other cutaneous affections.

A distinction has been drawn between the earlier and more superficial syphilides and those which are later in appearance, and affect the deepest layers of the skin. The former, to which some writers limit the term *secondary*, may appear consecutively to the early roseolous rash, or more usually, after an interval of a few weeks or months, but secondary eruptions are very seldom postponed beyond twelve months from the time of infection. The latter, which are often called *tertiary*, appear after the first year, and may break out for the first time after many years. This distinction of time is not, however, constant. "Tertiary" lesions are occasionally seen within the first year, and secondary syphilides sometimes appear when they relapse, it may be, at any period, but not after tertiary forms have developed.

Pathologically, the roseolous and other secondary eruptions may be compared to the exanthems of variola and typhus, while the tertiary syphilides have a less complete counterpart in the suppurations which occur as sequelæ of measles and enteric fever, or perhaps they may be more justly compared to the secondary growths which follow a malignant tumour.

Apart from the period of their development, secondary syphilides are often imperfectly symmetrical; tertiary almost always avoid even an approach to bilateral symmetry. The secondary rashes commonly consist of numerous isolated spots or patches. They seldom affect the hands or feet, are comparatively superficial, have little tendency to ulcerate, and leave no cicatrices behind them. On the other hand, the tertiary dermatoses of syphilis consist of comparatively few separate lesions, but they generally run together; they affect the deeper layers of the integument; they destroy the tissues, and are followed by scars. These distinctions do not apply equally to every form of syphilide belonging to the early or to the late group, but the exceptions are few.

The special character of the several syphilides will be most usefully discussed along with other diseases of the skin from which it is of the utmost importance to distinguish them; but the following brief account has respect to them as parts of the pathological evolution of the general specific disease.

1. *The macular or exanthemic syphilide* ("syphilitic roseola") is the earliest and most constant of all, and answers to the exanthem of measles and scarlatina, or perhaps more closely still to the occasional early rose-rash of smallpox. It consists of rather ill-defined, pale or dark, rose or "copper"-coloured spots, irregular in form, as large as a threepenny piece, or smaller; scarcely if at all raised above the surface, and disappearing under pressure. It is most constantly seen upon the chest and abdomen, but sometimes it covers most of the trunk, and appears on the neck and face; on the limbs it is less common, and it avoids the hands and feet. A pale and scanty eruption often fades within a fortnight, while one which is dark-coloured and abundant may remain visible for several weeks. It may then assume a papular form or disappear with slight desquamation.

2. *The papular syphilide* ("lenticular syphilitic lichen") is the most common form to follow the initial rash. It consists of red shining elevations, often of a marked coppery tinge. In size they vary from that of millet seeds to that of peas, and generally come out in successive crops.

The eruption is sometimes scattered irregularly over the whole body, sometimes the papules are grouped together in clusters. They are often numerous on the neck and forehead; sometimes in the naso-labial grooves, or at the angles of the mouth. They generally remain for some weeks, and not infrequently pass into a squamous form, or suppurate and form yellow or brown crusts.

In consequence of the thickness of the cuticle, the palms and soles show, not raised papules, but flat round horny plates, each with a reddish-brown border. After a time the surface becomes rough and scaly, and fissures may be formed. Thus a complicated affection arises, which used to be called "syphilitic palmar and plantar psoriasis." This form is the most symmetrical of all the syphilides.

3. *Mucous patches.*—Another syphilide is found on moist parts of the skin in contact with a neighbouring surface, and also on mucous membranes. The lesion is known as a "mucous" patch (*plaque muqueuse*), "flat, broad, or moist—*Condyloma latum*," in distinction from the "pointed condyloma" (*C. acuminatum*) or "wart" of the genital organs, which is often non-syphilitic in origin.

Mucous patches are raised, with a sharply defined edge, and a surface sometimes dry and warty-looking, but usually moist and coated with a dirty grey secretion, of a peculiarly nauseous odour. They occur, sometimes in large numbers, about the genitalia, and also along the perinæum and round the anus, in the fold of the nates, in the groins, at the umbilicus, in the folds of the axillæ, beneath the breasts, in the neck, between the toes, at the angles of the mouth, and elsewhere. Not infrequently they occupy opposed parts of the skin, so as strongly to suggest local infection.* They may certainly be the means by which syphilis is transmitted to other persons. In such cases they generally give rise to a typical primary indurated papule, which is followed after the usual interval by constitutional symptoms; but it is equally certain that in some cases the affection which they set up in non-syphilitic individuals is indistinguishable from a flat condyloma, and cannot be proved to contain the syphilitic virus. So common are flat condylomata, apart from any other indication of syphilis, that careful observers have in some cases attributed them to mere irritation of the surface by dirt and moist secretions. Moreover in some countries they have been known to prevail endemically. It seems therefore likely that the peculiar characters of a mucous patch are due to local conditions, and that though, as a rule, of specific origin, it may be developed out of a non-syphilitic papule, just as a papilloma of the skin or a villous growth in the bladder may be of either innocent or malignant nature.

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* If this is the case it is one of great theoretical interest, since a patient who already has syphilis is believed to be absolutely protected from the further influence of the virus, so that the flat condylomata would possess an independent contagion peculiar to themselves. Although the affection owes its origin to syphilis, it may yet acquire a contagious principle of its own, which is capable of surviving and reproducing itself by contagion after the original syphilitic virus has in some way disappeared or become exhausted; if so, this tends strongly to corroborate the doctrine that the soft chancre arises and spreads in a similar manner.—C. H. F.

4. *The squamous syphilide* ("syphilitic lepra or psoriasis") usually develops out of the roseolous or papular forms as an independent eruption. It is distinguished by its small and scanty scales, the copper-like tint of the patches, and the absence of the definite and symmetrical distribution characteristic of psoriasis.

5. *The pustular syphilide* ("syphilitic ecthyma and rupia").—This consists of pustules of all sizes, each seated on a firm red base. They are sometimes present in immense numbers, especially upon the face and trunk. They come out rapidly in successive crops, which may be prolonged over many weeks; and they may relapse even after a year has passed. They dry up into brown, or dark green, or black scabs, and leave large stains, which finally pass into shallow, flat, white cicatrices. Sometimes they closely simulate pustular acne.

Concomitant early affections.—Like scarlatina, syphilis affects the *fauces* as well as the skin; but the sore throat often causes no discomfort, and must therefore be looked for. There is diffused redness, with swelling of the tonsils; and afterwards flat, greyish mucous patches are seen on the tonsils or the palatine arches; or yellow ulcers, with sharply defined red borders. A curious affection, seen not only on the fauces, but also on the hard palate, the cheeks, and the lips, consists of scattered milk-white spots (*plaques opalines*), like those caused by nitrate of silver. Their shape may be round, oval, or indefinite; they vary in size, and may run together so as to cover a large surface with an irregular pattern. Sometimes they are slightly puckered, and parts of their surface may be reddened, with only a little white opacity here and there. All their varieties are deserving of careful study, for they are very characteristic of syphilis. They run an exceedingly slow course, and may break out again and again, not only during the early stage, but also long afterwards.

The most common affection of the *eyes* is iritis, with formation of minute nodules near the edge of the pupil. This is generally bilateral. In Hutchinson's experience it arises within the first six months of the disease, if at all. Later attacks are said to be always relapses; they are often limited to one eye at a time. Occasionally retinitis may occur; the fundus of the eye has a hazy appearance, the disc is reddened and swollen and its margin is indistinct, and there may be many small extravasations of blood. The recognition of these changes is important, because if the ophthalmoscope is not used, the retinitis may easily be overlooked. The patient notices "nothing except that his sight is very dim;

he has no pain, no congestion of the front of the eye, no intolerance of light" (Hutchinson).

The *lymph-glands* in various parts of the body remain swollen during the secondary stage of syphilis; we look carefully for enlargement of those in the humerus, the groin, the back of the neck, and above the elbow.

Another symptom is partial *alopecia;* the hair may come away with the comb so freely that the patient becomes prematurely bald. The short hairs from the limbs are shed, as well as those of the scalp. This occurs irregularly in patches, unlike the complete circumscribed baldness of area and the local frontal and occipital thinning of hair as age advances.

Transitory *albuminuria* is not uncommon. Dr Fagge had one patient whose urine was albuminous at a time when it could hardly be supposed that the vessels had already become lardaceous.* The present writer had for some years a patient under observation who was the subject of secondary syphilis, and passed albumen in his urine without any other sign of renal disease.

After the lapse of six months or a year, or sometimes a longer period still, during which one or more of these secondary symptoms has developed, the disease often becomes again latent, even without treatment. In rare cases it has run its course, and the patient henceforth is free of it. But usually he is troubled, at intervals, with slight symptoms of the disease. Perhaps small scattered papules appear on the scalp, which are scratched by the comb, and scab over again and again; perhaps the palm of one hand or the scrotum becomes scaly; perhaps some of the nails grow rough and discoloured, or slight ulcers form on the tongue. To these symptoms Hutchinson adds recurrent herpes of the prepuce.

Tertiary stage.—In some cases, months or even years after the subsidence of the early syphilides, there appears one of the later forms of dermatitis, usually squamous, pustular, or ulcerative.

In 1869 a woman was in Guy's Hospital who had been infected by her husband twenty years previously; in her the disease assumed the form of thin reddish-brown glazed patches, covering the greater part of the face: they were not at all raised, and there was only the slightest possible desquamation; in fact, they were scarcely more than maculæ. In other cases, thick white scales appear around a red ring or festooned line. Or, again, large patches of skin may become thickened, raised, of a reddish-brown colour, and rough with a bran-like scurf. One of the most common of the late eruptions of syphilis are reddish-brown nodules, which cohere in rings or patches, so as to cover a large surface or to form straggling festooned lines. They leave cicatrices even when they have not ulcerated; but usually they become covered with conical crusts, beneath which form small deep ulcers with vertical edges (*rupia*). One may see patches a foot or more in diameter, some parts of which have already cicatrised, while others show recent nodules, or serpiginous lines of scabs, spreading over the healthy skin around them. All these varieties are worthy of the most patient clinical study, for they are absolutely characteristic of syphilis, and the patient has often no suspicion of their nature. They particularly affect the face (forehead, nose, and lips), the nape of the neck, the shoulders, the back, and the extensor surfaces of the limbs. Occasionally tertiary ulceration may simulate lupus, but the essential difference in nature, prognosis, and treatment forbids us to acquiesce in such a phrase as syphilitic lupus. We might as well speak of syphilitic cancer or rheumatic gout. Mere patches of brownish pigmentation are an infrequent but characteristic appearance in both later secondary and tertiary syphilis. They are sometimes met with on the neck in women, where they were described by Hardy as coffee-coloured patches.

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* He also met with two cases of jaundice, one of which quickly subsided, but the other ended in acute atrophy of the liver (cf. Dr Wilks's case, 'Path. Trans.,' 1867, vols. viii and xvii).

Mucous membranes.—The tertiary period of the disease is also marked by lesions of the throat and mouth, but not the same as in the secondary stage. *Plaques opalines* may continue to form, but the tongue now first becomes affected. Smooth and glossy patches appear, or greyish ulcers, or the mucous membrane becomes thickened, with deep intersecting grooves. The contact of salt and pepper, of vinegar or hot liquids, is exceedingly painful.

In other cases, again, the soft *palate* becomes affected with deep sharp-cut ulcers, which rapidly perforate it, and eat away its substance. Such ulcers, when they heal, leave well-marked cicatrices. Some years ago a woman died in Guy's Hospital whose velum had long before been extensively destroyed on one side, so that the uvula was held in its place by two thread-like processes of mucous membrane, which looked as if they would be torn through in deglutition; the preparation is now in our museum (No. 481). Sometimes the palate may become adherent to the pharynx, so as to cut off the communication between the nose and the lower air-passages.

The rectum is frequently the seat of tertiary ulceration, which, particularly in women, may lead to stricture of the gut by cicatrisation, and also may simulate carcinoma or dysentery by causing pain and tenesmus, with diarrhœa and passage of blood, pus, and mucus.

Other tertiary lesions of viscera.—The most characteristic lesion of the tertiary period is the "gumma"* or node. In the subcutaneous tissue, or in the substance of the tongue, gummata often acquire a considerable size; and the skin or the mucous membrane over them may at length become ulcerated through, so as to expose a grey degenerating mass of the most typical kind. But we cannot call tertiary eruptions gummatous to the exclusion of the earlier syphilides; for it has been shown that the nodules found in iritis are small gummata, not mere fibrinous exudation. Again, Hutchinson records a case in which definite gummata were found in both testes and also in the spleen, although a secondary rash was still out on the skin of the patient, who died of "syphilitic disease of the heart—myocarditis with a gumma."

Tertiary affections of internal organs are the most important of the effects of syphilis. They will be described among the local diseases of the brain and the meninges, the lungs, larynx, and liver. Here we need only urge the importance of carefully examining the bones and the testes, as well as the eyes, throat, tongue, skin, and lymph-glands, whenever a syphilitic taint is suspected.

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* The terms *gumma* and *gummositas* are no novelties. Fallopius, in the sixteenth century, spoke of tumours of bones as having been called "*gummata gallica*," on account of their containing a matter resembling *gummi eliquatum*. Ernst Wagner proposed to substitute the name "syphiloma" for gumma; but the change is not worth making. "Tubercles" is rightly abandoned, for *tubercula syphilitica* have nothing to do with tubercle in the modern sense of the term, although v. Bärensprung supposed that they had. "Node" is still often used as a synonym of gumma, especially in superficial regions.

It is believed that those *bones* which, like the skull-cap, clavicles, ulnæ, and tibiæ, are but thinly covered with soft parts, are more than others liable to be affected. Perhaps they are only more easily examined. By running the finger along the surface of these bones it is easy to discover whether there is any swelling, or whether the patient shrinks from pressure. The enlargement caused by syphilitic periostitis generally rises gradually from the surrounding surface, and when recent feels soft and semi-elastic. One sometimes at an autopsy finds a grey soft material, more or less caseating, which can be shelled out of the excavated substance of a bone. At a later stage this may be absorbed, and there may be left a central depression, with a thickened zone around it. But upon the bones of the limbs the whole of the gumma is more often converted into a uniformly dense, raised, bony mass.

The *testes*, one or both, are often swollen in the secondary stage of syphilis. Afterwards firm yellow gummata form first in the epididymis, and can often be felt during life. But in many cases a diffused fibrous thickening, with atrophy of the secretory structure, is all that one discovers at an autopsy. This probably accounts for the fact that so many syphilitic patients are unable to beget children. But one could hardly in the living subject recognise such an affection by palpation.

Gummata are found not only under the periosteum, in the integuments, and in muscles, including the heart and the tongue, but also in the brain, the lungs, the liver, the nerves, the testes, and the abdominal viscera.

Sequelæ.—The gummatous and other tertiary lesions just described are by some authors called the sequelæ of syphilis; but they are truly parts of the morbid process, probably capable in favourable circumstances of transmitting the contagion, and certainly amenable to the same remedies.

There are, however, other more remote effects of syphilis, which extend its injurious influence more widely and for still longer periods. They differ from the most belated or protracted tertiary lesions in never taking the form of gummata or showing any anatomical characters of syphilis, and in being little amenable—some experienced physicians say not at all amenable—to mercury or iodides. We only recognise their relation to lues by the frequency of the sequence, a frequency far beyond what can be explained as accidental.

The first of these sequelæ (or "parasyphilitic" affections, as Fournier calls them) is the remarkable degeneration known as lardaceous (waxy, albuminous, or amyloid), which we shall meet among the diseases of the liver, spleen, kidneys, and intestines. Its relation to syphilis will be discussed in the chapter on diseases of the liver.

Another is acute and diffuse myelitis, or softening of the cord, which appears to have more than a fortuitous relation to preceding syphilis.

A third is chronic induration (sclerosis) of the posterior columns of the cord, *i. e.* clinically tabes. To this must be added a sclerosis of the brain known as general paralysis of the insane.

Lastly, we must mention aneurysm, particularly in women and in men under fifty years of age.

Transference of contagion.—Ricord taught that primary syphilitic lesions alone possess the power of propagating the disease. But clinical experience furnishes many instances of contagion from flat condylomata, and experiments have been made which show that it is possible to convey the virus to healthy persons by inoculation with the blood of syphilitic patients, or with matter from pustules of a secondary eruption, or from an ulcer of the tonsils. There seems to be no doubt that the surface of the skin must be abraded, or fissured, to allow of the penetration of the virus; and probably this is true even of mucous membranes.

The contagion appears never to be conveyed by the natural secretions of the salivary, mammary, or lachrymal glands; by the semen, without impregnation of an ovum; or by inflammatory exudation from a mucous membrane—as, for instance, the gonorrhœal pus in one who has also had syphilis. Cases of infection from the act of kissing, from glass-blowing when the same tube is used in turn, from smoking if a man uses another's pipe, or (as in a case related by Dr Baxter) from cleaning the teeth with another person's brush,—all these are probably explained by the presence of actual ulcers or mucous patches on the tongue or lips. We must not forget that we may unintentionally infect our patients if we omit thoroughly cleansing a tongue depressor, a speculum, or a laryngeal mirror, after using it in a syphilitic case. The disease has also been transferred by one person biting another in the hand. Nor must we forget the rare but too common cases in which lues has been inoculated along with vaccinia. The writer has only seen one case of this lamentable occurrence, but it is less rare than it should be. Lastly, accoucheurs and midwives have often acquired primary sores on the finger when they have overlooked a slight abrasion while attending a woman with specific affections of the vulva or cervix.*

An important observation, made by Dr Abraham Colles of Dublin, was that when a mother bears and suckles a syphilitic child she does not have an ulcer on the nipple, whereas a wet-nurse does; *i. e.* a wife may be infected from a syphilitic father through her own embryo, and so rendered immune (cf. *infra*, p. 355). Exceptions have been recorded to "Colles's law," but Mr Hutchinson has no doubt of its general validity.

John Hunter supposed that the virus of lues does not reach the blood until after it has multiplied itself at the seat of inoculation, and has passed along lymphatic vessels into the veins. But it is more likely that the lymph and blood are immediately poisoned, as in vaccination, and that the subsequent induration at the seat of inoculation is itself the result, not the cause, of infection of the whole body. The rarity of multiple primary syphilitic sores, and the fact that the secretion of a primary sore is scarcely ever inoculable upon the patient, point strongly in the same direction. Moreover the practice of early excising indurated chancres is now abandoned because found to be useless, the explanation being that the whole body is infected before the induration appears, and so the excision comes too late.

Wilks believes that visceral lesions are most frequently met with in cases where cutaneous eruptions and other symptoms of syphilis had been absent or slight; but, if so, we should still ask whether it is not the omission of early specific treatment in such cases which determines the remote effects. Hutchinson, moreover, says that "those who suffer severely in the secondary period often do so also in the later ones. All the cases of so-called 'malignant syphilis' are instances of a severe secondary relapse imperfectly combated." He regards the tertiary lesions as sequelæ rather than symptoms of syphilis—"regrowths" in morbid structures left behind from the secondary period; and remarks that tertiary symptoms sometimes break out after the patient has produced a family of healthy children.

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* With regard to the ways in which syphilis is transmitted, it is perhaps worth notice that among married women of the lower class the disease is often traced back to a confinement; probably the patient really derived it from her husband after her convalescence, he having become infected in adulterous intercourse while she was in childbed.—C. H. F.

Diagnosis.—The first glance at a patient often reveals to a skilled observer the presence of syphilis; and he is bound to treat it, particularly in a married woman, without asking questions which might cause endless domestic misery. Even when inquiries can be pushed without reserve, they may mislead rather than guide. It is important that the body should be stripped, and that every part of it should be looked at before one pronounces as to the character of a doubtful eruption. Some of the most puzzling cases are those in which independent diseases are present at the same time; as when a cutaneous syphilide is almost hidden among the nodules of acne indurata, or the papules and pustules of scabies. Of the other organs, the irides, the soft palate and tongue, the lymph-glands (particularly those of the groin and neck, and the supra-trochlear glands), the subcutaneous bones, and the testes are those which are most important to examine with eye and hand.

The following hints may perhaps be of service in making what is often a difficult and always an important diagnosis.

Apart from the colour and the multiformity of syphilitic lesions of the skin, the absence of itching with the superficial and of pain with deeper ones is a valuable character, as well as their irregularity of distribution. Lesions which occupy the upper arm, the flanks, the inner aspect of one thigh, or the fibular region of one leg, are likely to be specific, because not likely to be anything else. In all unusual, puzzling, obscure cases one should not forget the possible presence of lues. Syphilitic pains are worse at night, and are more frequent in the head and limbs than in the back and loins. Ptosis and ocular paralysis in general are suggestive of syphilis, and so are irregular patches of anæsthesia.

The test of reaction to mercury or iodides, though a true one, is not to be recommended. Diagnosis should precede, not follow treatment, and if we only give remedies in a tentative, doubtful way, we are not likely to give them in the doses or with the perseverance which will ensure success.

Lastly, the less we depend on history, and the more on facts, the less often shall we be deceived. The origin of syphilis is sometimes as difficult to make out as that of scabies, of scarlatina, or of enteric fever. The fact remains. Syphilis is sometimes non-venereal, it is often innocently acquired; whereas blennorrhagia and soft chancres are usually, and scabies occasionally venereal in origin.

Prognosis.—A patient affected with syphilis is naturally anxious to know whether the disease will leave a permanent taint. He has heard of instances in which, after the lapse of many years, tertiary symptoms have appeared, and has been told that the syphilitic poison differs from all other specific diseases in never being eradicated. But we are justified in assuring him that the distinction is one of degree rather than of kind. In the majority of cases a person who has had syphilis, and been properly treated, is after a few years free from it in every sense. As with variola, the only appreciable difference between the patient and other people is that he is almost incapable of taking the infection. Nevertheless there are many cases in which, notwithstanding careful and long-continued treatment, relapses occur again and again. True, they can be cured again and again; but the liability to them seems to be ineradicable.*

To adults syphilis is scarcely ever directly fatal.†

Indirectly, as we shall see in the chapters on diseases of the brain, larynx, lungs, liver, kidneys, syphilis is the cause of a very large mortality. If, therefore, its prognosis is favourable, it is because by proper treatment we can not only cure the early symptoms, but prevent their more dangerous after-effects.

Opinions probably differ widely as to the extent to which the presence of a syphilitic taint *modifies* other diseases. Some surgeons have held that it may interrupt the natural course of almost every morbid process; that a broken bone may fail to become united, and a wound refuse to heal, until the virus is neutralised by the administration of specific medicines. All this is, however, either hypothetical or practically very rare. No doubt an injured part may gradually take on the characters of a syphilitic sore, but there is no reason to believe that the effects of scarlatina or diphtheria on the throat, of rheumatism on the joints, or of psoriasis, scabies, and eczema on the skin, are the least modified by the presence of syphilis, for the two diseases may sometimes be seen in the same person, each unaffected by the other.

With regard to *protection*, it is well known that a person very rarely has an indurated chancre and afterwards a secondary eruption and sore throat on two different occasions; but several instances of such an occurrence have now been recorded. In a case of Hutchinson's the interval of health was only three years; it has generally been much longer. The second attack has always been very mild. On the whole, syphilis protects against itself more perfectly than measles or scarlatina, though perhaps not so perfectly as typhus and smallpox.

Treatment.—The use of mercury as a remedy for syphilis was known as early as about 1510, when Fracastorius wrote:

> "*Argento* melius persolvunt omnia *viro*
> Pars major; miranda etenim vis insita in illo est."—'De Morbo Gallico,' ii, 270.

Unfortunately the experience which appeared to justify its use was long vitiated by an obvious source of error; the natural course of the disease, when unmodified by drugs, had never been studied. Moreover the metal was given so freely as to cause profuse salivation, and that in all venereal diseases indiscriminately. Thus, when certain English army surgeons, in the early part of the present century, introduced a "simple treatment," in which mercury found no place, they had the advantage that their patients remained free from the evils of treatment by powerful means without previous diagnosis.

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* On this question see Dr Gowers' somewhat too gloomy statements in his admirable lectures on syphilis ('Brit. Med. Journal,' 1889).

† The only case that has come down to the *post-mortem* room at Guy's Hospital during the last thirty years (1880) in which death was attributed to syphilis, apart from any visceral lesions, is that of a man aged thirty-one, who lay in one of the wards for seven months with a rupial eruption on the skin, and complaining of pain and swelling in his joints. The most extreme wasting took place; his knees and his left arm were rapidly contracted; his wrists and ankles were enlarged. Towards the last he was drowsy and listless, and at the autopsy the brain appeared to contain an excess of fluid.—C. H. F.

It is, however, now established that although syphilis may subside spontaneously, yet this is the exception.

And experience tends irresistibly to the conclusion that a patient who has syphilis is greatly benefited by a judicious and thorough course of mercurial treatment. It has been longer tried than any other specific remedy, and has stood the test of accumulated experience. Among those who throughout the civilised world are best qualified to form a judgment upon the matter there is at the present time hardly any difference of opinion; and what adds to the force of this consensus is that it has been arrived at by divers paths, after prolonged controversy, and by many who were once in doubt.

It is certain that mercury, properly administered, is in no way injurious to the patient's general health. Even in the case of a person with a tuberculous tendency, the drug may be given with perfect safety. Nor should we forget that in a patient suffering from supposed phthisis, cirrhosis of the liver, or renal dropsy, the "phthisis" may possibly be syphilitic peribronchitis and interstitial pneumonia, the "cirrhosis" syphilitic gumma of the liver, and the "Bright's disease" lardaceous degeneration from syphilis. The cachexia resulting from syphilis, so far from being a reason for avoiding mercury, is often quickly curable by its use.

Mercury has a most marked effect upon the induration of a primary infecting papule or chancre, and if administered systematically during that period of the disease will often prevent the development of secondary symptoms. This is a most important point. Formerly Mr Hutchinson thought that secondary symptoms were for the most part inevitable whatever treatment might be adopted. But in a series of eleven cases of vaccino-syphilis in which he began to give mercury two months after contagion, and about a fortnight after the appearance of the specific induration, not only did every patient quickly get rid of chancre and glandular enlargement, but only about half of them ever showed secondary symptoms; these, moreover, were almost always of an exceedingly mild type, and did not appear until many weeks later than they would have done if no medicine had been taken. Now where syphilis has been intentionally inoculated upon healthy persons constitutional effects have scarcely ever been wanting; and the same may be said of cases of vaccino-syphilis in which early mercurial treatment has not been adopted.

If secondary symptoms occur, mercury renders them mild and of brief duration. Upon this point also Hutchinson speaks very decidedly. Formerly he cited instances in which a patient, while actually salivated for iritis in one eye, became attacked with the same affection in the other eye, as tending to establish an opposite conclusion; but now he says that in such cases the failure is probably due to the fact that the "salivation is premature,"—that is, that the remedy has not yet exerted its full influence upon the rest of the body, notwithstanding that it has affected the gums so severely. "The secret of success is to avoid any interruption of this kind," and he insists upon the fact that cases in which the secondary symptoms of syphilis are exceptionally severe are almost always cases in which mercury has not been given.

Mercury, if administered efficiently during the primary and secondary stages of syphilis, will probably diminish the frequency and severity of tertiary symptoms. Here, indeed, it is and must be difficult to obtain direct proof, for there are no certain data as to the proportion of untreated cases of syphilis which result in tertiary symptoms: nor do we yet know to what extent their occurrence may depend upon the severity and character of the secondary symptoms. But there is at least a strong presumption that if the drug can prevent the earlier and more constant symptoms of syphilis, it will not have less power over its more remote and exceptional effects.

If these statements are correct—and they are backed by the most experienced authorities in this country and abroad—it is evidently the bounden duty of a medical man to prescribe mercury to all patients suffering under primary or secondary syphilis.

Methods of administering mercury.—These are many, and the selection of one or the other a matter of convenience, and sometimes perhaps of fashion. Calomel or blue pill is only necessary to produce a rapid effect on iritis, retinitis, or gummatous meningitis. Plummer's pill is very useful in these not infrequent cases.

It is hard to believe that the hypodermic injection of the bichloride of mercury has any advantages which counterbalance its inconvenience, its costliness, its pain, and its tendency to set up inflammation; yet this is almost exclusively used as a method of treatment in some Continental cities, where patients undergo hundreds and thousands of intermuscular injections. Not only watery solution of corrosive sublimate, but various other vehicles have been devised for this purpose, one of the most popular being an emulsion of calomel in olive oil. Another plan, that of intravenous injection of mercurials, is also advocated. It can only be justified by the supposed necessity of rapidly combating some dangerous symptoms, but this can be done at least as rapidly and far more safely by inunction.

Mr Hutchinson relies exclusively on one-grain doses of grey powder (Hydr. c. Cretâ), with a fifth of a grain of opium to prevent diarrhœa, taken as a pill four, six, or seven times a day.

The inunction of grey ointment is an old-fashioned, but safe, speedy, and harmless method of exhibiting mercury. The only objections are its dirtiness and the difficulty of concealing the treatment. Another efficient plan is the calomel vapour-bath of Mr Lee. About fifteen grains may be volatilised by a spirit lamp over a water-bath, the apparatus being placed beneath a chair on which the patient sits naked, and wrapped round with blankets. The bath should last from fifteen to twenty minutes.

In chronic cases, such as usually come before the physicians, there is no better method than giving a course of solution of the perchloride in doses of one sixteenth to one twelfth of a grain (ʒj—ʒiss of the Pharmacopœial solution) three times a day. Another preparation which may often be used with advantage is grey powder, in doses of two or three grains. When the gums are affected, further inconvenience may be obviated by letting the patient at the same time take the chlorate of potass, or use it as a mouth-wash; but, as a rule, it is not desirable to mask the effect of the mercury in this way.

Except during the cold seasons of the year the patient may go out, and continue at his occupation, while he is carrying out a mercurial treatment. He should keep early hours, wear flannel, eat well, and abstain from stimulants. Pure air is important, and there are greater advantages in residence at the sea-side than at Aix-la-Chapelle or other Continental spas, where, if report says true, the real method of cure is neither the baths nor the waters, but mercury.

The old practice of salivating the patient followed the belief that the poison of syphilis was thus eliminated. It is unnecessary and harmful; but the rule of giving mercury "until the gums are touched" is a good one. The dose should then be diminished, so as to gain the utmost therapeutical short of the physiological effect.

Iodides.—In the tertiary stage, and particularly when periostitis or gummata are present, we have a second specific remedy for lues in Iodide of Potassium. Over the early macular and papular eruptions it has little if any power, but the effects of this drug as seen in the treatment of headache and nocturnal "rheumatism," of nodes and gummata, are among the most striking in therapeutics. Equally marked is its effect in gummatous meningitis, in syphilitic hepatitis ("cirrhosis" with gummata), and in tertiary lesions of the testes, bones, joints, and lungs. But after the pains and other symptoms of a gumma have been removed by its use, the patient should undergo a prolonged course of perchloride or biniodide of mercury. With regard to the dose of iodide there has been much difference of opinion. Of late it has become the general practice to give from ten to fifteen grains three times a day. It is best prescribed with aromatic spirit of ammonia, but some persons take it more readily in milk. When the iodide of potassium produces unpleasant symptoms, they may sometimes be avoided by substituting the sodium salt; and some patients appear to benefit by this remarkably. Very frequent small doses (four or five grains every or every other hour) is probably the best way of giving either of the iodides when it is important to bring the patient rapidly under the influence of the remedy.

Iodide of potassium exerts so marked an influence over the symptoms that the patient will persevere with it for years. In such cases it is generally desirable to interrupt its use from time to time; and important services are then rendered by the Chlorate of Potass. Obstinate forms of cutaneous eruption may be removed by this salt, given in doses of ten grains three times daily. Another medicine prescribed in the chronic dyscrasia of lues is Sarsaparilla. The fluid extract given in full doses is sometimes followed by incontestable results. Guaiacum, which three centuries ago had so high a reputation as a sudorific remedy in syphilis, has now completely lost it.

The success of what is called "serum-therapy" in the case of diphtheria, and its hoped-for success in other infectious diseases, has led to repeated attempts to cure syphilis by injection into the veins or the subcutaneous lymph-spaces of serum derived from an immune animal. Inasmuch as none of the domestic animals are susceptible to syphilitic contagion, there would seem to be no difficulty in finding antitoxic serum. Hollmann in 1890 appears to have made the first trial of this method, and reported complete failure after careful and repeated trials. The attempt has been renewed with lamb's and calf's serum by Tommasoli, Fournier, Cotterell, Valentine Mott, and Neumann. Some cases, more or less favourable, were reported, but on the whole the treatment has been found useless against the disease, and injurious by producing urticaria, erythema, and lymphatic inflammation (see the candid account given in Héricourt's 'Sérothérapie,' pp. 45—53).

It is sometimes well to apply remedies directly to the eruptions of syphilis; local tertiary affections rapidly subside when they are covered with diluted mercurial ointment, and calomel should always be dusted upon "mucous patches" (*i. e.* flat or moist condylomata). Iodoform ointment (ʒss—℥j) is the best application to foul and sloughing sores, and the phagedænic ulceration which is still sometimes seen is successfully treated by full doses of opium, with porter or spirits internally, and bark, quinine, or sarsaparilla.

In the sequelæ which follow syphilis, but are not themselves syphilitic, in aneurysm and endarteritis, in tabes, and general paralysis of the insane, it is doubtful whether iodides are of any service, and even mercury has only occasional and doubtful success.

Congenital Syphilis.—When lues, instead of being acquired by direct infection, is transmitted to the patient from one or both parents, it differs in some respects from ordinary syphilis in its symptoms, and is of course without a primary stage. Some writers prefer to call it "hereditary" or "inherited," because it does not show itself by well-marked symptoms until some weeks after birth. But the bones in the fœtus, the skin, and the placenta, are often affected by syphilis, and there is no doubt that the malady is present not only at but before birth. The term hereditary is better reserved for diseases such as gout and phthisis, which begin at a later age, usually many years after birth, whereas a child is syphilitic while still in the womb.

The case of congenital syphilis does not, it may be observed, raise the difficult question whether acquired peculiarities are transmitted. For syphilis is not, like scars or supernumerary fingers, a structural variation; nor is it, like grey hair or gout, the result of a supposed transmitted tendency; it is a poison which is directly conveyed by a process of infection from the parent to the sperm-cell or the ovum.

Paternal and maternal inheritance.—It was once thought that infection can only come to the fœtus *in utero* from the mother. But it is now ascertained that the mother of a syphilitic child often shows no sign of the disease, and it is now admitted that the semen may convey syphilis directly to the ovum. A man may transmit the disease to his children, although he may not marry until long after the disappearance of secondary symptoms; but such cases are rare and exceptional. If consulted about the propriety of marriage by a patient who has had syphilis, one should perhaps never declare it to be impossible for the offspring to be tainted: until two years have passed after the complete subsidence of the secondary symptoms of the disease marriage must be altogether forbidden, but after that interval it may be allowed; for lapse of time, as a rule, quickly diminishes the risk. Hence each succeeding child of the same parents is less likely to suffer than the previous one; sooner or later the taint wears out, and the children subsequently born remain free. Sometimes, however, one infant may escape, notwithstanding that those younger are attacked. There are modifying circumstances which may cause the later children of a marriage to suffer more than the earlier, even when the syphilis had been contracted long previously. A healthy woman, impregnated by a syphilitic husband, gradually becomes herself syphilitic without ever having suffered from a primary lesion, and thus forms a second source of infection for her future children.

The full extent and subtlety of the contamination which affects the mother of a syphilitic child were discovered by Colles, of Dublin, in 1837. As mentioned above (p. 348), he laid down the remarkable law which now bears his name, that "a newly born child, even although it may have symptoms in the mouth, never causes ulceration of the breast which it sucks if it be its mother who suckles it, though it is still capable of infecting a strange nurse." In other words, although the mother may have shown no sign of syphilis, she has yet undergone a modification of the disease, bearing somewhat the same relation to the ordinary form that vaccinia does to smallpox, and no less protective in its action. The *choc en retour* (as French writers have termed the infection of a woman through her fœtus) is not, however, always unattended with symptoms.*

Effects on the ovum.—The results of syphilitic infection on the fœtus vary greatly. Often it dies *in utero*, and is thrown off after an interval more or less decomposed, with its cuticle peeling in large flakes from maceration in the dark and fœtid liquor amnii. Thus repeated abortions and miscarriages afford valuable indications of a syphilitic taint.

The existence of a specific lesion of the *placenta* had long been suspected; and in 1863 Dr Wilks cited in the 'Guy's Hospital Reports' some cases observed by Mr Wilkinson King, in which the chorion had been found thickened and the amnion lined by a false membrane, as the result, it was supposed, of syphilis; in almost every instance abortion had taken place at the third month. Virchow afterwards described syphilitic disease of the decidua and the maternal part of the placenta. Most of the hard yellow masses in the placenta supposed to be gummata have been mere residues of accidental hæmorrhages. But gummata spreading from the maternal into the fœtal part of the placenta have been recorded.†

Effects at birth.—As a rule, a child infected with syphilis has no external symptoms of the disease when it is first born at full time.

One exception is a bullous eruption, which is known as *pemphigus neonatorum*, and which may either be present at birth or come out a few days later.‡ The bullæ are flaccid, and contain opaque serum or pus; they are most often seen on the soles of the feet and the palms of the hands. This eruption generally proves fatal.

The *thymus* has been found in a state of suppuration by Dubois; there was no abscess, but the organ when squeezed emitted drops of yellowish pus, easily distinguishable from the opaline liquid which it may contain when in a normal state. Another morbid condition, first pointed out by Depaul as occurring in the bodies of syphilitic children, consists in the presence of yellowish-grey indurated nodules in the *lungs*, softening in thei centres into cavities.

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* On this subject the reader is referred to Dr George Ogilvie's valuable papers on "Immunity in Congenital Syphilis," in the 'British Journal of Dermatology' for 1899, pp. 45 and 49.

† Fränkel, of Breslau, in the 'Archiv für Gynäkologie' for 1873 recorded a series of investigations on this subject. In sixteen cases he discovered a peculiar affection of the villi, *i. e.* of the ovum as distinct from the mother. The lesion consists in a dense growth of round- or spindle-cells in the substance of the villi, attended with destruction of their vascular loops, and ending in fatty degeneration. Villi so altered are swollen and opaque, and have irregular outlines and bulbous extremities. Sometimes the whole placenta is uniformly affected; it is then remarkably large and heavy (even to the weight of two pounds), close, tough, and of a pale greyish-yellow colour. In other instances only parts are diseased—opaque wedge-shaped masses, surrounded by zones of congestion. Once Fränkel found that the cell-growth had extended from the villi into the maternal part of the organ. Premature expulsion of the fœtus usually took place at varying periods from the sixth month onwards.—C. H. F.

‡ As early as 1851 there was a discussion upon this affection in the Academy of Medicine at Paris; Paul Dubois declared that it was syphilitic, while Cazeaux maintained the contrary.

Again, a peculiar change in the *liver* has been frequently recorded in cases of congenital syphilis, by Gubler, by Wilks, and by Lancereaux: the organ is not only enlarged, hard, and elastic, but its cut surface shows a number of small, white, opaque grains on a uniform yellowish ground. This is a diffuse intra-lobular cirrhosis.

The *spleen* is often larger and firmer than usual, and acute peritonitis, or pleurisy, or meningitis has sometimes been found to be the cause of death in a new-born syphilitic infant.

In many cases of congenital lues there is found after death a peculiar condition of the *bones*. This affection, first described by Wegner, of Berlin, in 'Virchow's Archiv' for 1870, but independently observed by a French physician, Parrot, seems to begin early in life. As in rickets, its chief seat is at the meeting lines between the shafts of the long bones and their epiphysial cartilages, and, in the case of the ribs, between them and the cartilages which tip them. The meeting lines in question become much thicker than natural, uneven, and irregular. But here the resemblance ceases. There is in syphilis little increase of the normal "zone of proliferation," whereas the "zone of incrustation of cartilage" is greatly exaggerated. It forms a thick layer, dense and homogeneous, but friable, white, and opaque, like mortar; long processes project from it into the substance of the cartilage beyond. As the affection advances, this layer becomes separated from the shaft by a soft greyish-red or yellowish material, consisting of granulation tissue, which may shade off into pus. Another feature is the formation of new bone round the shaft in the neighbourhood of the epiphysis.

Wegner terms this lesion a "syphilitic osteochondritis." It is widel diffused throughout the body, but it is said to be always most mark where the growth of osseous tissue is naturally most active; and what interesting is that precisely the same distribution is stated to obtain in t case of rickets. Thus the lower end of the femur is the favourite seat both diseases, while the corresponding part of the humerus is least of affected by either. Parrot, however, says that in syphilis the new bone most abundant just where the other changes are least developed; and t corresponds exactly with a remark long ago made by Hutchinson to effect that periostitis in congenital syphilis is more often met with j above the elbow-joint than anywhere else.

In a few exceptional instances, when an infant infected with syphili born alive, the soft material between the shafts of the long bones and th epiphyses increases to such an extent as to detach them from one anoth and form fluctuating purulent swellings beneath the periosteum. There then complete loss of power in the limbs, attended with so little pain t it has actually been mistaken for paralysis. The child's hands are descri by Parrot as lying pronated by its side; its legs are extended, and w it is lifted up they hang helpless and swing to and fro. After death alm all the principal epiphyses may be found separated from their shafts.

Cases of this severe kind are easily recognised, but it is otherwise the ordinary form of the disease, in which there is seldom sufficient largement of the ends of the bones to justify a positive diagnosis, at le in very young infants. Indeed, Köbner has shown that even after de the microscope is often required to discover the lesion.

These lesions of congenital syphilis are to be carefully distinguished from those of Rickets, as will be explained in a subsequent chapter on that disease.

Effects after birth.—The symptoms of congenital syphilis first show themselves towards the end of the first month after birth or in the course of the second month, rarely after the end of the third month, but sometimes when the child is a year old.

The earliest is usually *nasal catarrh*, producing what is called "the snuffles." The nostrils discharge a fluid, at first thin and serous, but afterwards viscid, so that it dries up and obstructs the passage of air. The child is no longer able to breathe while it is at the breast; it takes the nipple into the mouth only to drop it again, and thus, as Diday showed, it fails to get enough food, and rapidly loses flesh. Indeed, as a rule, syphilitic infants are from the first pale and wasted, or soon become so; they have a dull, sallow, wrinkled skin, and "look like little old men."

Soon after the catarrh there appears an *eruption*. Its favourite seats are the face and the nates, but it may cover the whole of the body and limbs. It consists of maculæ, blotches, or flat papules, usually brownish or copper-coloured. It is sometimes difficult to distinguish between syphilis and the effects of the nurse's negligence in keeping the parts about the anus clean, or in drying them sufficiently after washing. But the red blush produced by mere irritation is ill-defined and fades away at the edge, whereas at the margin of a specific rash there are seen isolated, sharply outlined blotches. The inflamed surface, moreover, is uniform and bright red in ordinary dermatitis; it is patchy and yellowish or brownish ("coppery") in congenital syphilis. The papules may have smooth and glazed surfaces, or they may be dry, raised here and there into flaccid bullæ, or superficially ulcerated. At the angles of the mouth, in the folds of the neck or of the groins, or round the anus, the papules often become covered with a moist opaque layer, and form flat condylomata. These "mucous patches" are seldom seen in children less than eight or ten months old. Sometimes the eruption takes a pustular form, and a great part of the body may be covered with scabs.

Ulcerative *stomatitis* is often present. It may involve the gums and dental sacs; Hutchinson has seen it lead to necrosis and exfoliation of the alveoli. The mucous membrane of the nose may likewise ulcerate and discharge a sanious fluid, and this may lead to perforation of the septum.

Iritis is rather rare at an early stage. It is usually seen at about the age of five weeks. Whether limited to one eye or not, it is marked by irregularity of the pupil, alteration of colour in the iris, and the exudation of white, yellow, or red lymph. It must be carefully looked for, as the cornea is generally clear.

Congenital lues is either cured by treatment or subsides of itself about the end of the first year, and it is rare for cutaneous eruptions to recur. But affections of the bones and joints may supervene, and are particularly apt to develop about the time of puberty.

Later effects.—Periostitis is the most frequent symptom between the child's third and twelfth years. Hutchinson says that the bone most often affected by nodes is the humerus at its lower end, and that they often reach such a size as to impair the movements of the elbow-joint. A girl of eight or ten years old once came to Guy's Hospital with large ulcerating rupial patches, covered with thick brown crusts, on her trunk and limbs. Under iodide of potassium they healed with marvellous rapidity, but she returned with extensive periosteal swellings upon the tibia and ulna, and it was two or three years before she was finally cured. At the Evelina Hospital a girl of about the same age had a sloughing ulcer which destroyed the soft palate, and also great enlargement of the metacarpal bones of one hand. Hutchinson cites the case of a boy aged eight, almost the whole of whose calvaria was involved in disease at first regarded as "strumous," but which proved to be syphilitic; he was the son of a clergyman, but his mother had contracted the disease from a former husband, an officer in the army. Nodular swellings, doubtless gummata, in syphilitic children's testes have once or twice been found at Guy's Hospital after death. Lardaceous disease, as the result of congenital syphilis, is extremely rare. Dr Hale White records one case in a child of seven, affecting the liver, spleen, and kidneys ('Path. Trans.,' 1888, p. 444).

The most interesting of the later effects of the hereditary disease differ altogether from those of acquired syphilis. Their discovery is due to the patience and acumen of Mr Hutchinson, to whose masterly account little has been added by any writers but himself. One peculiarity is that on each side of the forehead *the frontal eminence is protuberant.* Another is the presence of four eminences on the bones forming the sides of the anterior fontanelle; from their resemblance to the buttocks, Parrot has called such a calvaria "natiform."*

The other signs of congenital syphilis established by Hutchinson are more distinctive. One is a *sunken bridge of the nose;* another, the presence of radiating *linear cicatrices* at the angles of the mouth; and a third is a thick and sallow condition of *the skin*, the hair being scanty, and the nails apt to break.

Most characteristic of all is a peculiar change in certain of the permanent teeth, and particularly in the *central upper incisors* of the permanent set, the "test-teeth." Not only are they much smaller than usual, but they are "notched" and "pegged;" that is, they present a single deep crescentic excavation of their free edge, and a gradual convergence of their sides towards this edge. It is supposed that this alteration in their form is the result of their having been disturbed in their growth by specific stomatitis during infancy. Similar changes may sometimes be observed in the case of the other incisors. It is important to note that the single deep notch is alone indicative of syphilis: horizontal grooves on the surface, and numerous small notches in the edge, are common enough in persons who are altogether free from any congenital taint. It is, moreover, certain that the defects of the teeth due to mercurial salivation during dentition is quite distinct from that of syphilis. The milk-teeth are unattacked by congenital lues.

The appearances just described enabled Hutchinson to identify as syphilitic an affection of the eye, the real nature of which had not previously been suspected, and which was known as "strumous corneitis." There can be no doubt that the "ugly form" of scrofula (struma) described by Watson and other writers was for the most part syphilitic, just as the "pretty form" was tuberculous. *Interstitial keratitis*, as it is now called, occurs chiefly in children between eight and fifteen years old, but sometimes in adults up to the age of twenty-five or twenty-six. It is more common in girls than in boys. It seldom remains limited to one eye, but commonly attacks both in succession at an interval of a few weeks. It begins as a dotted haziness near the centre of the cornea, which spreads until almost the whole of it is opaque, like ground glass. The patient often complains but little of intolerance of light, and there is not often much congestion of the conjunctiva or sclerotic at first. Ulceration never occurs, but at a certain stage the cornea may become so vascular as to be uniformly pink or salmon-coloured. When the affection is at its worst, vision is often reduced to a bare perception of light; but in the course of a year or eighteen months surprising improvement takes place. The opacity slowly clears up, until perhaps only a few hazy patches remain; and the patient's vision may ultimately be little impaired. Sometimes iritis occurs as a complication, or choroido-retinitis.

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* He also claims for congenital syphilis the affection known as Craniotabes, which has hitherto been deemed rachitic. But this is more than doubtful. See on this and other points Fournier's 'La Syphilis héréditaire tardive,' 1886.

Another morbid condition occasionally seen in the subjects of congenital syphilis is *deafness*, apparently from some disease of the deeper parts of both ears. It belongs to the same age as interstitial keratitis, and with that affection of the eyes and the notched incisors makes up what Continental writers call the Hutchinsonian triad.

It is worthy of remark that not only are these symptoms peculiar to the congenital form of the disease, but that some of the common effects of acquired syphilis in its later stages are absent, or very rare, when it is hereditary, particularly lesions of the testes, of the liver, and of the arteries. Interstitial keratitis is very rare in acquired syphilis.

Transference from the child.—Although, in accordance with Colles's law, a child suffering under congenital syphilis is incapable of infecting its mother, it may give the disease to any other woman who suckles it. Diday records cases in which women advanced in years appear to have contracted syphilis by kissing such infants; and the use of a spoon that had previously been employed in feeding them has been followed by the same terrible result. Probably the source of the virus is always the secretion of an ulcer on the lips or in the mouth. The primary lesion on the nipple of a nurse is described by Diday as a small red desquamating papule. When once infected, the wet-nurse in her turn may convey the disease to other infants. That the milk is not the vehicle of the poison is shown by an observation of the late Mr Henry Lee's. A woman, who was nursing a syphilitic child, acquired an ulcer on that breast, and afterwards an eruption; but her own infant, for whom she reserved the other breast, went on sucking for six weeks and remained healthy.

Syphilis contracted by an infant from a wet-nurse does not appear to differ in its symptoms from the inherited form of the disease.

Protection.—Is a subject of congenital syphilis less liable than others to acquire the disease later on in life? Hutchinson has recorded instances in which chancres have been contracted under such circumstances, and one in which constitutional symptoms occurred; and his opinion is that the fact of a patient's parents having had syphilis renders the acquired disease milder, but does not altogether prevent it. The writer saw repeatedly a patient with obvious signs of congenital lues, who was also the subject of the acquired disease, with secondaries and subsequent nodes.

Transmission.—Is it possible for the taint to be handed down to a third generation? Hutchinson has repeatedly had opportunities of examining the children of persons who were undoubtedly the subjects of congenital syphilis, and with one exception they appeared to be healthy.

Treatment.—Congenital syphilis seldom proves directly fatal if the infected fœtus is born alive and survives the first week of extra-uterine life. In mild cases the eruption may subside in a few weeks without treatment: and in others specific treatment is very successful. The powder of mercury and chalk should be prescribed in doses of a grain or two grains, two or three times a day, according to the age of the infant; a minute quantity of Dover's powder or of carbonate of soda may be given with it, according to circumstances. Or the diluted mercurial ointment may be used externally: it may be applied on a flannel band sewn round the knee, as Sir Benjamin Brodie recommended; or ten grains of it may be rubbed into the child's armpits once a day. Mucous patches about the anus should be dusted over with calomel.

In the later forms of congenital lues, mercury and potassic iodide are to be used as in the acquired disease; that they are no less efficacious the following case serves to show.

A girl of twelve or thirteen was once brought to the writer by her mother with a gumma of one leg, which had sloughed and formed a cachectic though almost painless ulcer. There were other signs of congenital lues, and iodide of potassium was prescribed with the prediction that great improvement would be seen in a week. On the patient's next appearance, however, the sore was much worse. An increased dose of the remedy was ordered, but at the end of the second week the disease had gone on increasing, and the leg looked more threatening. The mother was then cross-examined, and at last confessed that the patient had not taken a single dose of the medicine. She was threatened with the possible loss of her leg, and her mother made her take the iodide. At the end of a week the ulcer was healing, and very speedily the girl was well.

TUBERCULOSIS

> **"All these facts speak, as it appears to me, so eloquently and decisively for the infectious nature of Tuberculosis, that we are not shaken in this conviction by the direct demonstration of the tuberculous virus being up to the present time an unsolved problem."**
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> COHNHEIM. 1881.

Theory of tuberculosis—Anatomy and histology of tubercle: views of Laennec, Bayle, and Louis: of Virchow, Schüppel, and Ziegler—Caseous and fibrous transformations of tubercle—Tubercle a specific infective disease—Koch's tubercle bacillus—Transference of tubercle—Tuberculosis in animals: Perlsucht—Predisposing causes—Invasion and spread of tubercle in the body—Acute tuberculosis—Varieties of chronic tuberculosis: phthisis pulmonum, laryngis, et ilei; tuberculous inflammation of serous membranes; genito-renal tuberculosis; adrenal tubercle; tuberculosis of the lymph-glands and spleen; tuberculous disease of bones and joints: multiple tubercle generally—Concluding summary.

Definition.—A specific infective disease, accompanied by fever, and due to the entrance of an ascertained microbe; running a prolonged course, and varying in its symptoms as it affects different organs.

General pathology.—Tuberculosis is one of the most important diseases, for under various forms and with very different local characters it affects almost every organ of the body. It must now be included in the extensive group of specific infective fevers. Like syphilis, it usually comes before us as a chronic malady, and under one of its local manifestations. It differs widely in its clinical aspect from the continued fevers and exanthems: but, as in the case of syphilis, it is of practical as well as of scientific importance to recognise its unity and its essential likeness to other bacterial infections.

The presence of the infecting virus is so wide-spread that it is absent in very few parts of the world, but susceptibility to it is happily far less common: hence the predisposing or resisting conditions are much more important in this than in any other infective disease. The healthiest organisms appear to have scarcely any power of resisting the invasion of smallpox, of syphilis, or of plague, but many are exposed to scarlatina who do not take it; and this power of resistance is far more common in the case of tubercle.

When the specific bacillus has gained an entrance it is at first only active locally, but it is always liable to spread further by direct contact, by the lymphatic channels, or by the blood-current; and the infection involves transference and multiplication of the microbes themselves, not merely of their chemical products, as in Diphtheria and Tetanus. The infection of the lymph, blood, and tissues is less rapid, and usually less complete, than in the typical specific fevers. There is a combination of local granuloma growth (the tubercles) with general febrile disturbance (so-called hectic fever), the former due to the bacilli, the latter to the toxines they secrete. In this respect tuberculosis forms one of a series of infective maladies which show gradations from purely general to purely local symptoms: typhus—enterica—syphilis—tubercle—leprosy—lupus.

So remarkable are the affinities of tubercle to specific fevers that Cohnheim predicted that one day phthisis and other tuberculous maladies would be regarded as due to a specific contagium; as shown by the passage at the head of this chapter from his 'Vorlesungen ü. allg. Pathologie' (Bd. ii, S. 709—712). This prediction was fulfilled by Kooh's discovery of the bacillus of tubercle in the following year (1882).

We must not, however, commit the error of ascribing all the symptoms and morbid changes of tuberculous diseases to the direct effect of the microbe or its secretions. Because these diseases are due to a specific infective cause, they are no less local inflammations, and are often complicated by septicæmic infection; so that we shall best consult pathological accuracy as well as practical use by treating in this place of tuberculosis generally, among specific fevers, and leaving the account of its local varieties to the chapters which relate to diseases of the brain, of the lungs, abdomen, and other parts; just as we have dealt with syphilis as a specific disorder among infective fevers, and reserved the account of its local effects to subsequent parts of this book.

Anatomy of tubercle.—The term *tuberculum*, now confined in accurate use to the products of the specific bacillus, was originally applied to any small tumour, as a gumma of the skin, or a large wart, or a granulation-tumour, or a "hobnail" on a cirrhotic liver. The earliest description of tubercles, in the modern sense of the word, is by Bayle in his treatise on Phthisis in 1810. He divided phthisis into different species, only one of which he called "tuberculous." To the second species he gave the name of "Granular Phthisis," and described the lungs as "stuffed with transparent shining granulations, of cartilaginous nature and consistence, never opaque, and without any tendency to soften." Laennec in 1827 described tubercles as "having the appearance of small semi-transparent grains, greyish or colourless, varying from the size of a millet seed to that of a hemp seed. . . . Afterwards they gradually increase in size, and as they do so they become yellowish and opaque, beginning in the centre. . . . Several unite together to form larger masses, pale yellow, ópaque, and of the consistence of very firm cheese. . . . At length they soften and finally liquefy; this change also begins in the centre, and progressively approaches the circumference." The grey semi-transparent granules he termed "miliary tubercles;" to the yellow cheesy masses he applied the unfortunate name of "crude or immature tubercle."

Louis, whom Laennec's premature death from phthisis left the leading pathologist of Paris, limited the application of the word tubercle to the yellow opaque stage, his method of statement being that the grey semi-transparent "granules" "undergo conversion into tuberculous matter." Most subsequent writers passed over the early grey stage altogether;

and, in accordance with the humoral pathology in vogue during the former half of the century, they taught that the yellow material was deposited as such from the blood.

Virchow effected an important reform in pathological doctrine when he showed that a yellow cheesy substance, like "crude tubercle," may be the result of degeneration of many tissues, from ordinary inflammation to gumma, sarcoma, and carcinoma; in other words, that caseation is merely a mode of retrograde metamorphosis, including fatty and calcareous change. Nevertheless Virchow certainly underrated the extent to which cheesy masses in the lungs and in other organs are really of tuberculous origin.

Evidence that grey and yellow tubercles are but stages of the same process is afforded, as Louis long ago pointed out, by the observation that the successive changes described by Laennec often occupy definite positions in a lung: towards the base there are grey semi-transparent tubercles; higher up they are of opaline aspect and yellowish on section; higher still they are opaque, yellow, and fused together. Moreover they become larger from below upwards. Now it is certain that the apex of the lung is almost always the earliest seat of tubercles, and that they gradually spread downwards through the organ, and thus the conclusion seems indisputable that they are first grey and afterwards become yellow.[*]

Miliary tubercles may be of three kinds:

1. *Soft grey granulations*, always of recent formation, "acute," and soon losing their characteristic appearance.

2. *Yellow granulations*, comparatively recent or old, tending to coalesce, soften, and ulcerate.

3. *Hard grey granulations*, chronic, fibrous, pigmented, and liable to no change except pigmentation.

The first are found in acute pulmonary tuberculosis of the lungs, in the pia mater and serous membranes. The second are constant in the lungs of phthisis, occasional in the pia mater, and very frequent in the liver, spleen, testes, and kidneys, as also in the bones and joints, and in the respiratory, intestinal, and urinary mucous membranes. The third are a fibrous instead of a caseous degeneration of the first, and are occasionally met with in cases of chronic phthisis, and in a few other still rarer events.[†]

Histological characters of tubercle.—When the microscope was applied to morbid anatomy the object of pathologists was naturally to discover distinctive characters of tuberculous lesions. Thus Lebert many years ago thought that he had found a specific "*tubercle-corpuscle*" in the yellow cheesy material which was at that time taken for the typical form of the morbid product. When Virchow taught that this material is already in a state of decay and degeneration, the search was actively prosecuted in recent grey tubercles. Virchow himself held that they are histologically a form of new growth, *lymphoma:* according to him, individual tubercles are comparable with the Malpighian bodies of the spleen; and, as he believed that they are apt to arise where no such lymphoid structures are naturally present, he regarded them as "heteroplastic" new growths. Subsequent writers further developed this view by insisting on the existence of a reticulated stroma, such as belongs to other lymphomata. It was believed by some that the true seat of tubercles was the interior of the lymph spaces and lymphatics throughout the body, and that their cells were formed by proliferation of the lymphatic endothelium. On the other hand, Sanderson endeavoured, in 1868, to demonstrate in those parts in which tubercles occur the normal presence of lymphoid tissue, an overgrowth of which might lead to their formation, and thus render them "homœoplastic."

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[*] Tubercles often undergo caseation before they are large enough to be visible with the naked eye. Both in the pia mater and in the liver I have repeatedly found tubercles of microscopic size which were already opaque, not only in the centre, but in the greater part of their substance.—C. H. F.

[†] Tubercles, instead of caseating, often undergo a different change, which leaves them permanently grey. Now it is curious that those pathologists who regarded a yellow cheesy condition as typical of tubercle, spoke of *recently formed* grey tubercles as the "grey granulations of Bayle;" yet a perusal of the four cases of granular phthisis related in detail by that writer shows clearly that in three of them, at any rate, the disease was of a *chronic* kind. Such hardness as he attributes to his granulations is, indeed, inconsistent with their having reached only an early stage; and he also mentions that they are sometimes pigmented or speckled with brilliant black dots—characters of *chronic* tuberculous affections of the lungs and bronchial glands. In fact, the grey granulations of Bayle are really tubercles which, instead of caseating, have undergone a fibroid change, and become

C. H. F.

About the same time the attention of histologists was drawn to another element of tubercle—the giant-cell. That very large cells are sometimes to be found in a tubercle had long been known; Virchow himself described cells containing twelve or more nuclei. Langhans and Schüppel insisted on the frequent presence of such bodies, and applied to them the name *Riesenzellen*, which had previously been invented by Virchow for similar bodies in certain sarcomatous tumours (v. *supra*, p. 84). Dr Hamilton, in 1880, figured and described giant-cells as occurring constantly in tubercles, sometimes placed in the centre of a tubercle, sometimes laterally. When a giant-cell is young it seems to consist of a large mass of granular protoplasm, sometimes with many nuclei in it, sometimes without them. As it grows older the peripheral part (the "periblast") becomes organised, and constitutes a fibrous sheath, in which great numbers of round or oval nuclei may be perceived. Schüppel attributed them to the coalescence of leucocytes in the interior of capillary vessels. Julius Arnold and Klein believe they may be produced by the fusion of epithelial cells, of which the nuclei persist.

The experiments of Ziegler have shown that giant-cells may be formed in ordinary inflammatory exudation. Köster discovered giant-cells in the granulations of diseased joints; and Friedländer found similar bodies constantly present in lupus and in "scrofulous" ulcers. These tissues were no doubt tuberculous; but giant-cells have been found in a shallow ulcer of the uterus, the stroma of a cancerous tumour, the floor of a phagedænic chancre, and bands of adhesion in the pleura or peritoneum.

According to the best modern histologists, grey tubercles (*granulations grises* of Bayle) may be made up of small nucleated cells, like leucocytes, or of large more or less flattened nucleated cells like epithelium, with giant-cells in either case among the others. The delicate intercellular reticulum, which Virchow described and compared to that of a lymph-follicle, is denied by many good observers. Most German pathologists follow Baumgarten in believing the origin of the tubercles to be in the epithelial lining of the alveoli of the lung or the secreting epithelium of the glands. Metschnikoff, however, believes them to begin in a collection of large leucocytes (phagocytes), which are attracted to the invading bacilli: they often swallow and destroy them; but if unsuccessful, form a mass increased by the proliferation of connective-tissue corpuscles and endothelial plates from the capillaries.

Caseous degeneration follows death of the constituent cells of the tubercle. Calcareous matter, *i. e.* phosphates of calcium and magnesia, with the corresponding earthy carbonates, is afterwards deposited very slowly. Like fibrous induration, it is a retrogressive change.

Tubercle and inflammatory exudation.—It is now generally admitted that, apart from the existing microbe, there is nothing specific in the histology of tubercle. A tubercle is an inflammatory granuloma. Niemeyer's denial that tubercles are an essential feature of pulmonary phthisis was based on the microscopical observation that in many specimens of phthisical lungs the morbid process which precedes the occurrence of caseation is not a development of lymphoid tissue, but a filling of the alveoli with epithelial cells, or in other words, a 'catarrhal pneumonia.' He accordingly declared that a chronic catarrhal pneumonia, ending in cheesy infiltration, really constitutes the primary anatomical lesion in many cases of phthisis. Hamilton also asserts that dry yellow nodules, such as were described in the lungs by Laennec, are generally nothing but patches of catarrhal pneumonia; bodies having an aspect similar to that of a tubercle, but as large as millet seeds, usually (he says) prove to be groups of pneumonic air-vesicles. The late Dr Wilson Fox maintained that, in acute tuberculosis, granulations 'composed of epithelial proliferation' are very common. And Hering went so far as to declare that the ordinary fatal miliary tuberculosis of the lungs ought to be called acute disseminated catarrhal pneumonia. Moreover, as was shown by Julius Arnold, precisely similar epithelial changes to those in the lungs occur in the liver, the kidneys, and the testes, when affected by acute tuberculosis.

Ziegler argued that a tubercle owes its rounded form and its definite size to the fact that it is produced by the circumscribed action of an irritant of but slight intensity upon the spot which afterwards becomes its centre; and he contrasts the effect of such an irritant with the more rapid operation of a septic particle, such as gives rise to a miliary abscess.

Koch's discovery of a tubercle bacillus supplied the "irritant of but slight intensity" that is needed for this theory. The slow growth of this organism, to which Koch himself draws attention, may perhaps account for the fact that the morbid process which results from its action is so much less acute than infective inflammations in general.*

Caseation of tubercle.—We have seen that grey granulations tend to caseate. Most pathologists since Virchow have been too ready to assume that caseation is of frequent occurrence in ordinary inflammation. No doubt the pus contained in a serous sac or in a large abscess may, and often does, dry up into a cheesy mass. The same thing is observed in the crypts of the tonsils, in the cæcal appendix, and in other pouches of mucous membrane, and it is seen in the walls of arteries when they become atheromatous. But in each of these instances the inflammatory exudation which caseates is more or less beyond the range of the blood-vessels. Does common inflammation ever lead to caseation in the substance of a vascular solid organ—in the liver, or in the spleen, or in a lymph-gland?

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* An affection (whether of the lung or of any other organ) may fail to present the characteristic granulations, and yet be really tuberculous. Laennec long ago described what he termed a tuberculous "infiltration of the lungs," when considerable portions of the pulmonary tissue become solidified by a greyish and semi-transparent, or by a paler and yellowish-white material; either without any previous development of distinct tubercles, or around tubercles already formed. Subsequent observers have very generally declared such lesions to be 'pneumonic.' But in their naked-eye characters these 'infiltrations' differ altogether from anything that is seen in ordinary pneumonia. Even when the growing edge of a patch of pinkish-grey infiltration appears quite homogeneous, one often finds that the less recent parts of it, where caseation is commencing, show yellow granulations corresponding exactly with tubercles in size and in general appearance. Again, in association with acute tuberculosis of other organs, an affection of the membranes of the brain is not infrequent, which seems to be a simple inflammation, since no tubercles are discoverable. In such cases the presence of recently formed tubercles elsewhere is strong evidence that the meningitis must be essentially of the same nature.—C. H. F.

One of the most typical features of tuberculosis is the presence of spreading ulcers in cavities, with yellow walls of definite thickness, the substance of which regularly undergoes caseation almost as soon as it is formed. This process, wherever found, whether as a pulmonary vomica, or in the liver, the kidney, the prostate, or the testicle, may be safely set down as tuberculosis.

The tendency to caseation in tubercles is probably due to the absence or scanty supply of blood-vessels.* Every museum contains injected specimens of phthisical lungs, from which it appears that not only isolated tubercles, but also patches of caseous infiltration, are non-vascular. No new vessels seem to be formed in them, and the pre-existing vessels undergo obliteration.

Fibrous transformation.—In certain cases, as already mentioned, tubercles, instead of caseating, undergo fibrous transformation. What determines them to take the one course rather than the other is uncertain; but it would seem that caseation is less frequent as the patient is older. In organs other than the lungs little is known about "fibroid" tubercles. But Schüppel has demonstrated their occurrence in lymph-glands, and has shown that the reticulated stroma grows at the expense of the cells, and is converted into a transparent, almost homogeneous, indistinctly fibrillated material; and Rindfleisch has recorded a remarkable specimen of fibrous tubercles in the great omentum. A similar change is, probably, the origin of what has been called "fibroid phthisis." Rindfleisch drew attention to the impossibility of forcing injection into indurated masses in the lungs, and contrasts their state with the abundant supply of vessels to the newly formed connective tissue in cirrhosis of the liver and in interstitial nephritis. Hamilton, however, believes that many of the so-called cirrhoses of organs, both in children and in adults, are in reality the remains of tubercle.

Experiments on the artificial production of tubercle.—In 1865 a French observer, M. Villemin, made known the fact that in certain animals, particularly rabbits and guinea-pigs, tuberculosis could be set up by the inoculation of tuberculous matter from the human subject. His experiments were soon repeated in this country by Sir John Simon, Dr J. B. Sanderson, Dr Wilson Fox, and others. Tubercles from the pia mater or from serous membranes, caseous tubercle from the lungs of phthisical patients, or the sputa of such patients during life, were introduced beneath the skin of animals. These were afterwards killed, or were allowed to die of the disease which followed the operation, and generally proved fatal in from six to ten weeks. It was then found that pus or a dry cheesy substance had been formed at the seat of inoculation, and that bands of induration extended away from it into the adjacent subcutaneous tissue. The lymph-glands in the neighbourhood were enlarged and caseous. Nodules of various sizes were present in the lungs, the liver, the spleen, and the peritoneum. These resembled tubercles both to the naked eye and in their histology. They consisted of a grey, semi-transparent material, which underwent caseation from the centre outwards. The infective character of the process was further shown by the fact that matter taken from the body of an animal after death was capable of setting up the same disease in other animals.

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* Virchow's comparison between tubercles and the Malpighian bodies of the spleen was objected to, and with justice, on the ground that the latter contain capillaries.

After a time, however, certain observations were made which seemed to contradict the obvious conclusion from these experiments. Dr Sanderson found that in guinea-pigs an "artificial tuberculosis" arose after the inoculation of pus from non-tuberculous abscesses. Dr Wilson Fox independently arrived at a like result by inoculating guinea-pigs with pieces of putrid muscle, or with vaccine lymph. In Germany, Cohnheim and Fränkel introduced into the peritoneal cavity of rodents portions of new growths, or of healthy organs from the human body, or pieces of charpie or gutta percha; and they succeeded in setting up a tubercular process not only in the serous cavity itself, but also in the lungs and in the liver.*

These experiments, however, were vitiated either by the accidental presence of tubercle when the operation was performed, or by the wound becoming infected with it afterwards. Cohnheim frankly acknowledged that, having repeated his experiments both at Kiel and at Breslau, he had utterly failed to obtain the same results as before; and he became one of the warmest supporters of the specific nature of tubercle.

At a meeting of the Pathological Society on December 4th, 1883, Dr Fox stated that at his request the experiment referred to in the text had been recently repeated by Dr Dawson Williams. The details of this series of experiments were stated, with the precautions against accidental infection; and the results were absolutely negative.

Cohnheim made a further advance, by showing that in rabbits and in guinea-pigs tuberculosis has a tolerably definite period of incubation—from fourteen to twenty-one days. When a minute fragment of tuberculous matter is introduced into the anterior chamber of a white rabbit's eye through an incision in the cornea, the slight reaction which arises after the operation quickly subsides, provided that the tubercle is perfectly fresh. The fragment can now be seen through the transparent cornea, and it diminishes in size day by day until it may altogether vanish. Then, at the end of a period of two or three weeks an eruption of small transparent grey granulations appears on the iris. Afterwards they caseate, and a destructive inflammation of the whole eyeball often results.

The tubercle bacillus.—In the 'Berliner klinische Wochenschrift' for April 10th, 1882, Dr Koch made known the important fact that he had discovered bacilli in the tubercular diseases of man and of animals, that he had succeeded in cultivating these bacilli upon the coagulated serum of blood, and had been able to set up tuberculosis in healthy animals by inoculating the cultivated products. His statements have since been abundantly confirmed.

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* These observations fitted in with a theory that in the human subject acute miliary tuberculosis is due to the absorption of caseous matters from yellow tubercles. This opinion had been expressed by Dittrich of Erlangen, and by Buhl of Munich. In ordinary phthisis it was thought that a catarrhal pneumonia, with caseation, was the earliest change, and that the disease only became tubercular by a secondary infective process. This was in entire accordance with the now forgotten doctrine of Niemeyer, that phthisis is a primary pneumonia with but little tuberculous about it.

The rod-shaped microbes are in length equal to a quarter or half the diameter of a red blood-disc (2—4 μ), and in breadth one fifth to one sixth of their length. They are slightly curved and have rounded ends. When prepared by Koch's original double staining, methylene blue and vesuvin, the bacilli are blue, while nuclei appear brown.*

The bacilli are readily detected in the tissues or in pus or mucus. They have often a beaded appearance, due to vacuoles in their protoplasm, and not, as was long supposed, to the formation of spores. The tubercle bacillus, like those of enteric fever and diphtheria, has a spurious analogue in the *Bacillus smegmatis;* but fortunately this does not occur in the lungs.

When grown upon the coagulum of blood-serum the bacilli form flat, scale-like masses, hardly as large as poppy seeds, which can be lifted off entire, and are so firm that it requires some force to break them up. Their development takes place very slowly, about ten days elapsing after infection of the coagulum before any change is discoverable. Another condition necessary to their growth is a temperature of 28°—42° C. (86°—106° F.). Hence they cannot grow in the open air in this climate, and, so far as is known, do not exist out of the living body of man or animals. They are killed by direct sunlight, they are aërobic, and purely parasitic (not saprophytes). They do not exhibit spontaneous movement. They are not numerous, except when the tuberculous process is recent and active; if it is slow they are often to be found only within the giant-cells.

In the twenty-first volume of the 'St Bartholomew's Hospital Reports' Dr Vincent Harris states that in twelve ancient specimens of tuberculous lungs, from forty to seventy years old, he succeeded in demonstrating the presence of Koch's bacillus microscopically.

The bacillus of Koch answers every test of a specific pathogenic organism (p. 18). It is itself identified by the microscope, by staining, and by cultivation. The disease is identified during life and after death by its well-known morbid anatomy. The bacillus is present in every case of the disease, and is not known to occur elsewhere. Lastly, when a pure cultivation is inoculated into an animal it multiplies in its tissues and reproduces the lesions of the original disease.

The clinical products of the bacillus to which the fever and other toxic effects are probably due are little known. Koch's "tuberculin," when injected, causes fever in men or animals previously affected with tuberculous diseases, and not in those that are free from them. It also produces vascularity of a tuberculous lesion of the skin. It is, no doubt, a mixed substance.

Transference of contagion.—The most frequent mode of contagion is by sputa containing the tubercle bacillus, which dry up, are blown about, and so enter the lungs in breathing. Milk from cows with tuberculous disease of the udder is a cause of tabes mesenterica in children. The skin may be infected by direct contact with tubercle, as in making post-mortem examinations when the hands become the seat of tuberculous warts (*verruca necrogenica*), which, however, do not ulcerate, and never become the source of tubercle elsewhere. Possibly lupus is also caused by direct contact with the skin. The occurrence of tuberculous disease of the bones and joints, the kidneys and testes, the brain and choroid, can only be explained by assuming special local proclivities or lack of resisting power in the organs affected, when the bacilli are already widely distributed over the whole body.

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* Ehrlich has since devised a better and more rapid method by which the bacilli appear magenta-red with fuchsin (hydrochlorate of rosanilin), while the tissues are counter-stained with methylene blue. Gramm's method, gentian violet for the tubercle bacilli, and eosin or vesuvin for counter-staining the tissues, is another double-staining used.

The disease of cattle known in Germany as *Perlsucht*, and in England as "the grapes,"[*] is now proved to be tuberculous. It is a chronic affection, which may run on for years without symptoms. Animals thus affected are often sold to the butcher, so that besides the risk of drinking their milk during the early part of their illness, there is the further risk of eating the diseased tissues in uncooked sausages. The frequency of Perlsucht may be judged from the fact that at Augsberg 2 per cent. of all cattle slaughtered were proved to be tuberculous, and of cows as many as 5 per cent. In Paris bovine tuberculosis has become less common of late years. Pigs, calves, rabbits, and guinea-pigs have been successfully infected by the milk from diseased cows, and a like result has followed feeding animals with portions of the affected tissues removed after the cows were slaughtered. One is strongly tempted to find in these experiments an explanation of the frequency of mesenteric disease in children, in whom milk forms so important an article of diet.

Another set of experiments was made by Tappeiner, in order to determine whether tuberculosis could be induced in dogs by the inhalation of the sputa of phthisical patients. His method was to employ dried sputa diffused in the air as a spray. The earliest period at which tubercles were found in the lungs after the inhalations were begun was on the twenty-third day. In no single instance were the results negative.

Carnivora are far less liable to tuberculosis and far more refractory to its bacillus than are rodents; horses are more refractory than cattle, and goats than sheep. Bollinger supposes that flesh-eating animals secrete a gastric juice which has a more active power of destroying the bacillus.

The origin of tuberculosis in man.—It appeared at first difficult to apply these experimental facts to explain the origin and transference of tubercle in man. For it is a rare exception for phthisis to spread from patients to other patients, or to near relatives, nurses, or physicians. Moreover its prevalence is greatly augmented by overcrowding and defective ventilation, by exposure to cold and wet, and by dampness of soil. Again, it seems that phthisis may be caused in some cases by inhalation of ordinary dust. Lastly, hereditary transmission plays an important part in the ætiology of tubercle.

It will, however, be shown in the chapter on Phthisis that cases of direct transmission to a fellow-patient in hospital or from a husband to a wife are not so rare as was once supposed: also that the contagion being not conveyed by the skin or breath, but only by dried sputa, the chances of direct infection are almost as small as we saw them to be in cases of enteric fever or cholera. Again, broncho-pneumonia, by making a breach in the protective epithelium of the bronchioles and air-vesicles, no doubt prepares the way for the entrance of contagion. And we have already seen in other specific fevers illustrations of the law that disease is the result partly of external influences, partly of the reaction to them of the living organism.

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[*] Dr Creighton has recorded several cases in which he believes that *post-mortem* appearances found in the human subject justify the conclusion that the disease was derived from the cow. Koch found his bacillus in ten cases of Perlsucht, chiefly in the interior of giant-cells surrounding the calcified nodules in the lungs, but also in the bronchial and even in the mesenteric glands.

The seed is barren if it falls on an unsuitable soil, and the healthy human body appears to be able to defend itself by chemotaxis or otherwise against the invasion of tubercle. There are probably great differences in the resistance of various persons to the bacillus. But this resistance must be far greater in the average human subject than in the rabbit or in the guinea-pig, though less than in the horse. Probably it varies in the same individual at different periods, and is not absent even among those in whose families phthisis is hereditary.

Apparently the tubercle bacillus is so widely diffused that it is continually finding access to the body, but fails to implant itself until the local resistance of the tissues happens to be lowered by local inflammation, or by some more general cause.

Cohnheim thought that the bacillus may remain latent in the body for an indefinite time until some accident calls it into active growth. He imagined that the hereditary transmission of phthisis means the direct transference of the virus, either in the spermatozoa or in the ovum, as in the case of syphilis or the *pébrine* of silkworms. This, however, seems improbable; for congenital tubercle, though not to be denied, is extremely rare. What is transmitted is not tuberculosis, which seldom appears before puberty, but a vulnerability or want of power of resistance to the invasion of a contagion which is probably as common and diffused as that of measles or mumps.

The spread of acute tuberculosis in the body.—Acute general miliary tuberculosis, with its myriads of minute centres, is explained by the dissemination throughout the blood-stream of the tuberculous bacilli from the original focus of infection.

But it must not be supposed that the growth and the distribution of tubercles are regulated solely by the way in which the bacilli are disseminated through the blood-stream. There seems to be a curious difference between the artificial tuberculosis of animals and the human disease with respect to the distribution of the tubercles. In the former they are more abundant in the pleura than in the substance of the lung, and have sometimes the form of blunt cones. In both respects artificial tuberculosis resembles the two other great examples of infection conveyed from a primary focus to different organs by the blood—pyæmia and sarcoma—as well as in the abundance of secondary lesions in the liver.

In the general miliary tuberculosis of man the liver is much less frequently and less obviously affected than the lung, although no doubt the microscope shows that hepatic tubercles are far more often present than used to be supposed. In the lungs the tubercles are not more numerous beneath the pleural surface than elsewhere, and they do not affect a conical or wedge-like form.

Moreover in most cases of acute tuberculosis the distribution of tubercles in the lung shows that it is determined by the local peculiarities of the organ. In the upper lobe the tubercles are more abundant, larger, and more advanced in their development. They often gradually diminish both in number and in size from the apex downwards, and while caseous above, may be grey and semi-transparent below. Thus, even when the blood-stream carries the bacilli to all parts of the lungs at once, the distribution of the tubercles is modified by local conditions. In tuberculous meningitis, again, it often happens that not a single tubercle exists anywhere except in the cerebro-spinal membranes; and yet bacilli in immense numbers must have been distributed throughout the body.

The distribution of chronic tuberculosis.—In chronic tuberculous affections a frequent mode of spreading is by gradual invasion of the lymph channels. Lines of progressive thickening and caseation run in the course of the subperitoneal lymphatics from the floors of intestinal ulcers. Dr Fagge recorded two or three cases in children, and one in an adult, of a continuous caseous mass, with a festooned growing border, extending from the bronchial glands directly through the lung. A still more striking case was met with by Buhl. A child had caries, with caseation, of the lower dorsal vertebræ; the pleura became adherent, and the ulcerative process spread continuously into the base of the left lung. Grancher recorded a case of tubercular peritonitis in which tubercles penetrated the diaphragm, and infected the visceral pleura, without adhesion having taken place.

But in most instances of chronic tuberculosis the special proclivities of the several organs and tissues play a most important part in determining and in limiting the distribution of the tubercles. For when the bacilli have once entered the blood-current, the resistance of certain tissues must be the reason why tubercles are not scattered irregularly through all parts of the body. Instead of this, the fact is, as Dr Fagge pointed out in the 'Pathological Transactions' for 1874, that several distinct varieties of chronic tuberculosis can be recognised, each of which has peculiarities of its own, according to the organs and tissues attacked.

The following are the chief local varieties of chronic tuberculosis arranged in groups:

1. Pulmonary phthisis, accompanied by tuberculosis and ulceration of the larynx, and of the intestine (especially the lower end of the ileum). It may be that the disease of the lungs directly causes the other lesions, the larynx being infected by the sputa which pass over it, and the ileum by the sputa which are swallowed. But in enteric fever likewise, ulceration of the lower part of the ileum occurs, together with ulceration over the bases of the arytænoid cartilages.

2. Wilks long ago remarked that tubercle sometimes attacks the pleura, the peritoneum, and the pericardium, without affecting any of the solid viscera. The same common fate of the pleuro-peritoneal cavity is seen in Bright's disease, and in some idiopathic cases of serous inflammation.

3. Tuberculous disease which began in one kidney not only spreads by continuity along the genito-urinary mucous membrane and the corresponding submucuous tissue, but is frequently attended with a like affection of the opposite kidney, and of one or both testes. The relation between tuberculous disease of the kidney and Pott's disease of the lumbar vertebræ at the same level is probably that of direct extension.

4. Addison's disease of the adrenals (the tuberculous nature of which is well ascertained) is sometimes associated with spinal caries at the same level, to which the adrenal lesion is probably secondary.

5. Tuberculous disease of the lymph-glands often spreads widely

groups of glands widely distant from one another. It is also frequently combined with tuberculosis of the spleen. A precisely similar association is seen in Hodgkin's disease.

6. Several of the joints are often affected by tuberculous disease in succession, without any corresponding disease of other structures, either the synovial membrane (pulpy disease or white swelling) or the heads of the bones (caries). Here, again, one is reminded of the frequent limitation of pyæmia to joints—another illustration of the effect of local peculiarities in determining the distribution of lesions, even when they are due to infection of the blood.

7. The bones are a frequent seat of tubercle in children. It causes caries and suppuration, and frequently runs its course and is cured without any of the internal organs being complicated.

Other instances of local tuberculosis are furnished by the occasional appearance of meningitis without other organs than the brain being affected, and by lupus or deep tuberculous dermatitis, which is rarely associated with disease of the lymph-glands, the lungs, or any other organs.

Summary.—Tuberculosis is a specific general infective disease, derived as a rule, but perhaps not invariably, from a caseous focus. The secondary growths are of the kind described as granuloma (p. 60), and constantly contain a specific bacillus, the contagium vivum to which the morbid process is due. But besides the granuloma-growth there is also ordinary secondary inflammation, purulent, ulcerative, or fibroid, which in most cases follows as a complication. The symptoms of tuberculosis are partly due to the bacteria themselves, partly to the toxines they secrete, and partly, in many cases, to a concomitant purulent affection due to micrococci.

The vehicle of contagion is most commonly dried sputa, occasionally, perhaps, the breath, or the mucus accidentally ejected in coughing, and not infrequently milk from tuberculous cows. Although there is no protection, one attack rather predisposing than indisposing to another, yet the tissues of many, perhaps the majority of human beings appear to be naturally immune to the tubercle bacillus. This immunity may at any age be diminished until infection becomes easy; or, on the other hand, it may be increased until infection becomes almost impossible.

The clinical course of tuberculosis is exceedingly protracted and irregular, more so than that of syphilis. It has no exanthem, and is always accompanied with pyrexia, which resembles the fever of Enterica and of Septicæmia more than any other.

TETANUS

> "Extentat nervos, torquetur, anhelit
> Inconstanter, et in jactando membra fatigat."
> LUCRETIUS, iii, 489.

History and pathology—The bacillus—Clinical symptoms—Varieties—Idiopathic tetanus—Diagnosis—Mode of death—Recovery—Prognosis—Treatment by drugs and by antitoxic serum.

Nomenclature.—Τέτανος (a stretching, strain, tension, from τείνω), used by Hippocrates and other classical writers—Rigor nervorum (Celsus)—Lockjaw.—*Fr.* Tétanos.—*Germ.* Starrkrampf.

Definition.—A specific disease, depending on the secretions of an infective microbe, affecting the spinal cord and producing tonic spasms of great severity.

History.—This terrible disease, dreaded at once from the pain it produces and from its fatality, has been known from the earliest historical times. The symptoms are severe tonic spasms or general convulsions, and are referable throughout to the spinal system. Since with few and doubtful exceptions it follows a wound, it has been more studied by surgeons than physicians. A striking picture of a case of acute traumatic tetanus given by Morgan in 1833 is quoted in the article in Holmes' 'System of Surgery' by the late Mr Poland. The immediate cause of the symptoms is obviously in the nervous system, and since the discoveries of Johannes Müller and Marshall Hall, no less obviously in the reflex action of the grey matter of the cord, from the origin of the fifth cranial nerve to the lowest spinal centre.

Since by far the most frequent and unmistakable cases are certainly traumatic in origin, it was natural to suppose that some foreign body acted as an irritant, and striking cases were published of fragments of glass, straw, or clothing being found in the wound, and even of recovery after they had been removed. It was often observed also that the wound was fouled with earth. Another hypothesis was that in some unexplained way the activity of the spinal centres was exalted just as they are by strychnia; indeed, the likeness between cases of poisoning by this drug and those of tetanus was obvious to anyone who had seen or read of both. When the microscope was applied to morbid anatomy Lockhart Clarke in 1865 ('Med.-Chir. Tr.,' xliv, p. 225), and many others down to Dr Dickinson ('Med.-Chir. Tr.,' liv) and Dr Coats, described evidences of inflammation in the cord of hæmorrhage or of dilatation of the perivascular sheaths with extravasation of leucocytes. The view taken by Clarke was that there was a *neuritis migrans* ascending from the wound to the spinal centres. Lastly, the opinion was expressed by Mr Poland in the above-mentioned article, by Sir James Simpson, and by many others, that tetanus is due to a toxic influence on the cord of a poison acting like strychnine, but of animal origin, and generated in the wound.

This hypothesis is now proved. Tetanus, which had before taken its place according to its symptoms among spasmodic diseases of the nervous system, now ranks among the specific infectious diseases.

The contagion.—As we saw in the case in diphtheria, the effects of tetanus are not due directly to multiplication of the infective microbes throughout the body, and probably none ever reach the spinal cord: they are due to the chemical poisons secreted by a specific microphyte.

Nicolaier in 1885 found that when garden mould was inserted under the skin of a rabbit an abscess was produced, the pus of which injected into other rabbits produced tetanic convulsions; and the same effect was obtained in the following year by Rosenbach with pus from a patient with tetanus. Both these observers described a very small slender bacillus as probably the specific microbe; but it was only in 1889 that a pure culture of this was obtained by Kitasato, a Japanese studying in Koch's laboratory at Berlin, and also by Tizzoni and Cattani in Italy.

It is a very minute organism, occurring in rods $4-5\,\mu$ long, and in slender filaments which form spores. As they develop, a swelling at one end of the rod produces a "pin-head" or "drumstick" form. The spores are very resistent to temperatures at which the bacillus is killed. It cannot grow in the presence of oxygen or in a bright light.

This bacillus has been found in ploughed fields, beneath the dust of the streets, in horse dung, and elsewhere protected from light and air. It has also been found in pus from cases of tetanus. It is separated in cultivation from other saprophytic organisms by its spores resisting heat and deprivation of oxygen. It lives, therefore, out of the body as well as in it, or, to adopt the current phrase, it is a "facultative" (*i. e.* occasional) parasite. The colony of bacilli and spores does not travel away from the seat of inoculation, and the secretion of poisonous chemical products goes on very slowly. If the bacillus alone infects the wound there is no local suppuration; and if this occurs, it is due to the presence of other pyogenic micrococci. As Dr Woodhead, from whom these facts are taken, remarks, it is unfortunate that the infected wound has no character to mark it like the faucial membrane of diphtheria, and so it is often not recognised as dengerous until the whole body is pervaded by the poisonous secretion of the bacillus.

Symptoms.—After an incubation of about ten days, the disease begins with stiffness of certain muscles, which is often first discovered when the patient wakes up in the morning. Commonly those first affected are the masseters, causing *trismus*, or "lockjaw." Sometimes the earliest symptom is a stiff neck; or a peculiar grin on the face. A little girl who some years ago died in Guy's Hospital was found fault with by her mother for making faces; but soon afterwards the poor child was nearly choked by spasms in attempting to swallow food.

The initial wound is often a suppurating one, but is frequently not severe enough to require surgical treatment, and sometimes is so slight as to be overlooked. Occasionally before the tetanus sets in darting pains are complained of in the injured part, which may shoot up the limb; there are four instances of this among seventy-two cases which were collected from our case-books for the 'Guy's Hospital Reports' in 1857 by the late Mr Poland. As a rule there are no premonitory symptoms whatever. The locality of the earliest spasm is not usually determined in man, as in experimental tetanus, by its proximity to the seat of infection.

The tonic spasms soon increase and spread to the muscles of the trunk and limbs. The face acquires an aged look, the forehead is wrinkled, and the features drawn. The angles of the mouth are wide apart, and the lips are stretched over the closed teeth, so as to produce a fixed smile—the *risus sardonicus*.* The naso-labial furrows are exaggerated. The eyelids are half closed, and are seldom affected by the cramp. The jaws are sometimes clenched so firmly that not even a paper-knife can be wedged in between the teeth. The trunk is rigid, and it is almost always curved, so that the back forms a deep hollow, while the occiput is buried in the pillow, and the throat stretched upwards. Occasionally a violent spasm of the dorsal and gluteal muscles will throw the patient's body into an arch, supported upon the head and the heels. This condition is called *opisthotonos*. The chest is thrown forwards, and is more or less fixed in a state of expiration, while the abdomen is flat or hollow. The tension of the affected muscles is obvious to the touch and sight; this is particularly the case with the recti abdominis, which feel "as hard as boards," and stand out in knotty masses. Sometimes their fibres give way, and blood is extravasated so as to form a palpable swelling. The limbs are commonly extended, and there may be a marked stiffness of the shoulders and hips, and less often of the elbows and knees. There is seldom impairment of the movements of the hands or fingers, but the writer has seen them the seat of marked tonic spasm.

From an early period of the disease there is pain in the muscles like that of cramp. There may also be a distressing sense of oppression from embarrassment of the breathing, and the same cause may produce aphonia. The patient is frequently unable to micturate in consequence of the rigidity of his abdominal muscles.

According to Rose, of Zürich, in his article in Pitha and Billroth's 'Hdbuch. d. Chirurgie,' many cases of tetanus end fatally with the symptoms above described; but as a rule the spasms undergo aggravation at longer or shorter intervals. During these paroxysms, which last from a few seconds to three or four minutes, the aspect of the patient becomes frightful from the distortion of the features and the opisthotonos. The tongue is often caught between the teeth and severely bitten, and the face and the hands become livid from cyanosis. The paroxysms sometimes arise spontaneously; sometimes they are obviously reflex, being brought on by a touch, a draught of cold air, a sudden noise, or some voluntary effort. They are generally attended with great increase in the pain, often by extreme anguish. Sir Gilbert Blane, however, met with a case in which, although it ended fatally, there was merely a tingling sensation.

Sleep is generally impossible from an early period, but Watson relates how, when a patient fell asleep, the spasms ceased for the time; even the abdominal muscles became perfectly soft and yielding, but instantly resumed their contracted state as soon as he awoke. The mind is clear and unclouded.

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* *I. e.*, as usually interpreted, the Sardinian laugh; from the classical tradition of an herb growing in Sardinia (Σαρδώ) which produced involuntary facial spasms. Cicero has *ridere γέλωτα σαρδάνιον*; and this form, used by all Greek writers from Homer to Plato and afterwards, makes the above derivation doubtful.

The pulse is at first natural, but towards the last it becomes very rapid, perhaps 160 or 200 in the minute.

It was long a disputed question whether tetanus is attended with fever. Recent observations have shown that the temperature may be normal throughout the whole course of even the most acute and severe cases; while in those which are comparatively slow in their progress the thermometer occasionally indicates 102° or 103°. Before death hyperpyrexia sometimes rapidly develops itself, temperatures of 110° or 112° being registered. This is not inflammatory fever, but due to disturbance of a central regulating machinery, as in the case of fractured spine, of cerebral hæmorrhage, or of the *status epilepticus*. Tetanus is among the few diseases in which the temperature has been observed to rise one or two degrees after death.

The skin is often bathed in sweat, and an eruption of sudamina is not infrequently present. Dr Wilks has recorded in the 'Guy's Hospital Reports' for 1872 an instance in which the perspiration from the forehead gave to white linen a reddish stain, which, however, Dr Stevenson found not to be due to the presence of blood.

The following is a case lately seen by the writer in consultation with Dr David Syme:

A well-grown, healthy youth of seventeen cut his hand in a fall from his bicycle on July 2nd, 1899. He washed the wound from dirt, but there remained a black stain, even after the wound healed. On the 12th, the cut being apparently well and almost forgotten, he found his jaw stiff. The stiffness increased, and three days later painful spasms began in the back, and afterwards less severely in the neck; he then had to go to bed. When seen on the 19th he had suffered three very bad nights, sleep being prevented by frequent spasm of the erector muscles of the back. The least touch on the flank or pressure over the spine brought on fresh contractions, which lasted a very short time, never more than ten or fifteen seconds. He could not open his mouth more than enough to take liquid food, and had no power of mastication. There was no trismus nor *risus sardonicus*, nor were the limbs affected at this time. The mind was perfectly clear; the pupils normal; the knee-jerks and plantar reflexes brisk, but not unusually so; there was no ankle-clonus, and the functions of the rectum and bladder were intact. There was no loss of sensation or of motion. The symptoms were clearly due, not to concussion or other injury to the spine or cord, but to poisoning by the toxines of the tetanus microbe. The only pain beside that of the spasms was due to the tongue having been twice bitten by convulsive closure of the jaws. The prognosis appeared to be favourable from the gradual advent, moderate severity, and restricted extent of the spasms.

July 19th.—Fifteen drops of laudanum and the same of tincture of cannabis indica were ordered instead of bromide, which was the drug previously given, and some antitoxic serum was procured from the Jenner Institute of Preventive Medicine, of which 10 c.c. were injected on its arrival the following day, July 20th, and a second dose at night.

"July 21st.—He had a much better night after the draught on the 19th and 20th, and to-day can open his mouth a little more. The back is constantly arched and rigid, but there are no violent spasms; the legs are stiff, and he has bitten his tongue again; the right hand (the one injured) is stiff, and the grasp weak. The *risus sardonicus* has appeared. A papular, irritable, erythematous rash occupies the flanks and thighs. No pyrexia. Highest temp. 101° F. on the 16th. Takes liquid food well, and feels more comfortable." Another injection of the antitoxic serum was given, and the laudanum and Indian hemp repeated at bedtime. The rash was no doubt due to the antitoxic serum.

During the next two days the erythema, to which urticarial wheals were added, spread over the trunk and limbs, and became very troublesome, interfering with the patient's sleep and exciting spasms when he tried to scratch the skin. After this subsided with desquamation, the temperature, which had risen to 101° and 101·5°, fell again to normal, and he slept so well that the draught at night was omitted. No more antitoxic serum was injected, but 15 grains of chloral hydrate were given twice or thrice in the twenty-four hours when the spasms were troublesome.

July 26th.—Spasms much less severe. Sleeps well.

July 30th, the nineteeth day of the illness, and the twenty-ninth since the patient had the injury.—Dr. Syme reports uninterrupted improvement, and considers the patient convalescent.

On the 7th of August he left home for the sea-side.

Varieties.—Cases sometimes deviate from the ordinary type. In ancient times several kinds of tetanus were distinguished. If the body was arched forwards instead of backwards, so that the head and the knees met in front of the chest, this was called *emprosthotonos.** In another variety, for which the name *pleurotonos* has been invented, the curve is on one side. But in comparison with opisthotonos, both are exceedingly infrequent, and it is absurd to make separate species of them. Indeed, Rose maintained that they are really never seen in this disease, although they may occur in hysteria.† In 1870, however, a woman aged forty died in Guy's Hospital of tetanus, in whom it is said that "the anterior muscles were mainly affected, so that there was a condition of emprosthotonos."

There is one aberrant variety, in which the earliest symptom is muscular spasm of the part originally injured, and in which the paroxysms, when they set in, affect those muscles far more than others. Such a case was recorded by the late Mr Key in the third volume of the 'Guy's Hospital Reports,' and Dr Fagge afterwards saw a well-marked instance of the same kind. The same fact was noted by the present writer in the case related above. It is frequent when the disease is produced in animals.

An occasional complication of tetanus, to which Rose has drawn attention, is facial paralysis. In 1871 a woman died under Mr Poland in Guy's Hospital, in whom this symptom was present on the left side, and whose ocular muscles were also affected in a strange way, the left eye being immoveable, turned upwards and outwards, while the right one was as rigidly set straight forwards. Neither meningitis nor any lesion of the brain was discovered at the autopsy. The original accident was a fracture of the orbital plate of the frontal bone, caused by the point of an umbrella. This corresponds with a statement of Rose's, that the starting-point of the tetanus in such cases is always within the distribution of the facial nerve; he supposes that the trunk of the nerve becomes swollen, and is compressed within the bony canal through which it has to pass.

Other writers have described similar cases as Tetanus limited to the head (*Kopftetanus*). The wound is also on the head, and beside spasm there is paralysis. Sir George Humphry narrated four cases ('Allbutt's System,' vol. i, p. 780), two of which ended favourably. The question is whether these are varieties of tetanus or some other spasmodic disease.

Ætiology.—Some of the supposed causes are imaginary, others are only accessory to the essential infection by the bacillus and its secretions. It was once erroneously supposed that tetanus was particularly likely to follow wounds of the thumb, or suppurating or lacerated wounds, or an injury to a nerve, or the presence in the wound of foreign bodies.* The really necessary condition is that the wound should be infected with the bacillus by earth or horse-dung; hence tetanus is most apt to follow wounds of the hands and feet, falls from horses, and grazing of the skin on the ground. Hence also it is less rare among gardeners, stablemen, and farm labourers than among artisans.

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* "Neque tamen alius importunior acutiorque morbus est quam is, qui quodam rigore nervorum, modo caput scapulis, modo mentum pectori annectit, modo rectam et immobilem cervicem intendit. Priorem Græci *ὀπισθότονον*, insequentem *ἐμπροσθότονον*, ultimum *τέτανον* appellant: quamvis minus subtiliter quidam indiscretis his nominibus utuntur" ('Celsus,' lib. iv, cap. iii; also 'Cælius Aurelianus de Acut. Morb.,' lib. iii, cap. vi).

† The great French military surgeon, Larrey, has been the authority for the occurrence of emprosthotonos in tetanus; but Rose shows by detailed criticism that all his cases, belonged to a very mild form of tetanus. Larrey entertained the notion, certainly without foundation, that the spasms were distributed according as the wound which caused the tetanus was in front or behind.

Tetanus is more common in the tropics than in temperate climates, more common in negroes than in Europeans, and more common in men than in women. There is a special ætiological variety of the disease—*tetanus neonatorum*—which affects infants, usually in the end of the first or during the second week after birth. It is the result of an ulcer of the navel after tying the umbilical cord.

It is important for the physician to be aware that tetanus often follows very slight cuts and trifling abrasions, which are apt to be forgotten by the patient. Rose says that nearly half of his cases were sent into the hospital as cases of "rheumatism" or of some internal disease. Instances have been recorded in which tetanus has been ascribed to extraction of a tooth, to venesection, to the application of a cupping-glass, or to a cut from a whip. It is even said to have followed simple fracture of the limbs, or after falls upon the back of the neck, without any breach of surface.†

In 1860 a girl, aged five, died in Guy's Hospital, who the day before she was attacked by tetanus had a fall in which she grazed her elbow. Three other patients showed, one a slight scar over the knee, another a small sore on the elbow, and the third a little scab with pus beneath it at the elbow. The last, however, had also a cicatrix half an inch long on the forearm, due to a cut received about six weeks previously. In 1873 a boy was admitted under Dr Fagge's care in whom stiffness of the neck and jaws had come on four or five days after the healing of a small "gathering" on one great toe, from a nail in his shoe. This patient recovered. Again, tetanus has sometimes occurred soon after parturition or abortion. In a case which was observed at Guy's in 1870 no cause could be discovered but a prolapsed and excoriated cervix uteri. A patient of Dr Fagge, besides having a festering sore on the elbow, had got wet through in a shower of rain three days before the tetanus set in. A man under Mr Bryant had a slight scratch on the little finger, which soon healed; he also was exposed to wet two days before being attacked with stiffness of the shoulders.

Army surgeons have often noted that after a battle the wounded are most likely to be attacked by tetanus when they lie in tents on a damp surface, or when cold nights follow hot days. This liability to exposure to sudden changes of temperature in tropical climates may be one reason of the frequency of the disease after injuries among the coloured populations of the East and West Indies. A similar explanation may perhaps apply to Greece and Italy, where tetanus was well known. It is among the diseases which Hippocrates ascribes to cold (Aphor. v, 17), and Celsus remarks (lib. ii, cap. i), "Frigus modo nervorum distentionem, modo rigorem infert: illud σπασμός, hoc τέτανος Græce nominatur."

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* See Mr. Bowlby's remarks on this subject ('Diseases and Injuries of Nerves,' 1889, p. 304).

† See a remarkable case recorded by Mr. Charles Lucas, and quoted by Sir George Humphry, in which tetanic spasms followed a traumatic abscess of the thigh, and were cured by its evacuation.

Idiopathic tetanus.—It has generally been believed that tetanus may be caused without previous injury, and that it is then the effect of a chill.

On July 17th, 1863, a man sat in a draught, and afterwards felt a stiffness in his limbs; this continued during the 18th and the 19th. On the 20th more marked tetanic symptoms set in, and he came to Guy's Hospital and was admitted. At 7 a.m. on the 21st he had a severe paroxysm affecting his chest, in which he died.

Another patient, in 1869, had got his feet wet the day before the disease began. Sir Thomas Watson cites a case from Dr Gregory of a man who, "having fallen asleep in moist grass, awoke with a stiff neck, which afterwards went on into regular tetanus."

Such cases are not convincing. Similar accounts have been and still are given of the origin of Enteric fever. Draughts, chills, and wet feet are too common not to precede tetanus now and then by mere coincidence. Moreover the supposed cause does not apply to all the cases. Three patients died at Guy's Hospital between 1863 and 1875, of idiopathic tetanus, which Dr Fagge was unable to assign to cold or any other cause.

The most reasonable view is to compare these cases with those we sometimes meet with of non-traumatic pyæmia, or of syphilis or scarlatina, in which no contact with a previous case can be ascertained. We must not force facts in pathology, and there are no rules without exceptions; but as an occasional inexplicable case does not shake our well-founded belief that every case of syphilis or scarlatina was really acquired by contact with another one, so we may still believe that every case of "idiopathic" pyæmia or tetanus is really due to some unnoticed or forgotten wound.

Diagnosis.—This is seldom difficult. It is only at the very commencement of *trismus* from dental irritation in infants, that one could mistake it for true "lockjaw." Rose says that even at the earliest period of tetanus he has always been able to discover a certain degree of stiffness of the back of the neck, such that the patient was unable to bring the chin freely down to touch the chest. Another practical advice of his is to introduce one's finger into the patient's mouth, so as to feel the hard edge of a rigid masseter much more distinctly than from outside the cheek. Inability to open the jaws, from ankylosis of the temporo-maxillary joints, is in this way easily distinguished from trismus.

Tetanus is sometimes simulated by *hysteria*. Sir Thomas Watson mentions an instance of this kind in a girl, who "would all at once be drawn into a position such that the top of her head and her feet alone supported her, while her body was bent backwards like a bow; then, after a time, with equal suddenness, the opposite position was assumed, her forehead and her knees being brought together." His statement suggests the criterion which is applicable to all cases of this kind; namely, the irregular and inconsistent nature of the symptoms.

Another affection which has perhaps to be considered is acute *spinal meningitis*. A case given in 'Reynolds' System' as a typical example of the latter disease would show that it may very closely resemble tetanus; but, as Moxon argued, that may, after all, have been tetanus.

Tetanus is distinguished from hydrophobia by the much shorter period of latency after the wound, by the persistent trismus, and by the absence of mental disturbance. It is doubtful whether tetanus has ever followed the bite of an animal.*

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* See a review of this part of the subject by Mr Wm. Anderson ('Lancet,' Feb. 4th, 1888).

Tetanilla, or tetany, resembles a mild attack of tetanus in its aspect, but there is more continued spasm, the clonic movements are slower, and the pain much less. It is not traumatic, occurs in children, and is very seldom fatal. It will find its place among spasmodic diseases of the nervous system of obscure origin.

The most important diagnosis is between tetanus and the effects of *strychnia.* A boy aged twelve was brought into Guy's Hospital at nine o'clock one morning, suffering from opisthotonos, and from spasms of the respiratory muscles, so severe that he almost ceased to breathe. He was a druggist's boy, and confessed that between 7.40 and 8.30 a.m. he picked up some black stuff (afterwards ascertained to be extract of nux vomica) and put it into his mouth for liquorice, until he found it bitter, and spat out as much as he could. Ice applied to his spine gave him relief. From some urine passed at 1.20 p.m. Dr Stevenson succeeded in obtaining the reactions of strychnia and brucia. By that time his symptoms had passed off, and he was discharged from the hospital a few days later. The characteristic features of such cases are the suddenness with which opisthotonos and the most violent general spasms set in, the absence of persistent rigidity of the affected muscles during the intervals (so that the mouth can be freely opened), the fact that the hands are involved, and the rapid death or recovery which ensues. Even if the poison were administered in small doses, and repeated at frequent intervals, strychnia could simulate the progressive development of tetanus, as was proved in the famous trial of Palmer for murdering Cooke at Rugeley by repeated doses of strychnine.

Fatal event.—As a rule tetanus ends fatally between the third and the seventh days. The occurrence of death within twenty-four hours from the first symptom is very infrequent. Among Poland's seventy-two cases it only occurred twice; one patient, a young man who was attacked six days after a compound fracture of the leg, died in nineteen hours; the other, a girl who had been burnt, is said to have lived only four or five hours.*

Death often occurs during a paroxysm, and may be due to spasm of the diaphragm or of the glottis. Poland mentions that in one of his cases the heart's action continued for a short time after the breathing had ceased. In 1875 a patient suffering from tetanus in Guy's Hospital repeatedly turned perfectly livid, so that he seemed to be at the point of death, although clenching of his hands was the only outward indication of an increase of spasm; at last, after four days, one of these seizures proved fatal. Bauer, in Ziemssen's 'Handbuch,' believes that the danger from dyspnœa in tetanus has been over-estimated, since carbonic acid poisoning relaxes the muscles before death can take place.

The immediate cause of death is occasionally sudden syncope. Rose relates an instance in which he had his finger on the pulse when it suddenly and finally stopped; but this case was complicated by hyperpyrexia. Some have supposed that in cases of this kind the ventricles are seized with spasm, and stress has been laid on the fact that after death the heart is often found closely contracted; but this is in all probability the result of rigor mortis. There is no reason to believe that the heart or any other involuntary muscles share in the spasms of the limbs and trunk. Rose argues that the real cause of death is cardiac paralysis; he thinks that the muscular contractions oppose a resistance to the circulation through the arteries which the heart is unable to overcome.

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* The most quickly fatal case on record is one of a negro servant, who lacerated his thumb in breaking a china dish, and who was almost instantly seized with convulsions, and died in a quarter of an hour; but Sir Thomas Watson, although he cites it, expresses a doubt as to whether this was genuine tetanus, and in this doubt most readers will share.

There are rare cases in which life is prolonged for three or four weeks, but which yet prove fatal at last. The spasms may have altogether subsided, so that the patient is thought to be in a fair way to recovery, although he is excessively thin, weak, and prostrate, with sunken features and a scarcely perceptible pulse. Among the cases collected by Poland there are two which proved fatal during a paroxysm as late as the twenty-first or the twenty-second day, and two others in which death was referred to exhaustion on the sixteenth and the thirty-second days respectively.

The following case was one of those in which the paroxysms, although they recur but seldom, destroy life early.

The patient's main complaint was of a spasmodic pain in the right hypochondrium, which he attributed to wind. When it came on he used to get up and walk about, declaring that he could not lie down; he would stand leaning against the bedpost, and would call to his wife to rub his back. About a fortnight ago he had run a garden fork into his great toe. Four days before I saw him he began to complain of stiffness of his jaws, and from that time he was unable to open his mouth fully. These facts led to a strong suspicion that the abdominal pain was due to tetanic spasm. He died three days after my first visit, and about twenty-four hours after sitting up in bed to make his will.—C. H. F.

Convalescence.—When recovery from tetanus takes place, a certain degree of stiffness of the muscles often remains for a considerable time, and very slowly passes off. Poland mentions a case in which there was still some stiffness of the jaws after a year had elapsed. In 1871 Mr Golding-Bird had a boy under his care in Guy's Hospital in whom the spasms lasted for fifty-one days, and even after they had subsided his limbs still remained rigidly flexed, he became exceedingly emaciated, and bedsores developed, so that he ultimately died on the one hundred and seventh day.

Prognosis.—As a rule the recoveries are seen among the more protracted cases, and those which set in soonest after an injury are more dangerous than when the period of incubation is longer. It is very doubtful whether "idiopathic" cases are really less dangerous than those which are manifestly traumatic in origin. Unfortunately rapid progress of symptoms points more surely to fatal event than a slow course does to recovery. The average mortality of tetanus among the seventy-two cases collected by Poland in 1857 was 86 per cent. That among 505 cases recorded in the 'History of the War of the Rebellion' in the United States was 451, or 89 per cent.

Treatment.—There are certain measures obviously necessary. The patient should be placed in a quiet, dark room, and should be protected from all noise and excitement, and from the visits of friends. He should speak and move as little as possible. The food which is given to him must be fluid, and should be given as frequently as the patient can take it, with wine or brandy. If he is unable to swallow, enemata of beef-tea and brandy may be administered at regular intervals, unless this brings on paroxysms of spasm. Rose advised that anæsthesia should be induced regularly once or twice a day by chloroform, for the purpose of injecting food into the stomach through a tube. Purgatives should not be given after the first day.

The amputation of an injured limb or division of a nerve leading from a wound is of no avail, notwithstanding the apparently good results reported in the American Civil War. A splinter of wood or some other foreign body has been now and then unexpectedly found under a cicatrix, or embedded in a nerve-trunk; but even then, as it was not the cause, its removal is unlikely to be the cure, of the disease.

As regards treatment by drugs, when this terrible malady runs a rapid course one can often do nothing but keep the patient continuously under chloroform, so as at least to secure euthanasia.

On the other hand, when the progress of tetanus is slow, it often seems that the death of the patient is little more than an accident, from the supervention of a paroxysm which happens to exceed a certain limit of severity. In such cases one might hope that narcotic medicines, or those which relax the muscles, would save life. With this object, as well as to relieve the patient's sufferings, *opium* has been often employed. Sir Thomas Watson mentions the case of a lady who took more than four ounces of laudanum a day during twenty days, and who recovered; and he refers to another case in which an ounce of solid opium was swallowed in divided doses every day for three weeks. The hydrate of *chloral*, with or without bromides, has sometimes been used in cases of the same kind with apparent success.

In 1870, for instance, Mr Birkett had under his care at Guy's Hospital a man aged twenty, who on June 24th had received a kick on an ulcer. Next day he felt rigidity of the muscles of the face, and was unable to swallow solids. He was admitted on the 28th. At first thirty grains of chloral were given every four hours, but on the 30th, thirteen doses having been taken, a grain of opium was ordered to be taken every three hours instead. However, the spasms became more severe; and on July 4th a drachm of chloral was prescribed at one dose, and half a drachm on the following evening. After this fifteen grains were given every other hour until the 11th, when the quantity was increased to twenty-five grains every other hour. Two days later it was reduced again, and the disease now gradually subsided in the course of the next three or four weeks.—A full report of this case will be found in the 'Guy's Hospital Reports' for 1878, by Dr Frederick Taylor.

The inhalation of chloroform, repeated or continuous, has been fairly tried, and on the whole has disappointed expectations. Still it is very useful as a means of relief and to allow of feeding. Cannabis indica has also been used, and sometimes with apparent benefit. Bromides appear to be only useful when combined with chloral hydrate.

Among the depressants of muscular action *physostigma* has been employed at Guy's Hospital in one case which ended favourably.

The patient was a man aged twenty-one, who, having got drunk on November 30th, 1874, began to suffer from stiffness in the back on December 1st. He was admitted, under Dr Wilks, with fully developed tetanus on December 5th, and spasms recurred every three or four minutes. The extract of Calabar bean was given at first in small doses, but afterwards one grain every two hours; and on the 10th, after a very severe seizure, seven doses of a grain each were administered at intervals of fifteen minutes in succession. He began to improve about December 17th, but the stiffness of the joints remained until after the middle of January, 1875.

Against this case must be set another one under Dr Fagge's care in the summer of 1875, in which death occurred on the sixteenth day, in spite of two and a half grains of extract of physostigma repeated at intervals of only an hour. Previous to these two cases one was published in the 'Practitioner' for 1874 (vol. xiii, p. 345), in which enormous doses of physostigma were followed by recovery.

Subcutaneous injections of *curare* were administered to a man under the care of Mr Durham in 1876; but it seems doubtful whether the doses employed were large enough to be efficacious. The danger of paralysing the muscles of respiration would be great if this drug were pushed. Another medicine which has now and then been followed by recovery is quinine. An instance of this was recorded by Dr Bright in the first volume of the 'Guy's Hospital Reports.'

In the collection of cases published by Dr Taylor in the same 'Reports' for 1878, there are recorded nine in which death occurred under chloral (six by the fourth day, and the other three on the eighth, tenth, and twelfth days), and seven which ended fatally under Calabar bean (six by the fourth day, the seventh on the eighth day).

Antitoxic serum.—It was in cases of tetanus that in 1890 Behring and Kitasato invented the antitoxic treatment, which led to the more important treatment of diphtheria by similar means. The least susceptible animals like the dog, and the more susceptible like the rabbit, the horse, and the sheep, may be rendered immune to the tetanus toxine, and their serum injected under the skin is an efficient antidote to the toxines artificially introduced into the system. Unfortunately, in the case of the human subject the intoxication has often gone too far before the initial spasm gives warning that the spinal cord is already affected.

It is nevertheless encouraging that in a large number (above 400) of domestic animals (horses, mules, cattle, sheep, and swine) which were injected with the antitoxin after various accidental injuries in France during 1897, not a single case of tetanus occurred.*

Probably the most effectual practice would be to inject the antitoxic serum in any case where a wound had been soiled by earth or dirt, even if no bacilli were discovered, for the search is a very difficult one. The hope of doing good when the symptoms of tetanus have once appeared is, however, well founded and increasing.

In 1896 M. Haushalten collected forty-four cases of tetanus healed by antitoxic serum, with twenty-six recoveries and eighteen deaths; but in 1898 Kœhler published in the 'Münchener med. Wochenblatt' ninety-six similar cases, with sixty-three recoveries and thirty-three deaths—a decidedly better result than that obtained by drugs.

A remarkable observation made by Wassermann was that by mixing tetanus toxine with pounded cerebral substance it was rendered innocuous when injected under the skin, and this was ascertained by Roux and Borrel to be due to the toxine uniting with the nervous substance so as to become insoluble. This fact probably explains the fact that intra-cerebral injections of tetanus toxine are more severe and more rapidly fatal in animals than when made under the skin; and Roux and Borrel further ascertained that if the antitoxic serum was injected into the brain of a guinea-pig poisoned with tetanus toxine, but not yet convulsed, it was completely protected. This mode of injection has been practised in human beings, but unfortunately only after the initial spasms have begun, and the result has been, as a rule, negative. A successful case has, however, been reported from New York.

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* A still larger series is quoted from M. Nocard by Dr Héricourt in his recent monograph on 'La Sérothérapie (1899);' but as these cases were those of operations their importance is but small, for no good veterinary surgeon would allow the entrance of the bacilli of tetanus any more than the micrococci of pyæmia.

AGUE

> "Ye ben full cholerik of complexion:
> Beware the sunne in his ascension
> Ne find you not replete in humours hote;
> An if he do, I dare well lay a grote,
> That ye shall have a fever tercian,
> Or an ague that may be your bane."
> CHAUCER.

Intermittent Fever—Its history and pathology—Incubation—The cold, hot, and sweating stages—Regular and irregular varieties—Remittent Fever—Blackwater fever—Dysentery—Malarial cachexia—The spleen, liver, and other organs in ague—Melanæmia—Nature and laws of malarious contagion—The specific amœbiform microzoon—Diagnosis—Treatment and Prevention.

Definition.—Intermittent or remittent pyrexia, with congestion of the spleen and other abdominal viscera, running an indefinitely prolonged course, liable to recurrence and to sequels, dependent on the presence of a parasitic microzoon in the blood-corpuscles.

Synonyms.—Intermittent Fever—Periodic, Malarial, Miasmatic, Paludal, or Marsh Fever.—*Fr.* Fièvre intermittente, palustre, paludéenne.—*Germ.* Wechselfieber, Sumpffieber. The English word "Ague" is derived from Fièvre aiguë, *Febris acuta.*

History.—The disease which forms the subject of the present chapter has happily lost much of its practical interest for English physicians. But it is of great historical interest, and continues the most frequent and serious disease in the Mediterranean countries, in the East or the West Indies, and in many other regions.

In its most striking forms its course is interrupted by definite intervals of apyrexia, so that, instead of being a "continued" fever, it consists of a series of paroxysms or "ague-fits." Such cases are called *intermittent fever;* but they cannot be separated from others due to the same cause but with only partial subsidence of the pyrexia, and therefore distinguished as *remittent.* The English term "Ague" includes both of these forms with their varieties and sequelæ,—in fact, all that owns the same origin.

The poison which excites ague was long known as *malaria* (*i. e.* bad air). It enters the human body from without; but, like the virus of tetanus, it never passes directly from one person to another. In other words, ague, though in a broad sense one of the "infective" diseases, *i. e.* one in which the whole body is affected by a morbid agent introduced from without, is not "contagious" nor even "infectious" in the narrow sense in which that epithet is generally employed. Vaccinia and Syphilis are contagious from person to person, *i. e.* inoculable by direct contact. but not infectious, *i. e.* not conveyed to persons at a distance. Typhus and Measles are contagious and infectious, *i. e.* conveyed from person to person by direct contact and also at a distance. Cholera probably, and Enterica almost certainly, are not conveyed through the air, nor yet by direct contact, but only by means of a contagium which is reabsorbed after its discharge from the body. Ague, like Cholera and Typhoid fever, is not conveyed from person to person directly, but through an intermediate host of the micro-organism which is its true cause.

Like the ovum of a tapeworm or a trichina, the *contagium vivum* of Ague can only complete its cycle of development in two hosts; and one of these is an insect, the other a human being.

This *contagium vivum* of Intermittent fever is not a microphyte, but an amœba, and this explains some of the broad differences which separate it from the bacterial fevers. The hæmatozoon was discovered by Laveran in 1880.

Descriptions of the different varieties of ague are to be found in Celsus and other ancient writers; for various forms of intermittent fever were and still are the commonest diseases in Greece and Italy. The first to ascribe it to "malaria," regarded as a noxious effluvium given off by marshes, appear to have been Richard Maton, in 1696, in his 'Pyretologia,' and Lancisi, in a work published at Geneva in 1716. The true ætiology of ague has only been discovered within the last ten years.

Down to a recent period intermittent fevers—usually tertians or quartans—were common over the greater part of England, particularly in London and in the counties of Lincoln, Cambridge, Essex, and Kent. Drainage has much limited the extent and severity of the disease, but it is still met with along the banks of the Thames, and the writer used to see more or less marked cases of malarial cachexia among the out-patients from Rotherhithe and Deptford.

The geographical distribution of malarial disorders has, however, so important a bearing on their ætiology that it is best deferred till later.

It is curious that malaria appears to be almost without influence upon domestic *animals*, at least in those countries where its effects have been most studied. In Italy, however, a few instances have been recorded in which horses or oxen have had intermittent attacks of fever, or have suffered from cachexia attended with enlargement of the spleen.

Incubation.—Most of those who are attacked by ague seem to succumb to repeated invasion of the parasite rather than to take the disease on any single occasion. When ague follows a single exposure it is after an interval of from six to twenty days.[*] On the other hand, sailors have fallen ill upon the open sea, weeks or months after leaving a malarious coast; but probably they had taken mosquitoes with them.

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[*] Dr Maclean, in 'Reynolds' System,' mentions the case of three German missionaries, fresh from Europe, who passed a night with an English officer in an unhealthy spot at the foot of the Segoor Pass in India; next morning they pursued their journey, but within less than twenty-four hours three out of four of the party were stricken with fever. Hertz, of Amsterdam, writing on this subject in 'Ziemssen's Cyclopædia,' declares that on several occasions, having purposely placed himself in a marshy ditch at a time when it was drying up, he has been attacked within half an hour by giddiness, shivering, nausea, and other symptoms, which ended in a slight paroxysm of fever.

Direct experiments with malarial blood show that the period of incubation after inoculation varies between a week and a fortnight.

Premonitory symptoms.—Among strangers who have recently entered a malarial district, the occurrence of a regular paroxysm is in most cases the earliest symptom of ague. But, according to Hertz, when the patient has been living for a long time in the same place, a prodromal stage may sometimes be observed. It lasts about a week, and consists of vague malaise, lassitude, headache, and pains in the limbs, in association with nausea and loss of appetite. There may also be sensations of chilliness, alternating with slight flushes of heat. Manson also has observed this premonitory stage as occasionally preceding the first sign by several days, a week or less.

The attack.—The ague fit is divided into three periods, which have long been known as the "cold," the "hot," and the "sweating" stages.

1. The *cold* stage begins with the patient feeling tired, weak, and listless. He yawns and stretches out his limbs. He complains of an uncomfortable sensation at the epigastrium, of headache, or back and leg-ache, or of giddiness. So mild a fit may easily be unrecognised, but in a fully developed one these symptoms are followed by shivering or a *rigor*. The patient feels chilly along the spine, and then all over him. He shudders, his teeth chatter, his knees knock together, his whole frame may be so violently agitated that the bed into which he has crept shakes beneath him.* His voice is feeble, and his speech is interrupted by the quivering of his lips. At the same time his appearance undergoes a remarkable change. He grows pale, his features shrink, his ears and nose and finger tips turn livid, his skin becomes dry and rough—*cutis anserina*, or "goose-skin." Yet the thermometer, as De Haen discovered more than a century ago, shows that in the central parts of the body the temperature is not lowered, but raised several degrees above normal. It must not be supposed that the patient's sensations deceive him as to the condition of the surface. One has only to feel the fingers or nose of the patient in order to be sure of the fact that they are really cold; and a surface thermometer, or an ordinary thermometer held in the patient's hand, will remain below the natural standard, while in the axilla, the mouth, or the rectum it shows marked pyrexia.

The temperature has begun to rise before any symptom of the onset of the ague is felt. At first its increase is gradual, but with the development of the rigor it runs up, and may pass through four or five degrees Fahrenheit in the course of an hour.

The patient during this stage feels dry and parched; his tongue is white; he often complains of nausea, and sometimes vomits. His pulse is quick and small, and may be irregular. His respiration is short, hurried, and distressed. His urine, although pale, is often very irritating, so that Maclean has found it desirable to administer bicarbonate of potass and even tincture of opium for the relief of this symptom. Watson spoke of the urine as scanty, although passed frequently; but the careful measurements and analyses of Redtenbacher and of Ringer have shown that it is in reality considerably increased in quantity, when compared with the amount passed each hour during the apyretic interval, and that there is also a marked increase of the urea and chloride of sodium. The excess, both of the water and of these solid constituents, begins before the patient feels chilly, and lasts throughout the fit.

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* "In young children it is not at all unusual to have a convulsive seizure at this stage" (Manson).

2. The *hot* stage gradually succeeds. The patient ceases to shiver, and begins to experience flushes of heat about the face and neck, which presently diffuse themselves over his body. But for a time, if he attempts to throw off the bedclothes he has piled over him, slight rigors reappear. At length, however, even the extremities become hot and glowing. His aspect is again altered. His face is flushed, his eyes are injected, his skin becomes smooth and turgid. The temperature, as measured by the thermometer, still continues to rise for some time, and reaches 104°, or sometimes 106·5°. The surface of his body, even of the distal parts, now feels pungently hot to the hand. Evidently there is not only an increased generation of heat, but it is now freely carried to the periphery, and abundantly dissipated. The pulse is not only rapid, but full and throbbing; and the breathing is deep as well as frequent. The patient complains more than ever of headache, and he is exceedingly restless and uncomfortable. The urine during this period is scanty, high-coloured, and concentrated. There is often an eruption of herpes on the lips, the nose, or the tongue. Albumen in the urine, accompanied by casts and even by blood, has been observed by Griesinger and Hertz, but this is a very rare complication. The hot stage usually lasts two or three hours.

3. The *sweating* stage follows in its turn. A little moisture breaks out on the forehead and the face, and before long the whole skin is bathed in the most copious perspiration. It is said that the thermometer sometimes continues to rise for a little while, so that the maximum temperature may occur during this, and not during the hot stage; but, as a rule, the pyrexia begins to decline from the moment when the skin becomes moist. The fall is at first slow, but after half an hour or an hour it becomes more rapid, and goes on until the normal temperature is reached. According to Wunderlich, this takes place by a series of steps, there being a drop of from one fifth to one third of a degree Fahr. about every fifteen to thirty minutes. All observers speak of the urine during this period as showing a thick deposit of lithates, and it sometimes gives the reaction of bilirubin. The breaking out of perspiration is attended with complete relief to the patient; he loses his pains, the throbbing of his arteries ceases, he is no longer tormented with thirst, and his tongue becomes moist. After three or four hours, or earlier, he falls asleep, and when he wakes feels perfectly well.

Recurrence.—The subsidence of the ague fit is followed by similar attacks, repeated again and again at definite intervals. If a fit occurs every day the ague is said to be *quotidian*. If it occurs every other day it is called *tertian*, for according to the Latin way of counting it comes every third day, or as we should say every other day. If there are two entire days between the paroxysms the ague is called *quartan*. In other words, an interval of about twenty-four hours characterises a quotidian, one of forty-eight hours a tertian, and one of seventy-two hours a quartan. Nor does this exhaust all the possible varieties. Sometimes when there is an attack every day, each paroxysm differs in severity or duration or in the hour of its occurrence from that of the preceding day, and exactly resembles that which occurred two days before. This kind of fever is called a *double tertian*. So, again, it is possible to have a double, or even a *treble quartan* fever. Or a patient may have two ague-fits one day, and a single fit on the following day, and the series be regularly repeated, so as to show that a quotidian and a tertian are combined. Such a fever is the ἡμιτριταῖος of Celsus, or, as subsequent Latin writers termed it, *semi-tertian*.

It was first ascertained by Golgi, whose statements are confirmed by subsequent observers, that these ancient varieties of ague are characterised by different forms of the hæmamœbidæ (*v. infra*, p. 398).

The return of the paroxysms of ague is often so regular that it can be foretold with absolute certainty; but sometimes it is earlier each time by a constant difference; and sometimes it is later. In the former case the disease is said to *anticipate;* in the latter case to *postpone*. While one is a sign that the severity of the case is on the increase, the other indicates that it is becoming milder. Such variations may even bring about a change of type; thus an anticipating tertian may at last pass into a quotidian, and a postponing quotidian may be gradually changed into a tertian. Sometimes the attacks of a quotidian fever are so prolonged that before the sweating stage of one fit is over the cold stage of the next one begins; the disease is then said to be *subintrant*.

With regard to the comparative severity of the three principal forms of ague, quartans are said to be the least dangerous but peculiarly obstinate.* This variety, which is comparatively rare, is most apt to occur in the autumn; and it has long been observed in Italy that autumnal agues are worse than those contracted in the spring.† Hertz states that in the tropics the disease never assumes the quartan type, and the same was stated to be true of Calcutta by Dr Crombie in 1894. But Ross has met with quartan fevers in Madras (1896). In hot countries the quotidian is the most common form of ague, whereas in temperate climates tertians and quartans are most frequent.

The fits of a quotidian are said generally to set in during the morning, those of a tertian about noon, those of a quartan still later in the day; and it is stated that the average duration of a paroxysm of quotidian ague is from ten to twelve hours, that of a tertian from six to eight hours, that of a quartan from four to six hours, but that in the last named the cold stage is the most prolonged.

It appears, however, that all these varieties are liable to mix and change the one with the other. For example, Dr North caught a double quotidian fever at La Fontana in August, 1885; it reappeared as tertian in June, 1886, and (in England) in July afterwards, and in May, 1888, as quartan.

Subsequent course.—Intermittent fever appears to be never directly fatal. Its duration is very variable. Sometimes, even when the patient remains in the place where he acquired it, it ceases after a few paroxysms. In other cases it comes to an end with a change of season. In others, again, ague recurs for an indefinitely long period, until the victim is compelled to remove to some other district. There is always a strong tendency to relapse, even without a fresh exposure to the ague poison; and it is a remarkable fact that, whether the type be tertian or quartan, the paroxysms of a relapse often occur on those very days on which they would have been due if the disease had gone on without break from the first. Graves has related an instance of quartan ague which he watched for twenty-seven months, and in which the periodic rate was maintained through thirteen out of sixteen intervals, some of which lasted more than two months. A similar observation had previously been made by Dr James Gregory in the case of a relative, who had marked on an almanac the days on which the fits of a tertian ague might be expected to recur, and who found that for a long while they kept tryst, but only when the east wind blew. Since the introduction of the thermometer into clinical practice it has been shown that there may be a distinct rise of temperature at the proper time for the paroxysm, although the patient does not feel ill. Moreover Ringer states that even when all febrile disturbance is absent the periodicity of the disease may be indicated by voiding a large amount of urine containing an excess of urea. These clinical observations are confirmed and explained by the evidence that minor swarms of *hæmamœbæ* appear in the blood during apparent intervals of health.

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* The Latin couplet runs: *Pro febre quartana Rara sonat campana.*

† "An Ague in the spring is Physick for a King." Yet James I died of a tertian fever in March.

Varieties.—Sometimes the cold stage is absent, sometimes the sweating stage.* In certain exceptional cases the paroxysms differ altogether from those just described.

The strangest form is that which has been termed *syncopal*, in which there is a condition of suspended animation so long that the patient runs a risk of being buried alive. Trousseau relates two instances of this.

One occurred in a station-master on the Avignon Railway. He had been subject for some time to paroxysms of intermittent fever, and had repeated fainting fits; once he became pulseless, was supposed to be dead, and was carried to the mortuary. After some hours a servant happened to enter the place, and found him groaning; he was therefore taken back to his bed, and under large doses of bark he regained his health. It is singular that Dr Chauffard, the physician who observed this case, also met with another one of the same kind. A man had fallen into a faint, was taken for dead, and his face was covered with a sheet; Dr Chauffard, however, detected slight movements of his heart, although the radial, axillary, and carotid arteries had ceased to beat; he immediately administered a quinine enema, and the man was saved.

Scarcely less remarkable is another variety, which may be called *comatose*. Hertz says that instances of it were from time to time sent into the hospital under his care for apoplexy, and Graves placed on record a case in which he made this mistake.

A gentleman awoke at about 4 a.m. with sensations of malaise, chilliness, nausea, and headache. After an hour he became extremely hot, the pain in the head was intense, and he passed from a drowsy condition into one of complete coma, with deep snoring, so that he "appeared to be labouring under a severe apoplectic fit." He seemed to derive much advantage from bleeding and other remedies, and in the evening he was perfectly well. The day but one after the same symptoms returned, and were removed by the same treatment; but when a third attack came on Graves saw that it was an example of *tertiana soporosa*, and cut it short by full doses of quinine.

In other cases it is said that epileptiform convulsions or tetanoid spasms have been present, or symptoms like those of hydrophobia. It is also believed that ague may imitate cholera, dysentery, peritonitis, or pneumonia.

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* It was formerly believed that sometimes the usual order is reversed. In a case cited by Watson from Maugenet, the patient was always first attacked with profuse sweating; then he became dry and hot; finally he felt cold and had distinct rigors. This probably groundless belief is expressed in Butler's lines:

> "'Tis but an ague that's reversed,
> Whose hot fit takes the patient first."
>
> *Hudibras*, part iii, canto i, 653.

Modern French writers describe such cases as *accès pernicieux*, under the two forms "cerebral," convulsive or paralytic; and "algide," syncopal or choleriform.

Hertz tells us of a man who was attacked at two o'clock in the morning with shivering and pain in the left side; at eight there was a distinct pleural friction-sound, but twenty-four hours later he was perfectly well. However, on the following night, at twelve o'clock, all the symptoms returned, and by nine the friction-sound was more evident than before; at four in the afternoon he was sweating and free from pain; full doses of quinine were therefore prescribed, and he had no further attacks.

With regard to such cases, we must remember that in districts where ague prevails all kinds of diseases assume a more or less intermittent character. Thus in so-called *masked agues*, the paroxysms are stated to be of the most diverse description, and attended with little or no fever. The most important of these is neuralgia, usually in the region supplied by the first division of the fifth nerve (and often called "brow-ague"), but sometimes assuming other forms. Not only have cases of sciatica been attributed to ague, but the same view has been taken of painful affections of the mammary gland or of the testicle, and even of alarming seizures attributed to cardialgia or to neuralgia of the vagi; but it is hard to say what evidence in favour of such interpretation would be conclusive. It is not enough to show that the attacks recur with a certain degree of regularity, and pass off under the influence of quinine, for almost as much may be said of neuralgia. The observance of a tertian or quartan type would no doubt be very significant, but masked agues appear to be usually quotidian.*

Remittent fever.—The effects of the presence of the *hæmamœba* in the blood are not limited to the production of the different forms of intermittent fevers. In hot countries, and during the hot seasons of the year, even in temperate climates, it causes fevers which are either continuous or interrupted only by *remissions*, intervals in which, instead of there being complete apyrexia, there is only a partial lowering of the temperature and of the pulse, with some degrees of abatement of the other symptoms. For cases of this kind the technical name is *remittent fever*; in India they are called *jungle fever* or *bilious remittent*. They are marked by symptoms which do not belong to the milder effects of malaria, as well as by the absence of some of the distinctive features of ordinary ague.

The cold stage is short, often slight and ill-defined; the hot stage is prolonged to six or even twelve hours, and this is followed by little or no sweating. The remissions, which often begin about midnight or in the morning, and which last for twelve hours or more, may be so slight as to require careful clinical observation for their detection.

Jaundice is frequently present in a moderate degree, and gives the term "bilious remittent" to such cases. When deep, jaundice is a bad symptom.

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* It must be borne in mind that the more extensive a man's experience in regard to intermittent fevers, the more likely is he to be led astray by tradition, or by personal bias, until he has recourse to the same convenient hypothesis for the solution of every obscure and indeterminate affection, such as occur frequently enough to those who practise in districts where no ague is met with. What, for example, is to be said of the case of Dr Macmichael, cited by Sir Thomas Watson, who "caught an ague many years before his death by sleeping on a rock somewhere in Greece, and was ever after subject to occasional attacks of periodic headache and other aguish symptoms, for which he was obliged to have recourse to bark or arsenic"? And what value can be assigned to Trousseau's statement that there are cases in which insomnia, unaccompanied by fever and not preceded by rigors, recurs every two or three nights, that these cases are of the nature of masked ague, and that they are cured by similar treatment?—C. H. F.

A feeling of oppression at the epigastrium is present from the outset of the patient's illness, and vomiting is usually a marked symptom throughout its course. The vomited matters may at first consist of food, but afterwards a watery fluid is ejected, often in surprising quantity, and it may ultimately become greenish yellow or brown. Headache is generally severe, but not delirium.

In the worst cases, in which the disease is said to be of an "adynamic" character, the patient rapidly becomes prostrate and insensible, his skin is yellowish and marked by petechiæ, his tongue is black, his teeth are covered with sordes, and hæmorrhages may occur from the nose, the mouth, or the kidneys. French writers have described the latter cases under the title *fièvre bilieuse hématurique*. Probably these cases are identical with "black-water fever." Maclean, from whose description of remittent fever in 'Reynold's System' most of these details are taken, says that the urine seldom contains albumen without blood: it is often abundant, and sometimes pale.

The duration of remittent fever is said to be generally from five to fourteen days. Even its worst forms ought, according to Maclean, to do well in most cases if seen early and skilfully treated. Sometimes it ends with a critical perspiration, sometimes it subsides gradually, sometimes it passes into one of the regular types of intermittent fever. Death seldom occurs before the seventh or the eighth day, a point which may distinguish this disease from yellow fever in countries where they both prevail.

Typho-malarial fever is a name used in the United States for cases which are probably not a separate form of disease, but enteric or other specific fevers occurring in persons subject to malaria. See Dr Johnston's paper ('Trans. Assoc. of American Phys.,' p. 40).

Black-water fever.—This is in all likelihood a malarial fever, and is known as hæmoglobinuric, bilious remittent, or West Africa fever, the *fièvre bilieuse mélanurique* of French authors. The presence of hæmoglobin in the urine is its distinguishing feature. It is endemic in equatorial Africa, and has been recognised in Madagascar and in the Malay archipelago. In the New World it is common in Cuba and other parts of the West Indies, and in Central and tropical South America. It is supposed to have become more common during the fifty years which have passed since it was first recognised.

Dr Crosse, who spent several years as principal medical officer to the Royal Niger Company, believes that the reason the hæmoglobin was overlooked was that it was attributed to the presence of bilirubin; for in this case there is always slight and sometimes deep jaundice. He believes that black-water fever is not a separate disease, but malarial remittent fever, with breaking down of the blood-discs and excretion of their hæmoglobin. If actual passage of the blood-corpuscles (true hæmaturia) occurs it is quite exceptional.

Its origin (beside the presence of the *plasmodium malariæ*) is ascribed to such exciting causes as exposure to the sun, neglecting to take quinine (v. p. 403), fatigue, and taking a chill while suffering from malaria. The hæmoglobinuria never appears until intermittent or remittent fever has lasted two or three days. There is often vomiting, but never hæmatemesis.

The prognosis is not unfavourable, particularly in a first case: but repeated attacks may end fatally, and even after return to England the black-water will accompany each relapse of fever. The most common immediate cause of death Dr Crosse finds to be suppression of urine.

The relation of endemic hæmoglobinuria, as a complication of bilious remittent fever, to sporadic hæmoglobinuria without fever, as we see it in England, is curious and interesting. The latter affection will be discussed in the second volume, under Disease of the Kidney.

Dysentery and Hepatitis.—The close clinical relation between ague and dysentery in its tropical and most characteristic form has been long recognised. They abound in the same regions, they follow much the same rules of affecting those who come within their range, and they frequently attack the same individual.

Moreover the abscess of the liver, which has long been known as tropical, and sometimes as dysenteric, is now found to harbour a similar parasite. Amœbic dysentery and amœbic abscess are therefore natural congeners of malarial fever. Nevertheless it would at present be premature to assume all tropical dysentery and all tropical hepatitis to be amœbic; and on this basis it would be impossible to draw any but an arbitrary line between cases. We defer the discussion of these affections, therefore, to the chapters which deal with diseases of the bowels and with diseases of the liver.

Malarial cachexia.—In persons who have long and frequently suffered from ague, a chronic condition of ill-health is apt to develop itself, which is known as malarial cachexia. The same condition is often seen in those who have lived for a long time where ague is prevalent, though they have never had the fever. The condition is marked by a pale, earthy, sallow complexion, and a damp, clammy state of the skin, especially noticeable in the hands. The patient is depressed in spirits, and wakes unrefreshed by sleep; he often suffers from giddiness, noises in the ears, tingling in the hands and feet, pains in the back, palpitation, loss of appetite, a furred tongue, and digestive disorders.

In such cases enlargement of the spleen can be made out on percussion and palpation, and sometimes it forms a conspicuous tumour, known as the "ague-cake." The spleen swells with every paroxysm of the disease, the yielding capsule of this viscus allowing it to receive the blood, which is driven inwards from the surface of the body in the cold stage. It subsides during the intervals, but gradually less and less completely, until at last it forms a permanent, firm, solid tumour.

Anatomy.—In cases of death from malarious cachexia or remittent fever the *spleen* is found enlarged. In acute cases it is soft or diffluent, and during life it may rupture from the slightest accident, pour blood into the peritoneal cavity, and bring the case directly to a fatal issue. In cases of chronic malaria the spleen becomes extremely indurated. Not only is its hardness discoverable by palpation during life, but after death it is found to have little or no pulp, while the fibrous elements of its substance have much increased. Its capsule is often thickened, and it may be fixed by adhesions to adjacent parts. The colour of the spleen is much darker than usual, owing to a quantity of pigment deposited in its tissue. The *liver* is found usually enlarged and indurated, and according to some authors may be in a state of cirrhosis. The kidneys often show signs of tubal or interstitial nephritis, but this is so common a condition that it may be only a coincidence.

Malarial *melanæmia* is marked by the presence of a brown or black pigment, partly free, partly enclosed in leucocytes, circulating in the blood, and also by the same pigment in the solid tissues. It is derived from the colouring matter of the blood by the agency of the specific microzoon (*infra*, p. 399). It causes the liver, the spleen, and the kidneys to assume a slaty-grey colour; the marrow of the bones becomes chocolate-brown, and the cortex of the brain looks as if rubbed over with black-lead.

A striking example of pigmentation of the brain, no doubt malarial in origin, occurred at Guy's Hospital in 1829 in the practice of Dr Bright, who figured it in his 'Medical Reports.' The case was that of a man who, with his wife, died of severe fever immediately after they had walked up to London from Horncastle in Lincolnshire. In Germany, as in England, melanæmia with abundant pigmentation of the organs appears to be very rare; all the well-known cases recorded by Frerichs belonged to an epidemic of ague which arose in Silesia after an inundation in 1854. But more than one observer has constantly been able to detect pigment in the blood, by microscopical examination, in cases of "pernicious" ague, or of malarial cachexia. In 1877 Dr Stephen Mackenzie made a similar observation several times in the case of a man who had brought ague with him from India. The red discs were normal; but many leucocytes contained granules which were arranged round their nuclei, or sometimes completely filled them so as to give them a uniform brown or black colour. After treatment with quinine the melanæmia was no longer discoverable. The pigment is supposed to be originally formed in the spleen, and to pass thence into the blood, to be carried all over the body. It often blocks up the capillaries, and is also found in the walls of larger blood-vessels. These facts do not prove that it may not have come from elsewhere, for wandering leucocytes may have carried it with them; but many pathologists think that, in some cases at least, it is developed *in situ* from extravasated red corpuscles. Whether melanæmia directly gives rise to any symptoms appears to be doubtful. Dr Mackenzie's patient had hæmorrhages into each retina, and the same thing has been observed in some other cases of ague. In 1880 a man who had caught ague in the Black Sea was in Guy's Hospital; he had a large hæmorrhage in one retina, but no pigment could be found in the blood. Capillary hæmorrhages in the brain have been attributed to pigmentary embolism of this organ.

Contributing causes.—The word *malaria* signifies bad air, but water has more to do with intermittent fever, because it is in water that the earlier stages are passed of the insect which is the host of the *hæmamœba*. That ague is apt to prevail in marshy districts is universally admitted; but in Ireland malaria is very seldom met with; nor, as observed long ago by Parkes, are salt marshes infested with ague when they are regularly overflowed by the tide. Ague has sometimes been seen in dry and barren districts, such, according to Hirsch, as the table-land of Castile, the plain of the Araxes, and the lofty plateaus of Northern India and Persia, all of which are malarious. British troops have been attacked with the disease while encamped upon dry sandy soils, both in Holland and in Spain; and malaria exists in Hong Kong, situated upon disintegrating granite rocks. These exceptional cases are fatal to an hypothesis which at one time was generally held, that malaria is nothing more than a product of decomposing vegetable matters, but are not incompatible with the presence of sufficient water to afford opportunity of insects breeding and biting. Ague is said to be entirely absent from the great Sahara and from the deserts of South Africa, but to appear in oases where there is water.

Ague has reappeared in places long free from the disease when the soil has been disturbed for building purposes, or for the construction of canals or fortifications. Dr Manson cites Hong Kong as a striking modern instance of this long observed fact. Conversely, much can be done towards preventing intermittent fevers by draining the ground, or by paving the streets and courts of towns.

At certain times intermittent fevers spread to regions which lie far beyond their usual limits, and assume somewhat of an *epidemic* character. The years 1558, 1678-9, 1718-22, 1807-12, 1824-7, 1845-8 are mentioned by Hertz as having been characterised by such a wide diffusion of ague. Watson says that he never knew ague widely prevalent in London except in 1827. From 1866 to 1868 there was an epidemic in Mauritius, although the island had previously been so free from malaria that the sufferers from Indian fevers resorted thither. In 1869 it broke out for the first time in Réunion.

Heat.—Malarial fever is more common and severe in the tropics than in temperate climates, and in very cold countries it is unknown; its boundaries are about 63° N. and 57° S. of the equator. In this hemisphere Hirsch finds the highest range of malaria toward the north to be between the isothermal lines of 60° and 61° Fahr. mean summer temperature. Again, in particular districts, malarial fevers during the winter disappear entirely; and, other things being equal, they are more severe in hot seasons. In the tropics the period of the year at which ague is most prevalent varies in different localities; generally speaking, it closely follows the rainy season. Hertz lays down the rule that, in order to escape malaria, one should arrive in the East Indies between November and January, and in the West Indies between January and March.

In temperate climates ague is more common in the spring and autumn than in the height of summer.

The liability to ague is less as altitude increases. Parkes says that in temperate climates an elevation of at least five hundred feet above a malarial spot should be reached in order to escape its influence; in the tropics, one of a thousand to fifteen hundred feet, or even higher still, *i.e.* the cooler the place the less malaria. There is some evidence that, both in barracks and in private houses, persons sleeping on the ground-floor are more apt to be attacked than those who occupy upper stories. It is at night, and for a short time after sunrise, that malaria is most feared.

The presence of growing vegetation appears to be adverse to the development of malaria; and the Eucalyptus is believed to be particularly useful in this respect. This is probably chiefly due to its drying the ground, as all fast-growing trees do, but also perhaps by protecting from cold winds, and possibly by keeping off mosquitoes.

When English troops occupied Walcheren and other parts of Holland, it was repeatedly noticed that only the soldiers who disembarked were attacked by ague; those who remained on board ship, even in narrow channels, escaped.

Chorography.—In England the chief seats of ague were along the eastern coast; Romney Marsh in Kent, the estuary of the Thames in Kent and Essex, the fens of Cambridge, Huntingdon, and Lincolnshire, and the marshy lands of the East Riding of Yorkshire. In all these districts the disease has become far less frequent than formerly, since population has increased and improved drainage has brought more and more land under cultivation. Until late years cases were common in London. James I and Oliver Cromwell * died of ague contracted in this city. At present its occurrence is altogether exceptional, even in the low-lying parts of Southwark and Battersea. In Scotland ague is now unknown. On the continent of Europe the regions in which the ague poison is most prevalent are the following:—the great plain of North Germany, with the Baltic provinces of Russia; Holland, with the adjacent parts of Germany and Belgium; the south-western departments of France, from Nantes to Bayonne; the valleys of the Ebro, Tagus, Douro, and Guadalquivir, in Spain and Portugal; almost the whole west and much of the eastern coast of Italy; with Sicily, Sardinia, and parts of Corsica; a great part of Greece and Turkey, with Candia and Cyprus; the plains of Hungary; and the shores of the Black and of the Caspian Seas, with most of the southern provinces of Russia. Nowhere is it so severe as in Italy, especially in the Maremma of Tuscany, the Campagna of Rome, and the Pontine Marshes. The only parts of the peninsula with equally severe agues are the coast opposite Venice and a small district between Feirara and Modena, the shores of the Gulf of Tarento, the south-east corner of Sicily, and the S.E. and W. coast of Sardinia.† What is of especial interest is that in these districts it has greatly increased in extent within the last four centuries. Places which were at one time thickly populated and well cultivated have become waste and deserted; at the same time they have grown malarious.

In Asia intermittent fevers abound, not only in many parts of India, but also in Persia and in China. In Africa the west coast is well known for the malignant character of the remittent fevers which prevail there; and ague is of frequent occurrence in Madagascar, Zanzibar, and in the forests which lead to the tropical interior table-land. On the American continent, the shores of the Gulf of Mexico are those in which malaria is most frequent and severe: but it is well known in parts of Brazil and of Peru, as well as in many of the West Indian islands. It is a remarkable fact that in Australia ague prevails only in the northern part of Queensland; it is absent from New Zealand, from Tasmania, and from the Argentines in South America.

Conveyance by water.—There is some evidence that the contagion of malaria may be conveyed in drinking-water. A case is recorded by M. Boudin of one hundred and twenty soldiers conveyed by the French transport ship "Argo" from Algiers to Marseilles in 1834. In the hurry of embarkation the water which was supplied to these men was taken from a marshy place near Bona. Thereupon all but nine of them became attacked with various forms of ague; and it turned out that these nine, instead of drinking the same water, had purchased wholesome water from the crew of the vessel, all of whom remained well. In two more ships, which made the voyage at the same time, there were six hundred and eighty soldiers, and they too escaped. Dr Manson has met with one similar case in which the microzoon was apparently conveyed in drinking-water. Another instance is that of a farmer's family at Houghton, near Bedford, who drank well water, and who were at one time almost the only persons free from ague in the parish, the other inhabitants of which had only ditch water.

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* 'The Court and Character of K. James,' by Sir A[nthony] W[eldon], 1651, p. 160. Dr Bates gives the following account of the spleen at the autopsy of the Protector:—"In naturalibus fons mali comparuit; liene, licet ad conspectum sano, intus tamen tabe instar amurcæ referto" ('Elenchus motuum nuperorum in Anglia,' 1662, p. 417).

† These districts are graphically indicated in the map drawn up by the Italian Government, which is reproduced by Dr North ('Roman Fever,' pl. v).

Predisposition.—It is well ascertained that *negroes* are as a rule proof against ague; so that black soldiers are invaluable for field service in certain parts of the West Indies. Even if this fact be due to a kind of acclimatisation, transmitted by inheritance, it is not the less remarkable. For, although ague often attacks Europeans who had recently arrived in a malarious district, yet those who have lived there for a longer time often suffer from malarial cachexia, whilst the negro enjoys life and health.

Contagium vivum.—All pathologists since the discoveries of Pasteur and Koch have expected to find ague dependent on a *contagium vivum*, but no serious proof was brought forward of the discovery until, in the spring of 1879, Professor Klebs, then of Prague, and Professor Tommasi-Crudeli, of Rome, as they believed, discovered the bacillus of ague.

They constructed a machine by means of which a large quantity of air could be rapidly made to pass over the surfaces of glass slides moistened with glycerine jelly. This they set to work in the Pontine Marshes and in Rome, taking the air from near the surface of the ground. Subsequent microscopical examination of some of the glass slides showed rod-shaped bodies and delicate threads; and by cultivation these were made to develop into jointed filaments, with spores. Similar structures were found in mud taken from pools in the same region. When this cultivation had been injected in rabbits the spleen was found enlarged after death, and often contained black pigment.[*]

Marchiafava afterwards discovered the spores and jointed filaments of the same parasite in the spleen and in the blood of two persons who had died of malarial fever in Rome.

These results have, however, not been confirmed by other investigation. Dr North, who with great perseverance and devotion followed the investigation of malaria at Rome, arrived at conclusions which widely differ from those just mentioned ('Brit. Med. Journ.,' April 23rd, 1887, p. 865, and 'Roman Fever,' 1896). He was inclined to regard ague as due to a primary derangement of the heat-controlling mechanism of the body, and not as the result of the invasion of microphytes or microzoa. The same negative conclusion was arrived at by Prof. Sternberg at New Orleans (1880).

In the absence of satisfactory evidence of any pathogenic microphytes, it was of great interest when the presence of a microscopic *animal* organism was announced as a constant occurrence in ague.[†]

The Italian physicians, Crudeli, Marchiafava, and Celli, had described and figured remarkable pigment granules in the blood-corpuscles of malarial cases.[‡] Next, Laveran in 1880, and Richard soon after, discovered pigmented amœboid bodies and flagellate organisms within the blood-discs ('Comptes-rendus' for 1882), and named them *Oscillatoria malariæ.* Celli and Guarnieri proposed the name *Plasmodium malariæ*, but this is zoologically inaccurate. Dr Osler confirmed these observations in America, and found outside the blood-discs, crescentic or "falciform" bodies containing granules, and pigmented corpuscles furnished with one, two, or more flagella in active motion ("The Hæmatozoa of Malaria," with figures, 'Brit. Med. Journal,' March 12th, 1887). In Philadelphia, Baltimore, and Washington, American physicians thirteen years ago found the crescentic bodies and pigment so constant that they were a means of diagnosis practically.*

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[*] I must confess that, after reading carefully the details of the experiments in question, and studying the temperature charts which Klebs and his Italian coadjutor give, I fail to see that there is anything definite or characteristic about the fever which occurred in their animals after injection of fluids containing the so-called *Bacillus malariæ*.—C. H. F. (1880).

[†] Dr North quotes ancient belief that malaria was due to animalcula in Varro's words ('De Re Rustica,' i, 12), *Crescunt animalia quædam minuta.*

[‡] See Professor Crudeli's paper, with drawings of blood-discs after Marchiafava and Celli, in the 'Report of the International Medical Congress at Copenhagen,' vol. ii, p. 23.

Independently of these observations in man, flagellate organisms have been described in the blood of mules in India suffering from a serious disease known in the Punjab as "Surra," by Dr Griffith Evans and Mr Steel (in Burma), and also by the late Dr Timothy Lewis in Indian rats ('Quart. Journ. Micr. Sci.,' 1879 and 1884). Dr Crookshank has found the same organism in the blood of rats in England ('Journal Roy. Micr. Soc.,' 1886). The Indian species were *Mus decumanus* and *M. rufescens*; the organisms in their blood (named Herpetomonas by Saville Kent in his 'Manual of the Infusoria') have been compared to the *Spirillum Obermeieri*, to spermatozoa, to Gaule's 'Cystozoa' in frog's blood, previously described by Lankester (*Drepanidium ranarum*), and to stages of Gregarinidæ or other forms of Leuckart's Sporozoa.†

The specific microzoon.—When the blood of a patient suffering from malarial fever is put under a high power of the microscope, a greater or less number of the red corpuscles are found to contain a minute mass of pigmented protoplasm. This amœba-like organism gradually divides into rosette-like wedges, and these form into globules which are reproductive spores. The red disc bursts, and they are thus transferred, together with free pigment granules, to the liquor sanguinis. The pigment is absorbed by the living cells of the blood and tissues (leucocytes and fixed corpuscles), which act as phagocytes and produce the characteristic pigmentation of malaria. The spores (or some of them) acquire a large, pale, vesicular nucleus with a nucleolus, and attach themselves to the red corpuscles, which they penetrate. Usually there is one only of the nucleated parasites which attack each blood-disc, but occasionally there are two. Here they show active amœboid movements, shooting out numerous pseudopodia, and growing on the globulin of the host, while the hæmatin forms the pigment. When stained with methylene blue, the nucleolus and scanty sphere of protoplasm, with the large vesicular nucleus, look in optical section like a "signet ring." Finally the active movements cease, the nucleus and nucleolus disappear, and the living parasite breaks up into spores as above described.

Two other objects are seen in malarial blood which are very characteristic, although their significance is at present not fully ascertained.

One is the "crescent" or "sausage-shaped" body which is seen attached to, and probably within, a red corpuscle. It is marked by pigment granules, and is believed by Mannaberg to be a *syzygium*, formed by the conjugation of two of the hæmamœbæ.

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* During a visit to the United States in 1888 the writer first saw marked specimens of the characteristic discs in blood taken from patients suffering from ague.

† On this and many other points the reader is referred to Dr Manson's 'Tropical Diseases,' 1898, to Dr Osler's article in 'Allbutt's System,' to Surgeon-Major Ross's papers in the 'Brit. Med. Journ.,' and in 'Nature,' March, 1900, p. 522, and to Thayer and Hewitson's "Malarial Fevers of Baltimore" (the fifth volume of the 'Johns Hopkins Hosp. Reports,' published in 1895), also to Dr Cross's 'Notes on Malarial Fevers of West Africa,' 1892.

The other object above mentioned as occurring in malarial blood is "the flagellated corpuscle." This is a pigmented globule which suddenly throws out long, actively moving threads, called *flagella*, although they are probably not identical with the long cilia of protozoa and protophyta, which are properly so called. These flagellated corpuscles are never seen in the freshly-drawn blood. They gradually appear in a drop under the microscope, and their appearance is hastened by exposure to air and admixture of a little water. They are probably external spores, and provide for the second phase of parasitic life, apparently necessary in addition to that passed in the blood of the human host.

The second host is, as first surmised by Laveran, worked out by Manson, and verified by Ross, a mosquito, which, when it pierces the skin of a malarial patient, takes spores of the hæmamœba into its gastric cavity along with the blood-discs. It is supposed that the larvæ and the shallow pools and puddles they inhabit are thus infected from the mosquito's body,* and that the coccidia or sporozoa thus transferred to mosquito larvæ and to the water they inhabit are the phase in which the contagium re-enters the body. The method of entrance is probably most often by an infected mosquito imago biting a healthy man, as a rabid dog's bite transfers the virus of hydrophobia; but it is also probable that, either in drinking-water or dried up in air, the parasites may gain an entrance to a human body without again passing through an insect host.

The mosquito in question is a kind of gnat belonging to the genus *Anopheles*, and distinguished by its proboscis and slender, sharp anterior part of the body, by the large angle it makes with the wall or other vertical surface on which it settles, and in most individuals by spots upon its wings.

It remains to state that following up the original observations of Golgi, in 1885, numerous researches have established the fact that the malarial parasite appears in different forms in malarial fevers of different kinds. In the milder winter and spring forms of quartan and tertian fever there are no "crescents" found, and the cycle of development occupies three days in the former, and forty-eight hours in the latter. In the more severe summer and autumn tertians of Italy "crescents" occur, as also in the quotidian fevers of the tropics, where the parasite completes its cycle in the blood of the patient in twenty-four hours. In the latter annular forms also occur; the spores are more minute than 1 μ in diameter, and some of the hæmatozoa are stated by Marchiafava to be unpigmented. Ross calls the parasite of quartan fever *Hæmamœba malariæ*, that of tertian *H. vivax*, and that of remittent *H. præcox*.

Although it has not yet been possible to "cultivate" these microzoa like parasite microphytes, yet there is reason to believe that each of the above varieties "breeds true." Moreover, beside these being present in all cases of malaria, and never being found in the blood of persons unaffected by it, there is correspondence between the cycle of the parasites in the blood and that of the fever in the patient; and, lastly, while they disappear when the fever subsides on the second, third, or fourth day, they also disappear when quinine cuts short the series of clinical events.

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* The process has been actually traced by Ross in the allied *Proteosoma*, and by McCallum of Philadelphia in the *Halteridium*, both of them parasitic coccidia which infest the blood-discs of birds.

The destruction of hæmoglobin by the parasite explains the anæmia of ague, as well as the frequent pigmentation of the spleen, liver, and other parts of the body, which is now referable to the pigment granules contained in the diseased blood-discs.*

We must now separate the intermittent and remittent fevers widely from the other specific fevers in which a bacterial origin is proved or probable, and include as a natural group not only ague in all its varieties, but bilious remittent and malignant malarial fevers, hæmogloblinuric fever, tropical dysentery, and tropical abscess of the liver. We have also learnt that tertian, quartan, and quotidian agues have corresponding differences in the form or stage of development of the plasmodium, and are thus pathologically as well as clinically distinct.

We can now explain why moisture is necessary for malaria, since the *Anopheles* lays her eggs and hatches them in shallow pools and puddles, not in deeper water, and not like the allied genus *Culex* in artificial tanks, waterbutts, and basins. The geographical distribution of malarial fever closely corresponds with that of mosquitoes, although there remain some seeming exceptions, one of which is the prevalence of ague in former times in England.

Lastly, the beneficial effect of quinine in all forms of malaria is now explained by its poisonous action upon the hæmatozoa.

Two points of practical importance are still uncertain. One is whether the chief or sole channel of entrance of the microzoon is by the bite of the infected mosquito; or whether the contagion from the dead mosquitoes or dried-up larvæ may sometimes be conveyed to the alimentary canal by drinking-water, or to the throat and lungs by air.

The other question is whether the true mosquitoes of the genus *Anopheles* are the sole conveyers of the hæmatozoa in the case of man, or whether other allied *Culicidæ* may take part in the production of malarious disease, as they undoubtedly do in the case of other animals than man, and other *sporozoa* than the *hæmamœba.* If it should prove to be so, the several kinds of malarial fever, and the several kinds of *hæmamœbidæ* which correspond with them, might perhaps have each a corresponding species of *Culex* or *Anopheles* for its conveyance.

The succession of symptoms in the paroxysm of ague must be regarded as analogous to that observed in cases of suppuration, in the rigors of pyrexia and sweating of pyæmia. Like an epileptic seizure, the ague-fit has a physiological basis. Its recurrence at the same hour on successive days, or every second or third day, is, as Cullen originally surmised, connected with the diurnal habit of body which is so plainly manifested in both health and disease. One, therefore, need not wonder that Griesinger and Duchek failed to modify the times at which ague-fits returned by changing the patient's hours of meals and his other habits of life.

Diagnosis.—The recognition of ague is for the most part easy, but it may present difficulties. In places which are free from malaria there is sometimes risk of the real nature of a patient's illness being overlooked,

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*** The white corpuscles are found almost always to be diminished in number during the presence of malarial fever or cachexia. See Rogers, 'Journ. Path. and Bact.,' December, 1898, p. 402.**

when he has acquired the disease elsewhere, perhaps while travelling in a country where its existence was unknown to him.

In regions notoriously malarious the physician has constantly to keep in mind that ague is apt to complicate other diseases, but may sometimes simulate them. Moreover there is danger of carelessly mistaking for results of malaria any recurrent rigors or paroxysms of febrile disturbance. Thus Graves relates an instance in which the intermittent hectic of *phthisis* was set down as ague. Another disease which is very apt to be overlooked in malarial districts is *pyæmia*. In reference to this, Hertz remarks that the pyæmic rigor generally sets in more gradually than that of ague; that its subsidence is more rapid than its onset, whereas in ague the reverse is the case; and, lastly, that in pyæmia there is very seldom an interval of complete apyrexia, lasting for twenty-four or even for twelve hours. But, as we have seen, ague itself may be merely remittent. The fever attending *internal suppuration*, particularly of the gall-ducts, or that of ulcerative *endocarditis*, may also be attributed to malaria.*

The remittent forms of malarial fever have often been confounded with *enteric fever*. Thus in Romney Marsh no kind of fever used to be recognised except that due to the endemic malaria; and in many parts of India it is only of late years that enteric fever has been recognised. "Typho-malarious fever" is probably enteric fever. The *fièvre bilieuse hématurique* is not always easily distinguished from yellow fever in countries where both diseases prevail.

The most important points for diagnosis used to be the periodicity of the symptoms, and the curative effect of quinine. To this we can now add the more certain and, in skilled hands, the easy recognition of the characteristic appearances in the blood-discs due to the hæmatozoon.

Prophylaxis.—The empirical rules which were laid down as the result of observation for avoiding malarial fevers in the south of Europe and in the tropics still hold good. The importance of wholesome food, pure water, or water purified by boiling, extreme temperance, avoidance of over-fatigue, or exposure to sudden chills, and the value of comparatively cool and dry spots for camps and houses, are explained by the same consideration as has frequently been mentioned before, that there is a certain power of resistance—or, to put it more definitely, of phagocytosis, by which the organism protects itself against the invasion of disease; and this applies to microzoa as well as to microphyta.

Again, the advantage of draining, and the protection afforded by growing plants and trees, is now intelligible.

But we are now able to advise more direct and definite measures to protect against the contagion. All shallow pools and puddles in the neighbourhood of houses should be filled up. Any small collection of water should be searched for larvæ: those of *Culex* hang downwards from the surface, and when disturbed sink; those of *Anopheles* float on the surface, and when disturbed wriggle away without sinking. They may be destroyed by emptying and brushing out all the receptacles of water, or by pouring kerosene or tar over pools and puddles. Mosquitoes are torpid during the day, and may be killed as they are found on walls; and mosquito-curtains should be always used at night.*

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* I once saw a case in which a medical man was firmly convinced of the presence of ague, until his patient voided a quantity of horribly fœtid pus with his urine, after which a rapid recovery took place.—C. H. F.

Lastly, the benefit of quinine as a prophylactic is not only attested by long and wide experience, but is now confirmed by its "germicide" effect. Persons passing through a malarious district should fortify themselves against infection by taking daily doses of sulphate of quinine; and whenever the first sign of malarial cachexia appears, the inhabitants of such districts should have recourse to the same drug.

Treatment.—Ague and other forms of malarial cachexia are happily amenable to the influence of a drug which acts in what used to be called a specific manner,—*i. e.* certainly and without any obvious physiological means.

The remedy was introduced into Europe in the seventeenth century by the Jesuit missionaries in South America, and was named Cinchona in honour of the wife of the Spanish Viceroy of Peru, the Countess Cinchon, who took an active part in promoting the use of the drug. Jesuits' bark, as it was first called, is taken from several trees of the natural order *Rubiaceæ* and the genus *Cinchona*—*C. succirubra*, red bark; *C. calisaya*, yellow bark; *C. officinalis*, pale bark; *C. lancifolia*, Columbian bark. They grow in the Andes of South America, in Jamaica, and have been successfully transported to India, where they flourish in the hill country.

The powdered bark is bulky and unpleasant to take, and the decoction and infusion and liquid extract are equally efficacious. But it is in every way better to give the pure and concentrated quinine: and of late years the synthetical production of this compound has greatly diminished its cost.

As soon as possible a full dose of quinine—10, 15, or 20 grains—should be given from one to two hours before the expected onset of the paroxysm. During the cold stage, if it has not been entirely prevented by the first dose of quinine, the patient should be covered up warm and drink freely of hot beverages. In the hot stage, Dr Crosse advises phenazone ("antipyrin"), cold sponging, and cool drinks. If the sweating does not come on kindly, hot liquids may again be taken and pilocarpine injected. At the end of the sweating stage the patient should be carefully dried and have a change of linen. He may then get up, but if he afterwards goes out of doors he must be very careful not to take cold.

The quinine should be repeated during the interval, until an hour or so before the next fit is expected. Maclean advises that the patient should never have less than thirty grains during this period. It is best given in solution with a little sulphuric, hydrochloric, or hydrobromic acid. Pills containing quinine are apt to become hardened by time, so as to pass through the stomach undissolved. If vomiting should occur, the quinine may be administered *per rectum* in a dose of fifteen grains suspended in thin starch.

When the stomach is loaded, an emetic is often useful, and if there is constipation a purgative; indeed, many experienced physicians advise a single dose of calomel and colocynth, followed by a saline draught, in every case at the onset; but it is a mistaken practice to put off the use of quinine until the tongue has become clean, and Sir Joseph Fayrer says that after a first dose calomel is better omitted.

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* A full account of the practical methods of carrying out these indications is given in a useful manual of 'Instructions for the Prevention of Malarial Fever,' issued by the School of Tropical Medicine, recently established at Liverpool (1899).

If in the cold stage symptoms of collapse threaten, recourse should be had to strong brandy, coffee, ammonia, or ether.

It very rarely happens in recent cases of ague, when quinine is properly given, that the patient has any subsequent attack of the same severity as before; and within a few days, or in a week or two at latest, they almost always cease. But in certain cases, especially those of long standing, Indian physicians used to prescribe Warburg's tincture as a diaphoretic and febrifuge.*

The writer has found Fowler's solution particularly useful in *neuralgia*, anæmia, and other sequelæ of malaria when a patient has returned to England after a long period spent in India, China, or Madagascar.

For the dangerous *remittent* and continuous forms of ague, and for those anomalous cases which simulate other diseases, quinine is still the remedy, and it must be employed boldly. Maclean insists on the importance of watching for even a slight remission, using it as an opportunity of giving fifteen or twenty grains of quinine by the mouth, and repeating the dose in two hours' time.

The *malarial cachexia* requires the administration of iron as well as quinine, but even these remedies are of secondary importance in comparison with the removal of the patient to a healthy locality.

Maclean speaks strongly of the value of the ointment of red iodide of mercury in the treatment of chronic enlargements of the spleen and of the liver. He directs that a piece of the size of a nutmeg should be rubbed into the hypochondriac region, and that these parts should be then exposed to the sun or to the heat of a fire. The writer has tried this plan with apparent benefit.

In *blackwater* fever Dr Crosse advises a free purge to begin with, followed by quinine and stimulants. To check vomiting, a mustard plaster should be laid on the pit of the stomach. If there is suppression of urine he prescribes mustard to the loins, diaphoretics, jaborandi, and chloral hydrate, with bicarbonate of sodium to diminish the acidity of the urine. During convalescence a sea voyage is the most valuable treatment, but iron, arsenic, and nitro-hydrochloric acid are also useful. Like other physicians of experience in tropical Africa, he entirely rejects Prof. Koch's somewhat hasty assertion that the hæmaturia of Blackwater fever is merely the result of treatment with overdoses of quinine.

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* Dr Hale White gives its composition as proof spirit with quinine, aloes, rhubarb, opium, and camphor, together with various aromatic herbs as saffron, fennel, elecampane, angelica, cubeb, and myrrh, to which are sometimes added zedoary and white agaric.

RABIES OR HYDROPHOBIA

"Hydrophobiam Græci vocant: miserrimum genus morbi."—CELSUS.

History—Course: incubation—symptoms—The contagion—Rabies in animals—Distribution—Anatomy—Pathology—Diagnosis—Prevention and treatment by an attenuated virus.

Synonyms.—Rabies canina—Lyssa (λύσσα, raging madness).—*Fr.* La rage.—*Germ.* Hundswuth, Wasserscheu.

Definition.—A specific infective disease, communicated to man by the bite of animals, with a long incubation, symptoms referable to the nervous centres, a short course, and a grave prognosis.

History.—From the time of Aristotle it has been known that dogs are liable to a fatal disease which they transmit by their bite; and this disease when occurring in man, was called "Hydrophobia," from the dread of water which is sometimes one of its symptoms. In most of the lower animals that symptom is absent, and in them it is called "rabies," *i. e.* madness. But though the names are two, the disease is one and the same; and rabies is the better term.

Although the symptoms of this terrible disease are those of paroxysmal nervous convulsions, its natural place is among infective diseases, either near Tetanus, which also is in its symptoms a neurosis, or near Glanders and Anthrax, which also are transferred from animals to man.

Incubation.—After the healing of the wound inflicted by the teeth of a rabid dog, a period of incubation follows, which is of uncertain duration, and often prolonged beyond that of all other infective diseases. Bollinger, in Ziemssen's 'Handbuch,' says that in 60 per cent. of all cases in the human subject it is between eighteen and sixty days, in 6 per cent. between three and eighteen days, and in 34 per cent. longer than sixty days. How protracted it may be we cannot positively tell. Instances have een recorded in which three years, five years, and even more were suposed to have elapsed. It is possible, however, that in these cases the ue infection took place subsequently, and without the patient's knowing t. Mr Youatt succeeded in tracing certain cases to sources that might asily have been overlooked: one, in a man, to his having tried to untie ith his teeth a knot in a cord by which a rabid dog had been confined; nd another, in a woman, to her having used her teeth to press down the eam in mending a tear in her dress caused by the bite of a rabid animal.

The following are the results of inquiries as to the period between the ite and the appearance of symptoms in cases admitted to Guy's Hospital.

Six were published by Dr Bright (1820-30). Seven more were reported by Mr Cooper Forster in the 'Guy's Hospital Reports' for 1866 (Third Series, vol. xii, p. 1). The incubation in these thirteen cases varied between four weeks and eleven months, except one in 1854, which occurred in the practice of Dr Hughes, in which it was supposed to be from five to seven years. In ten cases which have occurred since the publication of that report (1867-89), collected for the third edition of this book by Dr E. W. Goodall, the periods of incubation were nineteen weeks, thirty-five days, thirty-eight days, forty days, nine weeks, nearly twelve weeks, thirteen weeks, and about twelve weeks respectively, and, more doubtfully, four weeks; while in the tenth case (a man aged thirty-six, who died in 1878) no certain evidence could be obtained, either from the patient or his friends, that he had ever been bitten by a dog. No doubt the period of incubation varies more than in other specific diseases, but in all the most authentic and well-observed cases it is more than a month and less than a year.

In 132 cases of rabies selected by the Registrar-General (1886) on account of the circumstances being accurately known, the shortest incubation was eleven days, in a child bitten by a rabid cat. In 23 cases it was under a month, in 64 between one and two months, in 21 between two and three months, in 124 it was under five months, in 127 under ten months, and in 130 under two years. In one case it was supposed to be between three and four years, and in one other above four years.

The most frequent incubation period is about six weeks. It is never less than three weeks and seldom more than three months. When the infecting wound is on the face the incubation is probably shorter. In children also it is usually shorter than in adults.

Experimental inoculation in dogs, rabbits, and other animals shows, on the whole, shorter incubation than when the disease dates from the infliction of a bite by a rabid animal; and when the virus is introduced not subcutaneously, but beneath the dura mater after trephining the skull, the period of incubation is measured by days, a week being a very frequent time.[*]

Prodroma.—In some cases the earliest indication of the onset of rabies is afforded by an uneasy sensation in the seat of the bite, which becomes painful, or tingles, or itches, or feels cold; sometimes the cicatrix may be seen to be reddened, livid, or swollen. The pain or other morbid sensation extends upwards along the nerves from the hand into the arm and up to the shoulder, as in a coachman whose case is recorded by Sir Thomas Watson.

In a gentleman who came under the care of Mr Cooper Forster in 1866 the pain was of extraordinary severity: it came on in paroxysms, obliging him to stop suddenly in the street, and to cry out; it was not referred to the course of any particular nerve. There was in that instance no redness or tenderness of the cicatrix. The arm, however, felt much colder than the other one. He had been bitten eleven months previously, and in the intervening time he had three or four times complained of pain up the arm and twitchings in the hand.

Other early symptoms are restlessness, irritability, and depression of spirits. The patient suffers from nausea and loss of appetite; he complains of headache, is sleepless, and has a distressing sense of apprehension. It is curious that he often says nothing about having been bitten, or may vehemently deny it; and yet he may make other remarks which show that his mind is dwelling on the subject, and that he is trying to persuade himself that he need not be afraid.

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[*] See Horsley's lecture before the Epidemiological Society, Feb. 13th, 1889 ('Brit. Med. Journ.,' p. 342), and Bradford's Brown Lectures ('Lancet,' February, 1900).

Often these premonitory symptoms are absent. In that case, the first characteristic symptom is a repugnance to fluid. Sometimes there is difficulty in swallowing, from a feeling of tightness about the throat. Sir Thomas Watson's patient, the coachman, refrained on account of a similar sensation from sponging himself as usual with cold water, though he remarked that he "could not think how he could be so silly."

Symptoms when developed.—The above prodromal or early stage generally lasts from one to three days, but sometimes it is altogether absent. In either case the full development of the disease is ushered in by the sudden occurrence of violent convulsive paroxysms, affecting the muscles of deglutition and those of respiration, and repeated at frequent intervals. They are excited in many ways. The attempt to drink almost always causes a fit; the sight of water in a basin often has a similar effect. Watson mentions a case in which the patient was thrown into a violent state of agitation by hearing a dresser, who was sitting up with him, pass urine. The least draught of air, the waving of a mirror before the eyes, the opening or shutting of a door, the slightest touch upon the skin, the attempt to comb the hair, may excite an attack. Sometimes the patient succeeds in gulping down a little fluid by carrying it to his mouth with the eyes shut, or he may insist on being left alone in order to swallow better. Watson saw one man who so dreaded anyone's breath on his face that he would not converse with the apothecary of the hospital except in such a position that the chin of each of them rested on the other's shoulder. The fits often consist of a series of shuddering or sobbing movements, more or less like those which occur when one steps into a cold bath. Sometimes, however, they are attended with tonic spasms of the cervical muscles.

The following description of the more violent paroxysms was drawn up by Dr Bushell, now Physician to the British Legation at Pekin, from a case which occurred while he was dresser to Mr Cooper Forster. "At the onset of a severe spasm the patient springs up in bed, and puts his hands furiously to his throat, as if to tear something away; the head is thrown violently back, the mouth is opened, and the eyeballs are protruded; then he makes several expiratory efforts, sometimes with a shrill screaming cry; the head is thrown violently from side to side; the hands are tossed wildly about, beating his chest, and striking anything that is near." Sometimes the jaws are sharply brought together, so that credulous bystanders think the patient is trying to bite them. His barking like a dog is no doubt a fabulous addition. Bollinger denies that trismus ever occurs, or a general tonic spasm like that of tetanus. Nor is there an authentic case in which opisthotonos was present. Sometimes these convulsions seem to be spontaneous, but often they can be recognised as reflex spasms from a sensory excitation. The skin is hyperæsthetic, particularly to cold.

The duration of the paroxysms is variable. According to Bollinger, they occasionally last from thirty to forty-five minutes. When they are protracted the patient's face becomes pale and livid, and is covered with sweat. The countenance assumes a look of terror, and the pupils are widely dilated. The pulse is rapid, the respiration also quickened, irregular, and more or less forced.

The temperature varies greatly. It is, as a rule, slightly raised, sometimes normal, and occasionally rises to 103° or even 105°, as in a case noted by Mr Southam, of Manchester ('Brit. Med. Journ.,' 1881, vol. ii, p. 814).

The urine appears to be usually unaffected; sometimes febrile, rarely albuminous. The occasional presence of a reducing agent is perhaps due to inhalation of chloroform.

In women and young children the course of rabies is often comparatively mild, although it is even more quickly fatal than in men; this was so in two of Sir Thomas Watson's cases, one in a lady aged thirty-two, the other in a girl only five years old; also in a little girl who died in Guy's Hospital in 1875, who was so slightly affected that Mr Stocker supposed her to be only hysterical. In other cases, however, children affected by hydrophobia exhibit the most frantic mania.

There is an inability to swallow even during the intervals of the fits. The patient, who is much distressed by thirst, may resolutely carry the glass to his lips, but as soon as a little water has entered his mouth a sudden spasm of the pharynx throws it out again. A viscid saliva which collects in the mouth is a source of great annoyance; it is hawked up with noisy effort, and spat out upon the floor in all directions. Another symptom, only present in certain cases, is priapism, with frequent involuntary emissions. Trousseau observed painful hyperæsthesia of the genital organs.

The mental state between the convulsive attacks is one of great agitation. As the disease advances the patient raves at those around him, and sometimes has to be forcibly held down. Not infrequently death occurs by suffocation in the middle of a paroxysm, and more or less suddenly.

The whole duration of the disease is seldom a week, and sometimes it terminates within from twelve to forty-eight hours. In eight cases death occurred in twenty-four hours, in eighty-seven between the first and th seventh day, in five on the eighth, and in one case on the ninth, tenth and twelfth days respectively (Registrar-General's 'Report,' 1886).

Sir Thomas Watson's coachman did not die until the middle of th seventh day. His end was very gradual; the pulse grew rapid and weak the mental powers failed, he lay moaning and tossing from side to side frothy saliva ran from his mouth, he lost the power of moving his lef arm, liquid stools were passed involuntarily, the lower extremities gre cold, and the coldness extended up to the chest. Some patients hav towards the last become paraplegic, as we shall see rabbits do. In th more protracted cases the spasms may cease for some hours before deat the patient be calm and able to talk or drink or wash his hands witho discomfort; but nevertheless the coldness of his surface, and the absence pulse at the wrist, show that there is no real improvement.

Contagion from rabid animals.—Rabies in man is doubtless caused all cases by the transference of specific virus; but it is not complete known or determined whether the disease is ever, under any conditio communicated from one human being to another. No case is known of t kind, and medical men and nurses always escape, although the poisono saliva is often ejected on their clothes and hands and faces. In form times the dread of catching the disease was so great that the patient w sometimes smothered between feather beds, or was allowed to bleed *death* through an opened vein. Bollinger says that on the military fronti of Austria persons labouring under rabies, or suspected of it, were liable to be shot by their neighbours, and that those who have been bitten by rabid dogs sometimes commit suicide.

There is no doubt that the virus resides in the saliva and salivary glands, and there appears to be little doubt that it is present during the incubation period in a dog. Magendie long ago produced rabies in dogs by inoculating them with the saliva of hydrophobic patients. Pasteur has now proved that the spinal cord is also the seat of the virus; and that inoculations from it, especially if introduced under the dura mater, after trephining the skull, will reproduce the disease in dogs and rabbits.

The disease in animals.—Rabies may occur in many kinds of animals beside dogs. It is common in wolves, jackals, and foxes, and the bite of a rabid wolf is notoriously the most dangerous of all. The skunk is said to be very liable to rabies. Cats are sometimes affected by it, but far less frequently than dogs. A scratch from a cat is believed to have conveyed it to a child, no doubt from the claws having been moistened with her saliva. Among herbivora, oxen, horses, goats, sheep, pigs, rabbits, and guinea-pigs are capable of being infected by inoculation, or if they are bitten by rabid dogs. Youatt recorded a case in which a groom took hydrophobia through a scratch which he received from the tooth of a rabid horse.

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Rabies broke out as a destructive epidemic among the fallow-deer in the Royal park at Richmond in 1889, and soon afterwards in the Marquis of Bristol's park. More than 450 died out of a herd of between 600 and 700 in the course of three months.*

A most important question is what indications should lead one to decide, when a person is bitten by a dog, that the animal is rabid. Mr Youatt met with cases in which a dog exhibited no symptom of rabies when it inflicted the fatal bite, though it was soon afterwards attacked. No fewer than eighteen or nineteen instances of this kind have been collected. They seem to show that the disease is infective even during its period of incubation, which in dogs is believed to be generally of from three to five weeks' duration, but occasionally to be prolonged over as many months. It is evident, therefore, that when a dog is killed soon after having bitten a human being, one can never be quite sure that hydrophobia may not supervene. The proper course is to keep the animal securely confined for some weeks.

There are two varieties of the disease in dogs, though the symptoms often coincide in the same case; one characterised by maniacal excitement, the other by paralysis of the jaw, so that it hangs down and allows a frothy saliva to run out of the mouth. In each form the bark is altered into what is described as a "hoarse inward sound, dissimilar from its usual tone, and generally terminating with a peculiar howl." Towards the last the hind legs and the loins become paralysed, so that the dog staggers about and falls. Thus the popular distinction between ordinary rabies and "dumb rabies" with paralysis is not without foundation.

One of the earlier symptoms is an extraordinary perversion of the appetite, the animal eating hair, straw, sticks, bits of string or leather, earth, stones, and other substances, which may be discovered in the stomach after death, mixed with brownish mucus. Their presence often affords a valuable indirect proof that a dog which has been killed under the suspicion of rabies was really affected with that disease.

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* See Adami's interesting account in the 'Brit. Med. Journ.' for Oct. 12th, 1889.

The coat is rough, the dog refuses food, is restless, melancholy, and slinks away by itself. Change of disposition and of voice are also striking indications of rabies in a dog.

As already remarked, a dread of water is not a symptom of rabies in any animal except man. "Mad dogs," as they are commonly called, plunge their muzzles into water and lap it up eagerly, for they are very thirsty, although they may not be able to swallow.

Rabies is always fatal in dogs—usually within a week after the symptoms have appeared, occasionally after nine or ten days. The following case is taken from the 'Guy's Hospital Gazette,' January 30th, 1897:

> Coming home one afternoon, I was told that Kenny (a well-bred Irish terrier) had just swallowed a piece of string and run out. I followed him, and found him eating some geraniums. Remembering this habit as a symptom of rabies, I called him and locked him up in the yard where his kennel was, gave him food and water, and forbade anyone to go near him. On inquiry, the servants said that all that morning he had been dull and stupid, and had often rubbed his face on the floor. More important was the fact that he had been bitten in the ear by a strange dog three weeks and three days before.
>
> Next day he would not eat, but drank water and recognised me.
>
> On the third day of his illness his constant salivation, moping, and refusal of food made me determine to take him at once to the Brown Institution in the Wandsworth Road. The muzzle, which did not fit well, he soon worked off, but he sat quietly between my knees, and did just as he was bid. Before we arrived at our destination I noticed that his jaw began to droop. He followed me into the yard, showing no excitement, but great depression, like a dog tired out, or one that had been beaten. Thankful I was when I saw him safe in a large iron cage with a good padlock. Dr Sherrington was not there at the time, but the man who received him, and who had seen many cases of hydrophobia, said he felt sure that was the disease.
>
> I was not able to go to see him next day, but the one following, the fifth of his illness, I did so, and found him greatly changed. His beautiful coat was rough and staring; his jaw fallen and dripping with saliva. He could not bark, but howled in a most distressing way, and instead of coming to be made much of as usual, he rushed furiously at the bars of his cage like a wild beast.
>
> I was told that there was now no doubt at all of the diagnosis, and that he would probably live for another day, perhaps for two. There was no object in prolonging his life, and he was put painlessly out of his misery with a drachm of Scheele's hydrocyanic acid.

In *rabbits* the symptoms of rabies (transmitted from dogs) are like those of dumb madness, in the absence of excitement and the development of paraplegia, which, as in dogs, takes the form of "acute ascending paralysis." A man who was bitten by a rabid cat in 1886 died under Dr Bristowe in St Thomas's Hospital with symptoms of this kind.

The study of the disease when reproduced by inoculation in animals shows a very similar series of symptoms to those which are characteristic in man. There is first a stage of excitement with visual delusions; then hyperæsthesia with reflex spasms; next the stage of mania, and (particularly in rabbits) paraplegia, corresponding to the "dumb rabies" of dogs; and lastly, death, often by sudden syncope.

Distribution.—At one time rabies was supposed to occur chiefly in temperate climates, but this is not the case.* The only part of the world in which it is as yet unknown is Australia. Like other specific diseases it is often absent from a town for several years together, until some accident introduces it and it becomes epidemic. Extensive epidemics occurred in the west of Europe in 1720-23, in England in 1754-62, in Malta in 1847, and in Shanghai in 1867. It was much more common in London before the muzzling order was enforced and after it was given up.

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* The following statement of the deaths from hydrophobia which occurred in the Punjab is taken from a report by Brigade-Surgeon Bellew:—Population of the province, 17,514,978. Deaths from hydrophobia: in 1879, 69; 1880, 107; 1881, 139; 1882, 128; 1883, 117; 1884, 158; 1885, 146.

Between 1820 and 1830 six cases of human rabies occurred in Guy's Hospital. Two occurred in 1831, one in 1837, and then none for nearly twenty years. In 1856 there was a single case admitted, in 1865 two, and in 1866 the case of a private patient of Mr Forster's was alone recorded. Then again there was a pause for several years, the next case occurring in 1874. In 1875 another was admitted, three in 1877, and one in 1878. After five years' interval there was one case in 1883, two in 1885, and one in 1886. None were admitted in 1887-8-9, and two 1890—1900.

For London, the Registrar-General's returns show twelve deaths from hydrophobia in 1838, and four in 1839. Then only one, three, four, two, three, two, in the successive years to 1845, none in 1847, '49, and '52, and only one in 1846, '48, '50, '51, and '53. Seven were returned in 1854, and two in 1855 and '57. None in 1856 and none in 1858, '59, '60, '61, and '62. Two in 1863, and none again in 1864, but nine in 1865, and six in 1866. There were three in 1867 and '69, none in '68 and '70; one in 1871, '72, and '73. Then there were nine in 1874, six in 1875, six in 1876, and sixteen in 1877; five in 1878, two in 1879, three in 1880, five in 1881, four in 1882, eight in 1883, nine in 1884, and in 1885 no less than twenty-seven. In 1886 the number suddenly fell to nine—after muzzling was enforced,—and in 1887 and 1888 there was not a single death. In twenty years (1869-88) there were 780 deaths in England and Wales.

In England, the greatest number of cases of rabies occur in Lancashire, Cheshire, and the West Riding of Yorkshire, and the next greatest number in London and the home counties.

According to the popular belief, the disease is more frequent in the hot season than during winter and spring. Of 132 cases, throughout England and Wales, fifty-one occurred in July, August, and September.

Frequency.—Only some of those who are bitten by rabid animals are attacked by the disease; several writers agree in fixing the proportion at about 50 per cent., but this is certainly far too high. Inasmuch as the disease if left to itself is, so far as is known, always fatal, we may take the mortality among persons bitten by rabid dogs as indicative of the frequency with which the disease is developed. The statistics in the department of the Seine for 1887, reported by M. Dujardin Beaumetz, and those of the county of Lancaster for the same year very nearly agree: in and near Paris there were 7 deaths out of 44 bitten; in Lancashire 6 deaths among 36 bitten, or about 16 per cent.

Possibly some persons escape from an idiosyncrasy which renders them insusceptible of the virus. But a much more certain protection is the clothing, by which the teeth are wiped clean of the virus before they penetrate the skin. The undoubted fact that rabid wolves are more dangerous than dogs may be due to their flying straight at the naked throat. Youatt and all modern writers are agreed that a breach of the cutaneous surface is necessary to allow of the entrance of the poison. Mucous membranes may perhaps be capable of infection without any previous lesion.

The large experience obtained at the Pasteur Institute in Paris has amply confirmed Mr Forster's belief that bites on the face are the most fatal, and probably they have the shortest incubation. Next come the hands, and *lastly the parts habitually* covered.

Mr Youatt thought that the virus does not retain its powers after the death of a rabid animal; but Bollinger cites the case (happily unique) of a student at the Veterinary College at Copenhagen, who opened the body of a dog that had died of rabies the night before; his finger was slightly fissured at the time, and about six weeks afterwards he died of hydrophobia. Professor Sherrington tells the writer that at the Brown Institution it was found that the poison resisted putrefactive changes even in hot weather.

Seat.—Until lately it was supposed that hydrophobia, like other neuroses, had no morbid anatomy, at least so far as the nervous centres are concerned. But Allbutt in 1872 ('Path. Trans.,' xliv, p. 16), Benedikt in 1875 ('Virchow's Archiv' for that year, p. 537), Gowers ('Path. Trans.,' 1877), Coats ('Med.-Chir. Trans.,' 1878), and Ross ('Path. Trans.,' 1879), and since Babes and Woodhead, have found that the vessels in the bulb towards the floor of the fourth ventricle are surrounded by masses of leucocytes within their sheaths, and that collections of these exuded cells, forming miliary abscesses, may occur among the nervous elements. Similar changes, but less marked, were also found in the spinal cord. They do not occur in all cases, for in some beautiful preparations made by Dr Frederick Taylor no deviations from the normal appearances are to be recognised; but preparations made from the patient who died in Guy's Hospital in 1885, by Dr Hale White, showed unmistakable extravasation of leucocytes, and here and there of blood-discs, in the sheaths around the vessels in the bulb. In the brain similar lesions have been found. They are probably secondary effects of the paroxysms of the disease rather than their cause.

Experimental evidence confirms the belief that the central nervous system, cord, bulb, and cerebrum, is the seat of the disease. The poison of rabies is also present in the salivary glands and pancreas, but not in the blood or urine.

Other conditions, more or less constantly found after death, are congestion of the pharynx and epiglottis, of the stomach with hæmorrhagic erosion, and of the lungs. They are more uniform in rabbits than in human beings.

Pathology.—There can be no reasonable doubt since Pasteur's experiments, to be presently described, were made, that rabies is a specific contagious disease, an intoxication with an animal poison which exerts its chief effects on the central nervous system. The microbe on which it probably depends has not yet been discovered.

It is still a question what becomes of the virus during the prolonged and indefinite stage of incubation; and what new process gives rise to the first characteristic symptoms. Some writers suppose that a "recrudescence" takes place, the poison having hitherto been imprisoned in the wound, but being at this time absorbed in the blood. If, however, a dog can communicate the disease during the period of incubation, the theory of recrudescence must be given up.

Diagnosis.—Rabies is not generally difficult to recognise. There has been no doubt as to the nature of any of the cases, all of them fatal, that have been observed at Guy's Hospital within the last few years. One of the oddest notions is that there is really no such disease, and that all who are supposed to have died of hydrophobia have really succumbed either to tetanus or to fright. This idea seems to have been started early in the century by Bocquillon; it was upheld by Sir Isaac Pennington, Regius Professor of Physic at Cambridge, and more recently by Prof. Maschka, of Prague. But, as Sir Thomas Watson long ago remarked, young children and idiots, who could not have understood anything about the disease, have died of it, and many of the adults who have been attacked have been men of strong mind, who refused to believe that they were seriously ill. As for tetanus, the symptoms are altogether different.

It must, however, be admitted that medical literature contains several cases, of which it is impossible to say whether they were true rabies or not. The difficulty is with regard to those in which recovery took place. For all experience shows that whenever the clinical characters of the disease are unmistakably present, the patient dies. In other words, nearly every patient who escaped seems to have presented some feature or other which casts a doubt on the genuine nature of the case. For example, a case was recorded by Dr Nicholls, of Chelmsford, in the 'Lancet' for 1878, of a carter, aged twenty-five, who was bitten by a stray dog, and after violent spasmodic and maniacal symptoms, with opisthotonos and delusions, finally recovered.

The "dumb" or paralytic form of the disease closely resembles Landry's paralysis, as in Dr Bristowe's case.

In some cases of epilepsy, mania, or hysteria there is spasmodic difficulty in swallowing liquids, which may be more or less like that which occurs in rabies. But any doubt is dispelled by the progress of the disease. Hysteria is probably the explanation of some paradoxical cases of recovery.

In dogs rabies can, as a rule, be readily recognised by its symptoms: a *post-mortem* examination is less certain, but usually sufficient; the decisive test is inoculation of another dog or a rabbit.

Prophylaxis.—If all rabid animals could be destroyed, it would be possible to "stamp" out the disease; but this is impossible, for beside domestic dogs we have to account for wolves, foxes, and cats.

A more hopeful plan is to prevent dogs from biting by enforcing the use of a properly constructed muzzle. By this means rabies has been banished from Berlin; and its success when used in London has been remarkable. In 1885 twenty-seven persons died of rabies in London, beside twelve in the suburbs. In December of that year dogs were muzzled. In 1886 only nine persons died of the disease, and those persons were bitten before the muzzling order was issued. In December, 1886, the order lapsed. In 1887 and 1888 there were no deaths from hydrophobia in or around London. In 1889 there were 176 cases, in 1890 (with the order renewed) 44, in 1891 28, and in 1892 only 3. During 1893, '94, '95 the number rose again, until the order was again enforced in 1896. Happily the order has been reinforced in the county of London with good results (1896—1900); but until adjacent counties do the same, the disease is always liable to be introduced afresh.

Treatment.—No drugs have power over this terrible disease. Opium, chloral hydrate, or inhalation of chloroform relieve the distressing spasms for a time if employed boldly, but do not even postpone the fatal issue. Curare is probably equally useless, and the same must be said of cannabis indica, atropine, cocaine, salol, and methane, as well as of quinine, mercury, and the *other drugs employed by Morgan* and the older physicians (see

Mr Dowdeswell's paper in the 'Proc. Roy. Soc.' for 1887). Darkened rooms, hot baths, electricity, and every other appliance that reason or credulity could suggest have been fully tried with equally negative results.

Promoting free bleeding from the wound, sucking and washing it, and applying nitrate of silver or other suitable caustic,* are rational methods of treatment, and, if adopted early, may possibly prevent the virus from reaching the general circulation, and thus save the patient's life.

The Buisson or sweating treatment, still used in Russia, has been tried and found utterly useless.

Inoculation.—Since the first edition of this chapter was published, every one has heard of the researches of the late M. Pasteur, and of his method of preventing and curing hydrophobia by inoculation of an attenuated virus. The following is a brief account of these remarkable investigations.

Starting from the belief that rabies in man, in dogs, and other animals is a contagious disease, Pasteur ascertained that the fresh cord and bulb of a rabid dog, when pounded up with sterilised broth, makes a highly infective *materies morbi;* and this, injected under the skin or into the veins, or, most certainly and rapidly of all, under the dura mater, will reproduce the disease in a dog, cat, rabbit, or any other susceptible animal. As above stated, the symptoms vary for different species, and even for different individuals; but after death the cord of the inoculated animal will again furnish the contagium, so that the identity of the disease is proved.

Next he set to work to attenuate the virus, as he had that of anthrax. By drying the cord in a warm, dry, sterilised atmosphere (20° C. is the temperature chosen, or about 68° F.), the virus is rendered less powerful, and becomes progressively weaker day by day. When sterilised broth, treated with these dried cords, is introduced into a dog, he undergoes the disease in a mild form, and can then without danger be inoculated with a stronger virus, until at last he is rendered "refractory" to the most recent and intense broth, or to the effects of direct inoculation from the bite of a rabid dog.

The efficacy of this prophylactic or "preventive" treatment (called "vaccination" by Pasteur) is not a matter of question. If protected and unprotected dogs or rabbits are bitten by the same rabid dog or cat, the former escape and the latter die.

It would therefore be theoretically possible by inoculating all living dogs to "stamp out" hydrophobia for ever; just as, if vaccination were made compulsory everywhere for a generation or two, smallpox would become extinct, and vaccination itself no longer needful. But this is as impracticable for hydrophobia as it is at present hopeless for variola. All that can be done by way of prophylaxis is to diminish the number of dogs by taxation of the owners, to destroy masterless dogs, and to enforce the use of muzzles.

Pasteur next tried whether it is not possible to anticipate the march of the disease by taking advantage of its long period of incubation, and using attenuated virus with a shorter period, so as to overtake the original virus and fortify the organism against it, before its natural effects appear. Trials of this "protective" method on dogs and rabbits already bitten proved encouraging; and at last, when urged to do something to save a child named Joseph Meister, who had been severely bitten by a mad dog, Pasteur, as he tells us, with great anxiety, inoculated him with attenuated virus on July 6th, 1885. The result proved satisfactory; the boy recovered, and many other persons bitten by rabid (or often, in all probability, by non-rabid) dogs applied to Pasteur, and received "protective" inoculation. The numbers increased enormously, patients arriving from all parts of France, from Italy, Russia, England, and even from America and India. A certain number of deaths with undoubted hydrophobic symptoms occurred, particularly of several Russians who had been bitten by rabid wolves. Pasteur's results were not completely confirmed by independent investigators, *e. g.* Professor von Frisch, of Vienna, and he was violently attacked, not only by fanatical opponents of experimental pathology, but by certain French physicians. The difficulties in the way of a sound conclusion are great. For, first, no one knows how many of the people who have been inoculated had really been bitten by a rabid dog at all. Secondly, when a bite is inflicted on a part covered by clothing, the venomous saliva is generally wiped off, and so the person bitten escapes. Thirdly, many who were bitten had been well treated by caustics, and thus the virus may have been, and no doubt frequently was, destroyed before they were inoculated. Lastly, we do not know the proportion of men or animals who, from some "insusceptibility" or idiosyncrasy (*i. e.* some individual unknown cause), escape the disease even when the virus is fairly injected, though, judging by analogy from syphilis and from vaccinia, it is probably small.*

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* Mr Youatt did not practise excision, but trusted entirely to cauterisation with nitrate of silver. He himself was bitten seven times, and operated on 400 persons besides, among whom he had only one death, which he ascribed to fright.

Another question has also to be considered, whether the intended protective inoculation may not, if unwittingly employed on persons who have not really been infected before, produce a fatal form of the very disease against which it is supposed to protect.

It cannot be said that these questions are fully answered, or that all the difficulties have been surmounted. But a report, presented to the President of the Local Government Board by a committee appointed for the purpose of investigating this subject,† went far to prove to unprejudiced critics that not only has Pasteur discovered an efficient "preventive," *i. e.* strictly prophylactic treatment of hydrophobia—a fact of great scientific importance,—but that he has also succeeded in applying the same method to the therapeutical treatment of the same dreadful malady when it has already been contracted.

Among 2682 persons inoculated at the Pasteur Institute, forty died of hydrophobia. Nine of these deaths occurred among forty-eight persons who had been bitten by wolves—a peculiarly dangerous kind of rabies, and met by a more rapid and probably more hazardous process of inoculation.‡

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* See M. Roux's "Croonian Lecture before the Royal Society," reported in the 'Brit. Med. Journ.' for 1889, vol. i, p. 1269; also Dr Hime's paper in the 'Path. Trans.,' 1885-6.

† The committee consisted of Sir James Paget, chairman, Sir Joseph Lister, Sir Henry Roscoe, Professor Burdon Sanderson, Dr Quain, Dr Brunton, and Dr Fleming, with Mr Victor Horsley as secretary.

‡ The ordinary method was to inject on the first day of treatment virus derived from a cord dried for fourteen days; on the second, that of a cord dried for thirteen days; on the third, that of a cord dried twelve days; on the fourth, that of an eleven days' cord; fifth, ten days; sixth, nine; seventh, eight; eighth, seven; ninth, six; and tenth, five days. These injections were afterwards made more frequent, and the increase of strength more rapid. But for very severe cases, like those of wolf-bites, Pasteur now uses a *méthode intensive,* injecting on the first day of treatment virus derived from cords dried for fourteen

Excluding these, of 2634 persons bitten (or supposed to be bitten) by mad dogs, only thirty-three died, a percentage under 1·5, and far less than that assigned by any computation to cases treated in other ways or not treated at all.

Of 233 persons bitten by undoubtedly rabid animals (as proved by inoculation from the spinal cord or by rabies appearing in other animals bitten by them) only four died, instead of perhaps fifty and almost certainly twenty.

Of 186 persons bitten on the face (the most dangerous part) by undoubtedly rabid dogs only one twentieth died, instead of the previously probable proportion of at least a fourth.

Ninety cases were personally investigated by Dr Burdon Sanderson, Dr Brunton, and Mr Horsley, who went to Paris for the purpose. Among them no death had occurred. In thirty-one there was no clear evidence that the dog which had bitten the patient was rabid; but in twenty-four the bite had been inflicted on an uncovered part of the skin by undoubtedly rabid dogs.

Lastly, it appears certain that the ordinary inoculation as now practised at the Institut Pasteur is perfectly safe, although there is room for fear that the *méthode intensive* was attended with a certain risk.

Whether the protection will last a lifetime cannot of course be at present known. Further experience has confirmed the conclusion above stated in the last edition of this book. No dispassionate critic can withhold his admiration from the insight and ingenuity, the zeal and perseverance, which Pasteur displayed, or can doubt that his own and foreign countries were only paying a due tribute to his merit in the remarkable honours accorded him at his death.

During the last six or seven years Babes, of Bucharest, and Tizzoni and other Italian pathologists have elaborated a method of treating hydrophobia by antitoxic serum. The animal chosen for the purpose is the dog or the sheep, and when it has been rendered immune by inoculation with virus from the cord of a rabid dog of gradually increasing strength, it is found that their serum has antitoxic properties like those described above in the treatment of diphtheria and tetanus. One advantage of this method (which will be found fully described by Dr Woodhead in Allbutt's 'System of Medicine') is that the serum, when carefully dried, retains its power, and can be sent to the patient instead of the patient travelling to an "antirabic" institute.

and thirteen days; on the second, for twelve and eleven; on the third, for eleven and ten; on the fourth, ten days twice; on the fifth, nine days twice; on the sixth, nine days again; on the seventh, eighth, and ninth, eight days; on the tenth, eleventh, and twelfth, seven days; on the thirteenth and fourteenth, six days; and on the fifteenth and last day of treatment virus of maximum intensity derived from a cord of only five days' drying.

ANTHRAX

> **Nec tondere quidem morbo illuvieque peresa**
> **Vellera, nec telas possunt attingere putres.**
> **Verum etiam invisos si quis tentarat amictus,**
> **Ardentes papulæ atque immundus olentia sudor**
> **Membra sequebatur; nec longo deinde moranti**
> **Tempore contactos artus sacer ignis edebat.**
> VIRGIL.

Nomenclature—History and distribution—Varieties in animals—The Bacillus—Modes of infection—Varieties in man: Charbon, Intestinal and Thoracic Anthrax, their anatomy, prognosis, and treatment—Pathology—Prophylaxis.

Synonyms.—Splenio Fever—Splenic Apoplexy—Malignant Pustule—Woolsorters' Disease—Siberian Cattle Plague ("Jaswa"). In India it is known as "the Loodiana plague," and at the Cape as "Horse-sickness."—*Old English.* Blackbain. *Fr.* Charbon, Mal de Chabert, Mal de Rate.—*Germ.* Milzbrand.

Definition.—A specific infective disease, characterised by the presence of a pathogenic bacillus, conveyed from animals to man, running an acute course, attended with hæmorrhage, and frequently ending in death.

History.—This cattle plague has been known under various names from the earliest records. It was probably the grievous plague breaking out with boils and blains in man and beast which afflicted Egypt, and it is described by Virgil in the third Georgic. It was first clearly separated from other diseases by Chabert in 1780. The parts of Europe in which it is most common are Poland, Hungary, the countries of the Lower Danube, Italy, Prussian Saxony, and certain departments of France; and it is endemic in Catalonia, in the Romanga, and in Courland. Anthrax is prevalent in Siberia and in Lapland, in Western Asia, in Mexico and South America, in India, and in South Africa. It is not endemic in this country, nor in Australia, or the United States and Canada, but from time to time rapidly spreads as a highly contagious epidemic.

Anthrax occurs in domesticated animals, sheep, horses, oxen, and llamas; and also in deer, reindeer, buffaloes, and elephants. Camels, goats, and rodents are very susceptible to it. It also affects swine, and is easily transferred by inoculation to rabbits and guinea-pigs. Carnivorous animals are much less susceptible; but cats are more so than dogs. It is remarkable that birds by being cooled down, and frogs by being warmed up, are made artificially susceptible for the time.

Varieties in cattle.—In different kinds of animals the symptoms of infection by anthrax differ considerably. According to Bollinger, three principal varieties may be recognised:

1. Sometimes the animal (generally an ox or a sheep) becomes convulsed and insensible, with rapid breathing, and dies after a few hours.

2. Other cases are characterised by pyrexia, which is often remarkably remittent in type. Clonic spasms of the limbs are also observed. The fæces may contain a large quantity of blood.

3. In a third group of cases brawny inflammatory swellings form in and beneath the skin of the neck, chest, abdomen, or any other part, and often lead to extensive sloughing and ulceration.

Although veterinary pathologists formerly regarded the more rapidly fatal cases as examples of a specific fever without localisation, it is now known that there always is a local lesion in some part of the body. Anthrax is by no means always fatal to animals; the average mortality in horned cattle and in horses is said to be 70 per cent. The characteristic lesion found after death is enlargement of the spleen, which is from two to five times its normal size, softened and black.

The Bacillus.—The identity of the various forms of anthrax is proved by the constant presence of a single microphyte, which is now commonly known as the *Bacillus anthracis.*

This microbe was discovered independently by Rayer and Davaine, and by two German observers—Pollender, published in 1855, and Bäuell of Dorpat in 1857. But Davaine was the first who, in 1863, maintained that the contagion of the disease lay in these bodies, which on account of their being motionless he distinguished from the common mobile bacteria of putrefaction, and named *bacteridia.* They consist of straight or slightly bent rods, measuring 0·007—0·002 mm. in length. Dr Frisch and Dr Cossar Ewart have under certain circumstances observed movements, but as a rule they are motionless. They were classified by Cohn as a bacillus, and named *Bacillus anthracis.* This microbe is almost exactly like the *Bacillus subtilis* which secretes the butyric acid ferment, except that the latter is motile.

The rods sometimes cohere together at their extremities, and Koch and Ewart have described them, when cultivated in aqueous humour, as growing into long filaments, in the interior of which bright granules appear—the spores of the bacillus, far more capable of resisting heat and desiccation than the adult microphyte, and retaining for an indefinite length of time the power of development into the mature form. Nägeli maintained that the bacillus of anthrax, like putrefactive bacteria, multiplies only by fission. No doubt *Bacillus anthracis* does multiply by fission under the surface of an infusion, but when exposed to air it produces spores, and may, under certain conditions, assume a Torula form. These facts have been ascertained by Klein and other independent observers, so that Koch's original statement is abundantly confirmed. From their large size the bacilli are readily found in the blood, spleen, and many other organs (see Dr Crookshank's 'Bacteriology,' pl. xiv, fig 1; xvi and xvii, fig. 1).

Infection.—Anthrax, as it occurs in cattle, horses, and sheep, is readily transmissible by means of the blood from a diseased to a healthy animal. Inoculations are frequently and successfully practised by veterinary surgeons and by pathologists for diagnostic purposes.

There is reason to believe that the virus is often introduced by the stings of insects: Bollinger took gadflies from the body of an ox that had died of anthrax, and found by direct experiment that the contents of the stomach and intestines of these flies were capable of conveying the disease to rabbits. Another way in which it is sometimes given to sheep is by the bite of a dog that has just been feeding upon infected flesh.

There is no sufficient proof that effluvia from living animals affected with anthrax pass into the air and infect other animals through the lungs. Like enteric fever, yellow fever and cholera in man, this disorder is not directly contagious. The virus sometimes clings to stable utensils, harness, straw, or hay. Bollinger mentions a local outbreak which he carefully investigated at Weriken, where for four years the cattle in two sheds were decimated by anthrax, while it did not affect those in other buildings, closely adjacent. An analogy may be found in house-epidemics of enteric fever, and in plague and yellow fever.

Anthrax also resembles the miasmatic-contagious diseases of man in being often spread in ways still more indirect. It prevails among animals pastured on damp soils, peat-bogs, and near the borders of lakes or rivers that have overflowed. It is most frequent during the hot months of the year, particularly in August and September.

Buhl has included anthrax among the diseases to which he applies his "ground-water" theory (cf. pp. 145, 286), and he states that among the horses belonging to a large stud kept near Donauwörth the disease, which had been raging for a long time, ceased as soon as a system of drainage was carried out. But Bollinger argues that the reason why its prevalence is affected by dampness of soil is, in reality, that ground containing moisture affords conditions favourable for the multiplication of the *Bacillus anthracis*, which he supposes to be capable of maintaining an independent existence as a saprophyte.*

Anthrax is never purely "miasmatic," *i. e.* its specific bacilli exist in the soil or in water only when they have been introduced from a previous case. They may be derived either from the excreta of diseased animals, or from their dead bodies, buried as they often are in the fields, or lost and left to rot among brambles and nettles. Instances are recorded in which "enzootic" anthrax has ceased so soon as stringent rules for the disposal of all dead bodies were enforced. Possibly some of the bacilli from the infected carcases become free, undergo desiccation, are suspended in the air, and inhaled into the lungs; others may pass into drinking-water, and thus reach the stomach; while others, again, may find access to the lymph-spaces and blood by some breach of surface in the skin; but the usual mode of infection in cattle is by eating grass or fodder which has been infected by previous cases.

Conveyance to man.—Shepherds, farm labourers, and other persons who come into contact with living animals affected with anthrax never take the disease by mere contact. Veterinary surgeons may be infected in performing venesection, and slaughterers in killing or in skinning; the probability is that a slight abrasion or scratch upon the skin is directly inoculated with the virus.

Another way in which anthrax may arise in man is as the result of

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* Buchner asserted that, by a series of experiments, he converted a bacillus which is found in infusion of hay into an organism capable of producing in animals an infective disease identical with anthrax, and he asserts that to turn the anthrax-bacillus back into a hay-bacillus is comparatively easy. There is, however, good reason to disbelieve this alleged transformation. See Klein's Report to the Local Government Board, "On the Relation of Pathogenic to Septic Bacteria, as illustrated by Anthrax Cultivations" (1882).

eating the flesh of an infected animal. Leube, of Jena, has recorded an instance in which the imperfectly cooked liver of a diseased goat conveyed it. Thus the human gastric juice seems not necessarily to destroy the virus, as is the case with the gastric juice of the dog and other carnivorous animals. Thorough cooking removes all danger; but cooking is not always thorough, and there still remains the risk of injury to the butcher, and to those who prepare such meat for the table; and this is sufficient reason why the use of it, as food, should be prohibited. According to Heusinger the disease may be conveyed to the human stomach by milk or by butter.

But by far the most important cause of anthrax in man is infection *from the dried skins or hair* of diseased animals. In this way it is brought straight from Asia or South America to English workmen.

At Guy's Hospital cases are of not infrequent occurrence among the men engaged in the Bermondsey leather trade, and especially among those employed at wharves on the river-side where foreign hides are unshipped. Broca years ago remarked how often anthrax was set up by carrying skins upon the shoulders. It is remarkable that the danger appears to be greater when the hides are first brought ashore than afterwards. Rayer in 1835, and Trousseau in 1847, recorded cases of *charbon* contracted in this way, and in England, where anthrax was less common, Dr Budd traced the same connection in 1863 at Bradford, where a peculiar *Woolsorters' Disease* had for many years prevailed.

The source of infection at Bradford is chiefly "Van mohair," a material which contains much putrid matter and many "fallen fleeces," torn off the bodies of dead sheep. Dr Bell, who gave an excellent account of the affection in the 'Lancet' for 1880, remarked that the men who are attacked are chiefly the "bagmen"—that is, those who open the bags in which the mohair is packed, shake it out, and sort it into different qualities. Probably the spores are in a dried state, diffuse in the air, and are inhaled into the lungs. Perhaps the bacillus may multiply during the transmission of the bags from Asia Minor; for, according to Dr Bell, the hairs are not known to communicate disease to persons who handle them at Smyrna. Indeed, no form of anthrax so severe as the Woolsorters' Disease had hitherto been described as occurring in man. The work of sorting is said to be free from danger if the contents of the bags are first washed or even moistened with water.*

Other occupations which sometimes lead to infection with anthrax are the picking of Russian horsehair (as reported by Dr Russell, of Glasgow, in 1879), paper-making, and the manufacture of coarse woollen hats.

Varieties of human anthrax.—There are several forms of this disease in man, and they are not identical with those met with in animals.

1. *Malignant pustule* or *charbon* is the name by which the most common form has long been known. This consists in the formation of a vesicle, seated upon a more or less brawny base. Being usually produced by the inoculation of a scratch or slight sore, it occurs chiefly on the face,

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* See Reports to the Local Government Board by Mr John Spear and Dr Greenfield: "On Woolsorters' Disease," 1880, and "On Anthrax among persons engaged in the London Hide and Skin Trades," 1883. Also papers by Sanderson ('Journ. R. Agric. Soc.,' vol. xvi, p. 267) and Greenfield (ibid., xvii, p. 30). That the infection of anthrax is conveyed by the hides is implied by Virgil in the concluding lines of the third Georgic, which stand at the head of this chapter.

pecially the cheek, and also on the neck, the forearm, or some other posed part.

There is first a period of incubation, lasting generally several days, ten ten, but sometimes only a few hours. A slight pricking or burning nsation is then felt, which often leads the patient to think that he has st been stung by an insect. A papule quickly appears, and soon passes to a transparent vesicle, which may reach a considerable size. This ptures and dries up into a dark-coloured scab. Round it there sometimes ise smaller vesicles arranged in a ring. Meanwhile the base of the sicle becomes indurated, and a red or purple areola is developed round it. brawny œdema quickly spreads over the adjacent parts, affecting perhaps e whole of one arm, or of one side of the neck. Sometimes inflammaon of lymphatics follows vessels, and the lymph-glands swell.

During this time the health remains unaffected. The patient may ontinue at his work, and at most complains of slight malaise. But after bout forty-eight hours severe pyrexia sets in, often attended by delirium, ostration, diarrhœa, sweating, and acute pains in the limbs; and death rom collapse may follow between the fifth and the eighth days of the ttack. In one case which occurred at Guy's Hospital, the original vesicle had been situated an inch and a half behind the left angle of the lower jaw, and respiration became so difficult towards the last that recourse was had to tracheotomy. It was found after death that brawny infiltration had xtended deeply to the fauces, so that the entrance of the larynx was reatly swollen. In other instances the immediate cause of death is septi-æmia or concomitant anthrax of the intestine or lungs.

Diagnosis.—The only disease liable to be mistaken for anthrax affecting the skin is that which has been particularly noticed by Sir James Paget under the name of *facial carbuncle*. It presents no definite vesicle or central scab; and in most instances we have found the veins of the face obviously plugged with puriform thrombi, which has not been the case in anthrax. They were found to be so in a girl of 18 who died of this disease under the writer's care in 1870, and beside thrombi in the ophthalmic vein there was suppurative meningitis.

Bollinger, however, and other writers recognised as *anthrax-œdema* Bourgeois' *œdème malin*, a modification of the true charbon, in which there is neither vesicle nor eschar, but only a pale yellowish swelling of the subcutaneous tissues. The eyelids are its most frequent seat.

One way of arriving at a diagnosis of a suspected case of anthrax is to inoculate a rabbit, a guinea-pig, or a mouse with fluid from the part rimarily affected, or with the blood. Such small animals commonly die n two or three days, or still more rapidly, with dyspnœa, dilated pupils, nd perhaps convulsions. Their blood may then be seen to swarm with acilli.

Anatomy.—On examination after death the appearances are those of an fective fever,—congested lungs, flaccid heart, and more or less ecchy-osis or hæmorrhage. As a rule, the spleen is much enlarged and softened. t in a case under the writer's care in August, 1899, which proved fatal ter operation on the primary pustule, the spleen was not swollen, though the anthrax bacilli were present.

Treatment.—The recognition of the ordinary external form of anthrax of extreme importance, because surgical treatment at an early stage is able of arresting its progress. Cutaneous anthrax is not, indeed, always fatal, even when left to itself. Sometimes the central part of the swelling sloughs out, the surrounding induration subsides, and an ulcer is left which more or less quickly heals. Such a spontaneous cure is, however, very rare. Of nine instances recorded in 1863 by Dr William Budd, eight ended fatally. On the Continent it has long been the practice to destroy the local lesion by the actual cautery as soon as it is recognised. Bollinger cites the experience of a surgeon who lost only thirteen cases out of one hundred and forty-two; another had still greater success, since among two hundred and nine patients all but eleven recovered. In 1878 Mr Davies-Colley recorded in the 'British Medical Journal' two cases, in each of which recovery took place after excision of the entire mass of indurated tissue, with the application of chloride of zinc paste to the wound. Contrasting with these is a case which was left alone, and which ended fatally. Several other successful cases at Guy's Hospital have been recorded by the same writer in the 'Med.-Chir. Trans.,' vol. lxv, 1882. Since that date (June, 1882) forty-eight more cases were admitted into Guy's Hospital up to 1890; and of the total sixty-five cases of external anthrax forty-three recovered after operation. Two typical cases in butchers during an epidemic of anthrax among cattle were operated on by Dr Pitts, of Chelmsford, and recovered perfectly ('Brit. Med. Journ.,' March, 1887, p. 616).

The surrounding œdema often subsides after the operation with striking rapidity. This treatment may be effectual even when the case is at an advanced stage, and when severe general symptoms are present. Quinine should then be given in large doses, and the patient's strength must be kept up with nourishing food and with stimulants.

2. *Intestinal anthrax*, another form of "splenic fever," affects the gastro-intestinal mucous membrane. This is sometimes associated with an external malignant pustule, and even then is extremely rare in man. Dr Goodhart made an autopsy on a case at Guy's Hospital in 1877. A correct diagnosis is not likely to be made during life. However, Bollinger gives the following account of the symptoms:—The patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in with diarrhœa, and the evacuations often con tain blood. Dyspnœa and lividity appear, with sometimes excitemen sometimes with stupor. Epileptiform convulsions may occur, the arm may be affected with tetanic spasms, or there may be opisthotonos. Th pyrexia is but slight, and death is preceded by extreme collapse. Th duration of the disease is usually from two to seven days, but sometimes is scarcely twenty-four hours.

On *post-mortem* examination the abdominal cavity is found to contain moderate quantity of serous fluid which is often blood-stained. T lining membrane of the stomach and intestines shows patches of swellin generally of the size of lentils or coffee beans, but sometimes one or t inches in diameter. These on section are seen to consist of a pink fles infiltration of the mucous and submucous tissues, so that the valvulæ c niventes appear firm and prominent; the surface of the affected parts more or less excoriated and discoloured, or covered with an adherent la of extravasated blood. There are also spots of ecchymosis on both serous and the mucous aspect of the gastro-intestinal tract. The mes teric and the lumbar lymph-glands are often greatly enlarged, and o dark red colour. The mesentery may form a large brawny swelling, the connective tissue in front of the spine may be infiltrated in a similar manner. The spleen is softened to a pulp, but it is not generally much enlarged. A nodule in the stomach may slough out and repair begin, as in a case recorded by the late Dr Mahomed, in the 'Path. Trans.' for 1883.

Hitherto (1898) no instance of recovery from intestinal anthrax has been recorded. Leube has related a case in which the lower lip, the inner surface of the cheek, and the hard palate presented indurated patches of a bluish-red colour; epistaxis and hæmaturia appeared, but the malady ended favourably in about a fortnight. The patient took fifteen grains of carbolic acid and thirty grains of quinine each day, and the patches in the mouth were cauterised three times daily with carbolic acid. But the diagnosis of anthrax was not established beyond question; for only spores, no bacilli, were found in the blood. (Compare Mr John Poland's case, 'Path. Trans.,' 1886, p. 553.)

3. *Pulmonary anthrax.*—A third form of anthrax chiefly affects the thoracic viscera. It was first recognised among the woolsorters of Bradford by Dr J. H. Bell, of that town, in 1879. Mr Spear and Dr Greenfield investigated it for the Local Government Board in 1880, and described it as follows:—The earliest symptoms are great prostration and a sense of oppression of breathing. Shivering seldom occurs. The respiration is not much accelerated, but it is laboured and difficult, with a feeling of pressure or constriction. There may be more or less abundant bloody expectoration, or none at all. Auscultation seldom reveals anything more than slight rhonchus. The face is sometimes congested, sometimes pale, with a slight cyanotic tint. The extremities are cold and bluish; even in the axilla the temperature may be subnormal, but in the rectum the thermometer may rise to 102° or 103°. The pulse is rapid and weak, and sometimes irregular. There may be nausea and vomiting, but not generally diarrhœa. In many cases the appetite remains good, and digestion seems to be unimpaired. There is sometimes a sort of hysterical condition, or a state of mental anxiety and depression; but other patients have been so unconscious of danger as to refuse to send for medical help until the last few hours of life. Death may be preceded by delirium, convulsions, or coma, or the mind may be clear to the last, and the end come unexpectedly.

One patient lived only seventeen hours after he was first taken ill, and many others have died in from three to five days. Dr Bell says that those who survive for a week generally recover.

The *post-mortem* appearances vary considerably. Decomposition appears to be somewhat rapid. The lung tissue may either be congested or quite natural. There may be blood-stained secretion in the bronchial tubes, and their mucous membrane may be ecchymosed. Sometimes the pleuræ contain a pint or two of fluid, and the lungs are partially collapsed in consequence. There is often blood-stained serous or gelatinous infiltration of the mediastinal tissues, and the bronchial glands are swollen, softened, and ecchymosed. The pericardium may be marked with petechiæ, and may contain a blood-stained fluid; or a large quantity of blood may be found extravasated between it and the sternum. Sometimes the pharynx and adjacent tissues are infiltrated with blood or serum. There may also be ecchymoses in the pia mater, in the kidneys, and elsewhere. It is remarkable that the spleen is hardly, if at all, softened. In prolonged cases hæmorrhagic infarctions may be found in the lungs, attend with slight pleurisy; or there may be patches of broncho-pneumonia.

There is sometimes difficulty in distinguishing, by clinical eviden alone, this form of anthrax from a severe attack of pneumonia. Man fatal cases of the Woolsorters' Disease at Bradford have been registere under the head of pneumonia, bronchitis, or congestion of the lungs. Th characteristic bacilli have been found in blood drawn from the patient's ear; or a drop or two of blood injected under a mouse's skin will prove rapidly fatal.

The prognosis is very grave, but not so hopeless as in the intestinal form of anthrax.

General pathology of anthrax.—In the ordinary form of the disease affecting the skin, the bacilli may be found about the second or the third day, in clusters embedded in the rete mucosum at the centre of the vesicle. E. Wagner found that they are at this time so closely packed in the papillæ of the cutis as to conceal all the tissue elements. Thence they spread both laterally and towards the deeper structures, enter the vessels, and are carried all over the body with the blood. In the gastro-intestinal variety the bacilli infiltrate all the swollen œdematous tissues, so that Buhl described this affection under the name of *mycosis intestinalis* before it was recognised as a variety of anthrax.

At an early stage of the disease the blood may contain no bacilli, so that a negative result is inconclusive, although the presence of the characteristic rods decides the diagnosis. In man, even when a fatal termination is approaching, the bacilli seem to be seldom found in such immense numbers, and so generally distributed in the blood, as they are in animals. Davaine calculated that from eight to ten millions are present in a single drop of a diseased beast's blood. In Buhl's first case of intestinal anthrax they are said to have been present in the blood of the portal vein, but not in that of other vessels. They may often be detected in the substance o the spleen and of the solid tissues generally, and also in the fluid poure out into the serous cavities.

Mr Barker, in a case published in the 'Med.-Chir. Trans.,' vol. lxix, 127, found the bacilli most abundant in the rete and the papillary layer the cutis, and they appear to be long confined to that part. An e cellent account of the histology of the disease in this country given by Dr F. C. Turner, in the sixty-fifth volume of the same 'Tra actions.'

In anthrax we have a typical example of a specific contagious mala which is due to the presence of microphytes. All the conditions sta on p. 18 are fulfilled. For the disease is recognisable from its clini features, and breeds true; the organism is well marked in its mic scopical characters, size, and shape, in its reproduction and its cultivatio it exists in the blood and tissues of every case of the disease; and it exi nowhere else, for its non-mobility distinguishes it from *Bacillus subti* Lastly, when a "pure cultivation" has been obtained, it can be inoculate and will reproduce the disease, with fresh broods of organisms in t blood of the new host.

Prophylaxis by inoculation of the attenuated virus, though inapplicabl as a practical measure for human beings, is of too great theoretic impor ance to be wholly passed over

Pasteur ascertained that by repeated cultivations of *Bacillus anthracis* in mutton broth, at a temperature above 104°—110° Fahr., he obtained a modified virus which when inoculated into an animal produced a mild attack of anthrax which protected from future attacks. These results were tried on a large scale at Milan and afterwards at Chartres, and proved brilliantly successful (see a paper by Mr George Fleming, in the 'Nineteenth Century' magazine for March, 1882). Unfortunately the result is not always so complete, for even the attenuated virus is occasionally fatal. Moreover the Algerian breed of sheep appears to be insusceptible of the same protection, and in Hungary the results were much less successful than in France. See an excellent abstract by Dr Dawson Williams of Pasteur's and other methods of attenuation, and of Koch's criticism thereon ('Microparasites in Disease,' N. Syd. Soc., p. 560). Dr Roy found in Buenos Ayres that the Viscacha (*Lagostomus trichodactylus*), a rodent allied to the chinchilla, is capable of receiving the disease, and that anthrax thence transferred by inoculation to cattle is both mild and protective, at least for a considerable time; so that the analogy to smallpox and cow-pox is very close.

The late Dr Wooldridge succeeded in protecting rabbits from anthrax by injection of a thymus extract, and he began chemical researches on the cultivation of the anthrax bacillus ('Proc. Royal Soc.,' 1887), which have been followed by those of Hankin ('Brit. Med. Journ.,' 1889), and Sidney Martin ('Journ. Path. and Bact.,' 1893), in this country, and by many other investigators abroad.

The practically preventive measures against anthrax are inspection of cattle which are imported, destruction of the dead bodies and excreta of any that may still die of the disease, and inspection of the hair and hides which may carry the infection to factories.

GLANDERS

> *Οἱ δὲ ὄνοι μάλιστα νοσοῦσι νόσον μίαν ἣν καλοῦσι μηλίδα· γίνεται δὲ περὶ τὴν κεφαλὴν πρῶτον, καὶ ῥεῖ φλέγμα κατὰ τοὺς μυκτῆρας παχὺ καὶ πυῤῥόν.*—ARISTOTLE, 'Hist. Animal.,' lib. viii, cap. 25.[*]

History and definition—Natural history of its contagion—The specific bacillus—Acute and chronic forms of the disease—Anatomy—Prognosis—Diagnosis and Treatment.

FOOT-AND-MOUTH DISEASE.—*In cattle—In man—Its course, diagnosis, and treatment.*

Synonyms.—Equinia, Farcy, Μᾶλις, Malleus *v.* Maliasmus, Morbus glandulosus.—*Fr.* La Morve, Le Farcin.—*Germ.* Rotzkrankheit, Wurmkrankheit, Drüse.

Definition.—An infective epizootic disease, caused by a specific microbe, and chiefly affecting the respiratory mucous membrane and the lymph-glands.

This horrible disease was known to the ancients under the names *μᾶλις* and *malleus*. In England, writers on veterinary medicine distinguish two varieties, "glanders" and "farcy," the former a disease of the nasal mucous membrane, the latter marked by nodules and abscesses beneath the skin, and by swelling of the lymph-glands, with the formation of thrombi in the lymphatics at the valves ("farcy buds"). The first case in the human subject which was correctly interpreted in this country was recorded in 1821 by Dr Muscroft, of Pontefract. In 1830, Dr Elliotson drew the attention of the Royal Medical and Chirurgical Society to the disease, and afterwards proposed to call it *Equinia*.

Conveyance of contagion.—As a rule, the virus of glanders is directly introduced into a wound or sore, or fissure in the skin, especially of the face or of one of the hands. A horse-slaughterer who died in Guy's Hospital in 1866 appeared to have been poisoned through a slight cut on the lip which had been made by a barber in shaving him. Injuries to the hand in skinning dead horses, or in dissecting them, have sometimes been the starting-point of the disease. It has also been transmitted by a bite from a glandered animal, probably through the saliva. Or a diseased horse in sneezing may propel a drop of muco-purulent secretion from its nose directly into the eye, or the nose, or the open mouth of anyone standing near it. The contagion remains active in a dried state for a fortnight.

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ses are particularly liable to one disease which they call Malis (Melis, malleus). It d first, and causes a thick purulent discharge from the nostrils.

and if moist for a much longer time; for horses in a particular stable have been attacked many weeks after the occurrence of the disease.

According to Bollinger, who describes glanders in 'Ziemssen's Handbuch,' it may be communicated by eating the flesh of a glandered animal, at least in a raw state. The disease in man has always been exceedingly rare, even in countries where (as in France during the first half of this century) it was committing terrible ravages among horses. When it affects human beings it may spread from one person to another. Dr Elliotson mentions in the 'Lancet' for 1838 that a laundress who washed the clothes of one of his patients contracted it. In at least one case infection has taken place through a cut received in examining the body of a person who had died of the disease. Dr Sherrington found that the bacilli are passed out in the urine without any discoverable lesion of the kidneys.

Among the lower animals, goats and rabbits are said by Bollinger to be the most suitable subjects for experiment. Oxen and sheep are not susceptible, nor are cats and dogs; but lions and tigers in captivity have contracted glanders from diseased horseflesh. Next to horses, glanders is most apt to affect asses and mules.

Grooms are sometimes attacked with glanders when they are not aware that any of their horses have been diseased. This is due to the disease being frequently latent in the horse, *i. e.* without either the ozæna of traditional glanders or the swollen lymphatics of traditional farcy. The horse falls off in flesh, passes too much urine, and begins to swell in one of its limbs; and the lungs are already attacked before the disease is recognised. (See the account of glanders in horses by Professor MacFadyean and Dr Woodhead in 'Journ. Comp. Path. and Ther.,' March, 1895; 'Procs. Nat. Vet. Assoc.,' 1886; and Allbutt's 'System of Medicine,' vol. ii, p. 513).

Bacillus.—Schütz and Löffler, in 1882, ascertained the constant presence of minute rod-shaped organisms in the "farcy buds," lungs, and spleen of animals dying of glanders. Löffler succeeded in growing this bacillus, and inoculation with the pure cultivation has reproduced the disease in horses or asses, and also in guinea-pigs, rabbits, and mice. His results agree with those of Schütz, Bouchard, and other observers, and are now generally accepted.[*]

The *Bacillus mallei* is $\frac{1}{5}$—$\frac{3}{5}\,\mu$ long (*i. e.* the same length as that of tubercle, but thicker), non-motile, and does not form spores. It is readily cultivated, and from the pure cultivation is prepared a solution of its chemical products which is called *mallein.* This is of great practical importance, for when injected into a horse with latent glanders it produces a local and general febrile reaction, while it has but slight or no effect on one free from the disease.

Glanders in man.—The disease occurs in two forms in the human subject, of which one is termed acute and the other chronic.

1. In *acute glanders*, after a very short period of incubation, the patient is first attacked with malaise, headache, and pains in the limbs. At this stage the disease is generally mistaken for acute rheumatism or for enteric fever. But if a wound or scratch has been infected with the virus, the part commonly becomes red, swollen, and very painful; an

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[*] 'Deutsche med. Wochensch.,' 1882 and 1883; 'Revue Médicale Française,' Dec., 1882; 'Microparasites in Disease,' New Syd. Soc., p. 387, and pl. vi. Klein ('Micro-organisms and Disease,' 3rd ed., p. 128), and Payne ('Pathology,' p. 667, with fig. after Flügge).

erysipelatous inflammation may diffuse itself over a wide area, affecting the whole side of the face, or the hands, and part of the forearm; or raised red patches like Erythema nodosum, or subcutaneous abscesses appear. In such cases the diagnosis naturally inclines towards septicæmia or pyæmia from a poisoned wound. In other instances, as in that of a man who died in Guy's Hospital in 1863, the first complaint is of pain in the side with dyspnœa, so that acute pleuro-pneumonia is suspected.

In many cases an *eruption* appears on the limbs and the trunk as papules, which rapidly pass into flat vesicles and then into bullæ or large pustules. They become depressed in the centre, and rupture, allowing a thin purulent fluid to escape, which is often blood-stained. When an incision is made through a pustule after the patient's death, its floor is found to consist of an ashen-grey necrotic layer. The eruption sometimes appears within twenty-four or forty-eight hours after the patient is first taken ill, sometimes not for a week, or even longer. When present it is characteristic of glanders, although it might be mistaken at an early stage for small-pox or chicken-pox, and perhaps later on for pemphigus or syphilis.

There is often mixed with it a lesion which may be taken as the representative of "farcy" in horses. This consists in the formation of hard painful lumps or swellings in the subcutaneous tissues and muscles; they suppurate, and if incised may ulcerate deeply, so as to expose tendons or bones beneath. It is curious that in most cases the lymph-glands do not become enlarged, although a marked instance to the contrary is recorded by Travers in his work on 'Constitutional Irritation.' Not only was there suppuration of the glands of the arm originally affected, but those at each angle of the lower jaw and in the groin were also swollen. The joints in glanders not infrequently become inflamed, and suppurate.

The other characteristic but not constant symptom is an affection of the mucous membrane of the nose and of adjacent parts (*ozæna*), representing what in horses is termed "glanders" in the narrower meaning of the term. First a thin whitish mucus runs from one or both nostrils; afterwards it becomes purulent, blood-stained, and fœtid. The nose itself now becomes swollen, red, and very painful, and inflammation may spread from it towards the forehead or over the cheeks. (See models in the Guy's Museum, Nos. 52, 53, 55, 57.) If the parts are examined after death, the lining of the nasal passages is found to be ulcerated, and the septum perhaps necrosed. In the human subject a discharge from the nose is by no means always present in glanders; Hauff observed it in only thirty out of seventy cases which he collected. It is often not an early symptom, but appears in the second or the third week. Thus, during the first days of a doubtful case, the fact that the nose is healthy must never exclude the diagnosis of glanders.

Other mucous membranes are often affected. The conjunctivæ may be severely inflamed, and the eyelids greatly swollen. Virchow relates a case which came under von Graefe as one of acute exophthalmos, and its real nature was not suspected until after the autopsy. Sores may form in the mouth, and the gums may become spongy. The pharynx and the palate may ulcerate or become covered with false membrane. Sometimes ulcers form in the larynx, and œdematous laryngitis may cause a need of tracheotomy. Symptoms of bronchial or of intestinal catarrh may be present. Pustules and sores which might easily be supposed to be venereal, are said by Virchow to occur on the glans penis.

The general condition of the patient becomes worse from day to day. The pulse is usually accelerated, though sometimes it remains slow; the temperature rises irregularly until it may reach 104°; the tongue becomes dry and brown; albumen appears in the urine, and sometimes leucin and tyrosin. Delirium and sleeplessness pass into stupor and coma, and finally collapse, with involuntary escape of the urine and fæces. Death usually occurs towards the end of the second or in the third week. Sometimes the disease has ended fatally within a week, or even in three or four days, sometimes not for four weeks.

2. *Chronic glanders* is much more insidious and less fatal. It is often characterised by the formation of intractable ulcers with thick livid edges. In the Museum of Guy's Hospital are two models of such ulcers (Nos. 58, 59), the part affected being in one (doubtful) case the back of the hand, while in the other there are separate sores upon the forehead, the lobule of the ear, and the side of the face. In other instances, abscesses form about the joints and give rise to fistulous sores; or inflammatory swellings appear on the limbs, beneath the skin, or in the muscles. Or, again, there may be an eruption of pustules like those which are seen in acute glanders, but more slowly developed. The nose often remains free, but sometimes there is an erysipelatous redness of the skin or a fœtid purulent discharge from the mucous surface, beginning perhaps after two or three months have passed. The nostrils are often blocked with dark-coloured crusts. Such an affection is very apt to be regarded as syphilitic, or to be classed under the vague title of ozæna. It is of great importance to remember that the diagnosis may be cleared up by inoculating a goat or a rabbit with some of the discharge. This practice was adopted in some of the earliest cases of glanders that are known to have been observed in this country, those recorded by Travers. Bollinger gives details of two such experiments which he performed with matter taken from horses; the inoculated animals, which lived two or three months, showed characteristic symptoms. In a case at Guy's Hospital in 1866 the late Mr Poland introduced pus from the patient's face beneath the skin of his shoulder, and set up pustules there, but probably this result was not of diagnostic significance. Virchow mentions that at Würzburg a man was for six months in the hospital on account of refractory sores upon the limbs; and at the autopsy lesions characteristic of glanders were discovered. It was not then known that the disease existed in the neighbourhood, but inquiries were set on foot, and led to the discovery of an epizootic prevailing over a wide area among the horses used for towing barges on the river Main.

In some cases of chronic glanders, the chief symptoms are cough with sanguineous expectoration, hoarseness, pyrexia, and emaciation, so that phthisis may be suspected.

Morbid anatomy of glanders.—Some of the *internal lesions* which are found in cases of glanders in man appear to be the result of secondary pyæmia. Virchow placed glanders, in association with syphilis and lupus, among "granulation tumours." In animals, both he and Bollinger described the affection of the nasal mucous membrane as beginning with minute miliary papules. In the lungs are found nodules of various sizes, some almost exactly like tubercles, others as large as peas or larger; they also differ from tubercles in being hæmorrhagic and less numerous. On careful examination, characteristic small yellow bodies may be seen in the mucous membrane of the nose, of the frontal sinuses, and of the larynx and trachea. Von Wyss has since observed them in the gastric mucous membrane. It is interesting to find in one of Elliotson's cases, recorded in the 'Med.-Chir. Trans.' for 1833, a description of similar appearances.

> "On cutting into the various tumefactions on the head, trunk, and extremities, they were found to be full of pus, underneath which, in many, a number of small white granules were seen, and others, in several instances, were closely attached to the periosteum and perichondrium. The frontal sinuses contained a jelly-like mucus, and a number of similar granules, and on the septum narium was one ulcer exactly like those I have seen in the nostrils of glandered horses, and upon it lay a cluster of granules. About an inch below the valve of the colon, for three inches in extent, upon the whole of the surface, were white granules exactly like those in other parts."

Beside intermuscular abscesses, and suppuration of joints, periosteum, and medullary cavities of the bones, there is often acute suppurative lobular pneumonia.

The means of *diagnosis* formerly adopted in cavalry stables was to inoculate an ass with the mucus from the nostrils of a horse suspected to be glandered. The method now extensively used throughout Europe is the injection of mallein and observation of its general and local effects.

Prognosis.—Acute glanders is uniformly fatal. The average duration of chronic glanders is three or four months; among thirty-four cases which Bollinger collected from various sources, including many of slight severity, there were seventeen recoveries. The convalescence is slow and sometimes incomplete; a veterinary surgeon at the end of eleven years was still cachectic, and was troubled with cough and other symptoms due to the presence of cicatrices in the larynx and in the nose. But some patients, after a severe illness lasting for months, recover quickly and regain the flesh they have lost.

Treatment.—In acute cases all that can be done in the way of treatment is to sustain the patient's strength with quinine, tincture of iron, nourishing food, and stimulants. The pustules should be opened and the nostrils cleansed by syringing them out with some antiseptic solution.

In cases of chronic glanders, Bollinger thought carbolic acid and iodide of potassium the internal medicines most likely to be useful; Gamgee recommended arsenic in combination with strychnia. Others advise frequent doses of benzoate of soda. Ulceration of the interior of the nose should be treated with injections of creasote water, or a solution of carbolic acid, and the application of nitrate of silver.

Babes and Bonome recommend two drops of mallein by subcutaneous injection in cases of chronic glanders, repeated again and again until constitutional disturbance no longer follows; and Dr J. Macfadyean has employed this treatment in horses with success.

FOOT-AND-MOUTH DISEASE.*—In striking contrast with the dangerous epizootic disease just described is one which both in cattle and when communicated to man generally produces but transitory effects. On the Continent this disease has been known for centuries, but it is said to have been first introduced into England in 1839. In 1867 this country was again free from it, but it soon returned, and in 1871 it is believed to have affected more than 700,000 animals. In 1878 the Contagious Diseases Act (Animals) came into force, and though the distemper has shown itself more than once since (last in 1893) it has never been allowed to spread.

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* *Synonyms.*—Aphthous Fever—Aphtha Epizootica—Glossanthrax—Eczema Epizooticum. Some writers have described this disease under the mediæval name of *murrain*, which is familiar from its being used in the English translation of the Bible.

It occurs chiefly in cattle and sheep; pigs are also liable to it, but probably only as the result of infection by feeding upon the milk of diseased cows. It is very rarely seen in horses or dogs.

It is an acute specific fever, highly contagious, and attended with the formation of vesicles and bullæ upon the mucous membrane of the mouth, including the lips and the tongue, and with a somewhat similar eruption on the feet, round the borders of the hoofs, and in their clefts. The vesicles in the mouth soon break, and form little ulcers with a grey floor; on the feet they become pustular, and dry up into crusts. They give the animal pain in walking or standing, so that it generally remains lying down. The vesicles also appear upon the udder and the teats. After twelve or fourteen days all the effects of the complaint pass off. It almost always ends in recovery, except in calves. In sheep and pigs the stomatitis and salivation are less severe, but the affection of the hoofs is worse.

The *contagium* appears to reside in the fluid contained in the vesicles. By cultivating in agar-agar, in broth, and in milk (which it turns acid, but does not curdle), Klein in 1885 isolated an apparently distinct micrococcus; but the proof that this or other microbes since discovered is the specific contagium is still wanting, and a prize of 3000 marks (£150) offered by the Prussian Minister of Agriculture for its discovery in 1893 remains unpaid.

The disease in man.—Although foot-and-mouth disease is capable of transmission to man, this is not frequent. Prof. McBride could find only twenty-two cases recorded during nearly thirty years (1839-67); and most of these were taken, not from medical journals, but from the 'Veterinary Review.'

Conveyance.—There are two ways in which the complaint may be communicated to man. One is by the direct *inoculation* of the specific virus into a crack or sore place, generally on the hand or on a finger. This is most apt to befall a person engaged in milking a cow with the eruption upon the teats, but sometimes saliva from the beast's mouth conveys the contagion to a veterinary surgeon; and a butcher is said to have taken it by holding his knife between his lips while dressing an infected carcass.

The other way is by *drinking the milk* of a diseased cow. This is far more important, not only because it concerns children, who suffer much more than adults, but also because it is very likely to be overlooked. As to the characters of the milk which is yielded by cows suffering from the foot-and-mouth disease, there have been some discrepancies of statement. Sometimes it is offensive to the smell and the taste, and it may even be mixed with blood or pus in considerable quantity; but this seems to be only when the teats are severely excoriated, or when the mammary glands themselves have become inflamed as the result of retention. In other cases it is less obviously altered; but is apt to coagulate when boiled, or even at a much lower temperature, forming innumerable little flocculent masses, which float in a bluish whey. Sometimes it seems to have a yellowish tint, and when examined microscopically displays granular cells like those of the colostrum. But sometimes it is said to have a perfectly natural appearance. It seems generally to be diminished in quantity by about one half. Two German veterinary surgeons, Hertwig and Jacob, succeeded in infecting themselves experimentally by drinking the milk of diseased cows. No doubt, however, it is often drunk with impunity, not only by adults, but even by children. In any case boiling may be assumed to render it innocuous.

Course.—The *incubation* of the "foot-and-mouth" disease in man is said to be from three to seven days. Slight pyrexia, with loss of appetite, is followed by a sensation of dryness and heat in the mouth, and vesicles quickly appear on the inside of the lips, on the tongue, and sometimes upon the fauces and hard palate. They reach the size of peas, their contents become opaque and yellowish, and in from one to three days they break, forming shallow, dark red ulcers. There is some pain, which is increased by mastication, swallowing, or talking. The lips become swollen, and mucus and saliva are poured out in excess. In a case related by Mr Briscoe, of Chippenham, in the 'Brit. Med. Journ.' for 1872, the tongue swelled until it protruded more than two inches from the mouth, and it was so firmly wedged between the teeth that for thirty-six days the patient took no food except milk; there was great dyspnœa, and suffocation appeared at one time imminent; sloughs peeled off the tongue, and the discharge became very fœtid. Such a condition, however, is altogether exceptional. There is often slight diarrhœa and abdominal pain.

In some patients an eruption appears upon the fingers, especially round the nails. It consists of small clear vesicles, which pass into pustules, and sometimes run together. The inflammation very seldom causes shedding of the nails. The feet are seldom affected, but sometimes vesicles have been observed between the toes. Mr Amyot ('Med. Times and Gaz.,' 1871) mentions the case of a woman in whom the feet "became hot and covered with painful tubercles." In milkmaids vesicles have been observed on the breasts; and in the case of a clergyman in Germany, who caught the disease from eating butter made from his own cows' milk, the vesicles appeared on the face as well as in the mouth. This was during the epizootic prevalence of the disease in 1892.

Diagnosis.—The foot-and-mouth disease seems most likely to be mistaken for herpes or aphthous stomatitis. It is not impossible that inflammation of the mouth, with sore throat in children, may be a more common result of infection by diseased milk than is suspected. A doubtful case might be cleared up by inoculating a sheep.

Prognosis.—The duration of the foot-and-mouth disease in man is usually from ten to fourteen days, sometimes not more than a week. With one doubtful exception death has occurred only in infants.

The *treatment* required is local application of a solution of borax to the mouth, or of the solid nitrate of silver to any painful ulcers; the eruption on the fingers should be treated with lead ointment, and protected from the patients' nails.

ACTINOMYCOSIS

> "Et quoniam variant morbi, variabimus artes;
> Mille mali species, mille salutis erunt."
>
> Ovid, Rem. Amor., v. 525.

History—Discovery of the infective organism in animals and in man—The botanical position of Actinomyces—Clinical characters as it affects the lungs, the liver, the intestines, and the skin—Treatment, external and internal.

This remarkable disease is transferred by contagion from animals to man; but its effects are local and suppurative rather than general and toxic. It depends on the presence of a specific vegetable parasite, but it is not yet certainly determined whether it is a fungus or a bacterium. Its effects in some respects approach nearest to those of the tubercle bacillus.

Actinomycosis was long known among cattle as a suppurative inflammation, usually affecting the tongue or jaw, occasionally the skin or the lungs (cf. Rivolta, of Pisa, in 'Virchow's Archiv,' 1875, vol. lxxxviii, p. 309). It was called "wooden-tongue" by the herdsmen, "scrofula" and osteosarcoma by veterinary surgeons. The vegetable parasite which gives rise to it was first described in cattle by Bollinger in 1877, and named Actinomyces (*i. e.* ray-fungus), from the striking rosette-like figure it often presents.

In man it has probably been often mistaken for tuberculous nodules, for caseous inflammation, or for sarcomata, in the liver or the lungs.

The first two undoubted cases were published by Dr James Israel, of Berlin, in 1878;* one occurred in a man of thirty-nine, with multiple abscesses and serous inflammations, and the other in a man of thirty-six, with a submaxillary abscess, who recovered. In both the characteristic nodules and the "club-shaped or pear-shaped bodies," which make up the rayed rosette, are unmistakable (pl. i, fig. 2; iii, fig. 5). In his article he published notes of a third case observed by Professor von Langenbeck at Kiel in 1845 of vertebral caries in a man; and the drawings then made, and reproduced by Israel (pl. iii, fig. 9, *a* and *c*), prove that it was Actinomycosis. In 1848 Lebert had made drawings of the same fungus from a patient of Louis in Paris, but without either of them recognising its true nature. Ponfick first recognised these *Mycoses* to be the Actinomyces discovered in cattle by Bollinger ('Die Actinomycose des Menschen,' 1882).

The first case in man recognised in this country was described by Dr Acland in the 'Pathological Transactions' for 1886 (pp. 545-6). A remarkable case was brought before the Hunterian Society by Dr F. C. Turner (March, 1887). Here it was combined with true tuberculous disease, probably of independent origin. Numerous specimens in the jaws and tongue of cattle are in the Museum of the Royal College of Surgeons (Nos. 2254, *b*, *c*, &c., and 2274, *b*, &c.).

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* 'Virchow's Archiv,' 1878, vol. lxxiv, p. 15, "Neue Beobachtungen auf dem Gebiete der Mykosen des Menschen," 'Klin. Beitr. zur Kenntniss der Actinomycose des Menschen,' Berlin, 1885; translated in abstract for the New Sydenham Soc. in their vol. on 'Microparasites in Disease,' by Dr Hime, of Bradford, 1886. In Dr Acland's article in Allbutt's 'System of Medicine' (vol. ii, p. 81) is given a full historical account with copious references.

The peculiarity of the disease is that each inflammatory nodule or abscess has in its centre a parasitic fungous growth, which varies from the 100th to the 10th of an inch in diameter. In one of the earlier cases observed in this country a section from the morbid tissues of the tongue of an ox was shown the writer by Professor Roy at the Brown Institute. The large size, glittering aspect, globular shape, and surrounding mass of radiating club-shaped structures distinguished the organism at once from any known morbid product, and suggested the deposition of lime salts or some other crystalline material. This, however, is not the case.[*] It stains well with eosin or with magenta and picric acid. Actinomyces, as the fungus was named by the botanist Hartz in allusion to its radiating gonidia, seems very unlike the Schizomycetes to which the micrococci, sarcinæ, bacteria, bacilli, and vibriones of anthrax, glanders, and other specific contagious febrile diseases belong. It is apparently one of the Hyphomycetes, the group to which moulds like *Penicillium glaucum* and *Mucor* belong; and the centre of each minute yellowish nodule consists of an immense number of interlacing threads like mycelium.

If this view of its botanical position is correct, actinomycosis does not belong to the same group of acute febrile infective diseases as anthrax and glanders. It belongs to a group widely represented in the pathology of the lower animals, of which the *pébrine* of silkworms is a well-known example. The *Saprolegnia*, which causes salmon disease, is another. Grobe, Loch, and other pathologists have produced artificial "mycosis" of the kidneys and other viscera in the rabbit by injection of the spores of *Aspergillus*. In Favus and Ringworm, as in Thrush, the parasite is a fungus; but, owing to its not penetrating the skin or mucous membrane, its effects are only superficial. It is possible that cases may hereafter be recognised in man of other internal diseases due to invasion of parasitic fungi, and therefore analogous to actinomycosis.[†]

The results of cultivation, which has been carried out by Crookshank (1889), by Boström (1890), and by Wolff and Israel (1891), are opposed to this view of the botanical position of actinomyces. Though these observers differ in the interpretation of some points, they agree that the clubs or pseudo-gonidia are not to be found in artificial cultivations, and that the threads are not mycelium.

Nevertheless some good botanists, both French and German, still regard Actinomyces as belonging to the Mucedinæ, *i. e.* as one of the hyphomycetes, and the name *Oospora bovis* had been proposed to mark this position. Whatever place be ultimately assigned this organism, it appears to be closely related to, if not identical with, *Chionyphe Carteri*, the cause of the disease known as *Mycetoma* or Madura foot. (See a paper by the late Dr Kanthack in the 'Journal of Pathology and Bacteriology,' 1892, p. 140).

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[*] See Israel's original paper above quoted, where it is stated that Dr Ferd. Cohn, who recognised the fungus, at first thought them to be fat crystals; also the discussion on Dr John Harley's case of so-called "Actinomycosis of the Liver," 'Med.-Chir. Trans.,' 1886, p. 135, and 'Proc.,' N. S., vol. ii, p. 20.

[†] In Dr Paltauf's fatal case of *Mycosis mucorina* in man, the fungus invaded the body through the intestines, and led to abscesses in the lungs, brain, and other organs (see 'Virchow's Archiv,' vol. cii, with references to fungi in the air-passages, etc., p. 543).

The striking rosette-form with glittering "clubs" is often absent in the human subject. What is then characteristic are the globules of waving tangled threads or filaments. These are well shown unstained in plate ii of Dr W. B. Ransom's paper in the 'Med.-Chir. Trans.' (vol. lxxv, p. 85), and stained in plate iii, illustrating the same case of Actinomycosis of the rectum and prostate. In the same volume, beside a report of a case affecting the cæcum and liver by Dr Ransom, sen., there is also one by Mr Anderson of the disease affecting the face and neck (p. 103).

The diagnosis depends upon the discovery of the characteristic yellowish-brown nodules in the discharge from a wound, or occasionally, in the urine.

The mode of invasion of Actinomycosis is unknown. It is remarkable that it has never been traced to direct contagion from cattle; and it is possible that both cattle and man derive it from some vegetable source, and that it is introduced through carious teeth, or directly from the mouth, intestines, or bronchi. Probably barley or some other grass is the conveyer of the microbe to the mouth. It has been reproduced in animals by inoculation of the pure culture.

When established in a living tissue, each mass reproduces itself; and as it grows, a zone forms around it of large nucleated cells. They are surrounded by leucocytes (inflammatory or exudation corpuscles), and these again, in the larger and older specimens, by a capsule of spindle-cells or fibrous tissue. The inflammatory nodules thus formed coalesce and gradually become large tumours. While still scattered and small, in the lungs they closely resemble tubercles.

Actinomyces does not infect the blood, lymph, and tissues like bacteria and their allies, but acts as a local irritant, spreading slowly, and limited in its action to the tissues and organs successively affected.

There are more than five hundred cases (some not certainly genuine*) now recorded of this disease in man. Usually, as in cattle, it affects the mouth and jaw, sometimes the bronchi and lungs, or the intestines and peritoneum, and sometimes the liver only. Two specimens of the last group were described by Mr Shattock ('Path. Trans.,' 1885), one of them an old specimen from the museum of St Thomas's Hospital.

The fungus differs considerably in some recorded cases in man from the characters described in cattle. Whether these are specific differences, or depend on a stage of development or some other modifying cause, remains to be seen. (Compare Dr Acland's figures, 'Path. Trans.,' 1886, pl. xxv, with those in Dr Harley's case, and both with Israel's, Ponfick's, and Crookshank's drawings.)

The disease also differs as it affects different organs. In the lung it simulates miliary or diffused tubercle, and can only be distinguished by microscopical examination. In the liver it forms large spongy "worm-eaten" yellowish masses, infiltrated with pus; and may be mistaken for a tropical abscess. In the intestine it forms tumours, which suppurate and ulcerate, and simulates typhlitis or carcinoma of the colon, or of the rectum. In the skin, as described by Dr Pringle ('Med.-Chir. Trans.,' 1895), it forms tumours like sarcoma, which break down and form shallow ulcers with clear tenacious secretion. The pus of actinomyces is often viscid, and contains minute brownish granules, which are very characteristic. The suppuration spreads like a cancer, irrespective of the tissues invaded.

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* One, for instance, tabulated by Israel, is Mr Treves's "Case of Supposed Actinomycosis" (as he rightly called it), which proved to be large-celled alveolar sarcoma ('Path. Trans.,' 1884, p. 356).

The most frequent seat of Actinomyces is the neck and head (tongue, jaw, and orbit), next the abdomen, next the lungs, and lastly, the skin.

When actinomycosis runs a chronic course, there is little suppuration and much fibrous degeneration, so that it may simulate cirrhosis of the lung.

In a remarkable case, seen by the writer with Dr W. B. Ransom of Nottingham, the patient, a strong and healthy man of about fifty, was attacked with what appeared to be typhlitis, followed by extensive suppuration in the subperitoneal tissues, and at last by abscess of the liver. The correct diagnosis was made by Dr Ransom discovering in the pus discharged after incision minute brown granules, just visible to the naked eye, which collected at the bottom of a glass, and showed the characteristic club-shaped structures above described ('Med.-Chir. Trans.,' 1892, vol. lxxv, p. 63).

When the tumours or abscesses are accessible to surgical treatment, actinomycosis may be cured both in animals and in man. Hence the best prognosis is when the disease affects the tongue, jaws, or neck, and the worst when it affects the lungs or the liver.

Iodide of potassium given in full doses internally, as first used by Thomassen, has proved an effectual remedy, both in cattle at Chicago, and in human beings (see papers by Dr W. B. Ransom in the 'Brit. Med. Journ.,' 1894, vol. i, p. 61, and June 27th, 1896).

DIABETES

Si tibi nulla sitim finiret copia lymphæ,
Narrares medicis.
Hor. Epist., II, ii, 146.

Its isolated position—Its history and definition: polyuria, hydruria, and glycosuria—Detection of glucose in the urine: the cupric, potash, fermentation, and other tests—Quantitative estimate by volumetric and other methods—The amount, specific gravity, and other characters of diabetic urine—Glycosuria as a symptom—Artificial glycosuria.

Symptoms and course of diabetes—Complications: phthisis—coma—acetonæmia—Modes of death and post-mortem appearances—Nervous system—Pancreas.

Physiology of glycogenesis—Glycosuria and glycosæmia—Theories of diabetes—escape of sugar—over-formation of sugar—increased glycogenetic function of the liver—diminished destruction of sugar—Nervous, toxic, and pancreatic glycosuria—incomplete conclusion.

General ætiology of diabetes—inheritance—distribution—age and sex.

Prognosis, course and duration.

Treatment: by diet—by drugs—treatment of complications.

Having now completed the first series of diseases, those which affect the whole body, which are attended by fever, which run a more or less defined and limited course and are infective, or in many cases actually contagious, the most convenient arrangement of the greater number of the remaining internal maladies to which mankind is liable is that of the various organs which are more or less exclusively the seat of each disorder.

This anatomical arrangement, like every other, is open to objection, for "if one member is grieved the whole body suffers;" and, to take an example, chronic Bright's disease is a more "general" disease than actinomycosis. So Gout and Rickets are certainly not merely diseases of the joints or bones; and yet it seems undesirable to separate the one from Rheumatism or the other from Mollities ossium. A pathological arrangement is only possible when we know the causes and nature of disturbed physiology far better than we do at present; while a clinical arrangement is always useful, even if it brings together contrasts no less than resemblances. Nothing is gained by classing Rheumatism with infective fevers before we know its true pathology, or by forming an ætiological group of toxic disorders, and including in it plumbism and delirium tremens and gout, or by dealing in a single chapter devoted to animal parasites with hydatid of the liver, scabies of the skin, and tertian fever.

Diabetes is a "general disease" which has no local seat, which is certainly not a disease of the kidneys, and which we cannot range with those of the liver or the nervous system any more than with febrile, zymotic, or bacterial diseases. If we were compelled to classify all maladies, as Cullen and Sauvages did, we might group diabetes under "Disorders of Nutrition," with Obesity and General Atrophy, or under "Physiological Disturbances," with Epilepsy and Jaundice. But at present Diabetes stands alone, its origin and nature undiscovered, and its relation to other diseases uncertain. We therefore place it by itself as a non-febrile General Disease, with no ascertained pathology or anatomy.

Synonyms.—Diabetes mellitus, as it was named by Willis—D. anglicus, because described by an English physician—Saccharine diabetes—Persistent glycosuria.—*Fr.* Diabète sucré.—*Germ.* Zuckerharnruhr.

Definition.—A condition of persistent glycosuria, due to excess of sugar in the blood, and accompanied by wasting, thirst, and other characteristic symptoms.

History.—It was known from early times that the quantity of urine daily passed may sometimes be greatly increased, without previous excess in drink or other obvious cause; and the Greeks named this disorder Diabetes.[*]

Polyuria is a frequent sign of the most chronic form of Bright's disease, as well as of hydronephrosis, cystic degeneration, and probably all forms of renal atrophy. But, after death from diabetes, the kidneys are found to be healthy. Although renal tissue is prone to compensatory hypertrophy, this is never a primary process; nor do we ever meet with "a morbid excess of function;" all disease is impaired function. Polyuria is, with few exceptions, hydruria, for the total amounts of urea and saline constituents excreted in the twenty-four hours are very rarely increased, and are sometimes diminished. It is not increased secretion of urine, but increased flow of water.

In the latter part of the seventeenth century (1674) the anatomist, Dr Thomas Willis, made the remarkable observation that in most cases of diabetes (*i. e.* polyuria) the urine contains sugar. The distinction he made between *diabetes mellitus* and *diabetes insipidus* is no longer necessary, for the former is not only a far more frequent and important malady, but its clinical and pathological characters are very different. It therefore may now be called diabetes without qualification.

It was early observed that patients affected with this disorder not only are continually thirsty and hungry, but grow thin, weak, and dried up, in spite of all they eat and drink. It was supposed that their flesh liquefied, and so ran through them; and we shall see that in the worst cases in all probability some of the sugar excreted is really derived from the tissues.

The abundance of the urine is partly the physical result of the presence of sugar in the blood, partly it has an independent source in the nervous system. Hydruria occurs in diabetes insipidus and other conditions, but glycosuria is not met with otherwise except as a temporary effect of certain drugs, to be discussed hereafter.

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[*] The term Diabetes (διαβήτης, a siphon) is used by Aretæus, Galen, Alexander of Tralles, and Paulus Ægineta, but does not occur in the writings of Hippocrates, Celsus, or Cælius Aurelianus. The *locus classicus* is in the treatise of Aretæus on chronical diseases (lib. ii, cap. ii), θώϋμα τὸ διαβήτεω πάθος, κ. τ. λ. The name is thus explained:—"Atque hinc equidem adfectum *diabetem* vocatum esse arbitror, perinde ac si διαβήτην, *i. e. siphonem hunc* dixeris; quia humor in corpore non remanet, sed homine, tanquam canali quodam, ad *effluendum* utitur."

The pathology of this disease is still obscure, for it depends on a disturbance of the chemical processes called metabolism, the seat and course of which are still in great part secret. We know, however, that the most essential symptom is the passage of sugar in the urine; we know that this glycosuria depends on the presence of sugar in the blood, and this again upon some disturbance in the changes which the food undergoes after digestion and absorption. It is not a disease of the kidneys, of the urine, or of the blood, but is a derangement of the chemical labour of nutrition. Its place in a nosology is at present arbitrary.

*Glycosuria.**—Chemically, diabetic sugar is identical with grape-sugar or *glycose*, also known as *dextrose*, from the fact of its solution turning a polarised ray of light which passes through it to the right hand. Fruit-sugar, *fructose* or *lævulose*, rotates to the left, while cane-sugar or *sucrose* and milk-sugar or *lactose* have no such rotary power. When taken as food, sucrose is split up by a special ferment in the stomach into dextrose and lævulose, and lactose into dextrose and galactose. *Maltose*, the sugar formed in the process of making ale, is readily converted into dextrose. Ordinary starch or *granulose* yields maltose by hydration under the influence of salivary or pancreatic digestion, while a vegetable starch called *inulin*, found in the dahlia and some other plants, yields lævulose. Lastly, *glycogen*, the animal starch, readily becomes dextrose by hydration.

We shall see that these chemical facts find a practical application in the treatment of diabetes.

Tests for glycosuria.—There are several chemical processes by which we can detect the presence of glycose in the urine, but only a few of them are used in practice.

(1) The chief of these is the *copper test*, which may be applied in several ways; but they are all based upon the fact that diabetic sugar or glycose possesses the property of rapidly reducing the oxide of copper to a suboxide at the temperature of 212° or lower. The hydrated oxide of copper is blue, and liquids containing it in solution have a deep blue colour; while the hydrated suboxide is orange-yellow, so that there is no difficulty in seeing whether reduction takes place or not.

Trommer's method consisted in adding a few drops of solution of cupric sulphate to the urine, and then an excess of liquor potassæ. The precipitate of the protoxide first thrown down is redissolved if sugar is present, and forms a deep blue clear solution, from which on heating is precipitated the orange-yellow and, at last, brick-red suboxide of copper. Liquor sodæ may be used instead of liquor potassæ.

Barreswil and Fehling introduced the method of keeping the copper in solution in excess of potash by means of tartrate of potash or potassio-tartrate of soda (soda tartarata), so as to have the test-liquid ready for use.†

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* Glycosuria, *i. e.* the presence in the urine of glycose or glucose (from γλύκυς, sweet).

† The following is Dr Pavy's modification of Fehling's formula:—640 grains of neutral tartrate of potass and 1280 grains of caustic potass are dissolved in ten ounces of distilled water, and 320 grains of sulphate of copper are dissolved in other ten ounces; the cupric solution is then poured into that of the potass salt, and forms a clear blue liquid. Dr Pavy has also devised an ammoniated cupric solution for quantitative analysis. The precipitated suboxide is kept in solution by ammonia, and the test becomes one of colour only.

On adding urine to a rather larger amount of the liquid in a test-tube, no change is observed until the test-tube is heated to the boiling-point. If sugar is present, the liquid will deposit a yellow, orange, or red precipitate of the hydrated suboxide of copper.

There are several points which require comment in the application of the copper test.

It is said that if the solution has been kept for some time exposed to light, it may undergo a slight reduction when boiled. In a hospital ward, in assurance practice, or wherever the excellent rule is followed of testing the urine of every new patient, there is no real risk of this fallacy, particularly if the bottle in which the test solution is kept is of dark glass; but if long kept in the light, gradual reduction takes place even at an ordinary temperature.

The late Sir William Roberts advised heating Fehling's solution first, and then adding the urine gradually; for in the production of the deposit of the suboxide it is necessary that the sugar should not be in great excess, since unaltered glycose has the property of dissolving the suboxide of copper. Hence, if urine containing a large proportion of sugar be added to the copper solution in considerable quantity, no precipitate will result, but merely an opaque yellow solution. But if only a few drops of the saccharine urine are added, a deposit is produced of a characteristic orange-red colour. On the other hand, when the urine contains only a small proportion of sugar, it must be added in larger quantity. Then, as soon as the boiling-point is reached, the liquid changes to an opaque yellowish green, and a bright yellow deposit is slowly formed.

Roberts determined the exact limits of the copper test, and found that one tenth of a grain per fluid ounce can be detected with certainty.

It is well known that many substances besides sugar are capable of reducing the oxide of copper; but few of them are present in the urine.

One is *chloroform*, and hence reduction of the copper solution in the case of a patient who has recently undergone an operation under chloroform is no proof of glycosuria; the drug is, however, rapidly eliminated by the lungs as well as the kidneys. The same effect may be produced in the urine of patients who are taking chloral hydrate (Dr Sherwin, 'Boston Medical Journal,' November, 1886, quoted by Johnson). Leucin acts in the same way, but it nevers occurs in the urine except in cases of acute yellow atrophy of the liver.

The only drug frequently taken which causes a reduction of Fehling's solution is *salicylic acid* and its compounds. This effect is not constant, but frequent enough to be worth remembering. The writer discovered it several years ago, and finds it the rule in patients who are taking salicylates for rheumatism. The reducing agent is salicyluric acid, in which form salicyl compounds are partly excreted by the kidneys.

According to Dr Pavy, the orange precipitate sometimes fails to be produced when there is albumen in the urine; but as a rule albuminous urine containing sugar answers perfectly to the copper test.

Another fallacy in the detection of diabetes may be due to excess of *lithates* or of *kreatinin* in the urine. Either of these normal constituents of urine may, if in sufficient amount, produce a slight deposit of the suboxide; more often there is only discoloration of Fehling's liquid.

(2) Although the copper test for sugar fulfils all practical requirements *in* accuracy and delicacy, there are other methods of detecting diabetes

which also have their value. One is Moore's, or the *potash test*. It consists in boiling one or two drachms of the urine in a test-tube with half its bulk of liquor potassæ. As the ebullition goes on, the liquid becomes darker, passing through a series of colours like those of different kinds of sherry wine, until it becomes brown if the sugar is abundant: if less so the shade is lighter. The colour deepens on cooling, and, if nitric acid be added, a distinctive odour of caramel is perceived. This test is not a very delicate one; it does not succeed with urine containing less than a grain and a half or two grains of sugar in the ounce. Moreover most high-coloured urines become somewhat darker when boiled with liquor potassæ; and if the potash solution contains lead, albuminous urine may give a dark porter-brown colour, which might be mistaken for that which sugar produces.

(3) Another reduction test was introduced by Braun, and strongly advocated by the late Sir George Johnson. Liquor potassæ and a solution of *picric acid* (carbazotic acid or trinitro-phenol) are added to the urine. Heat is then applied, and when the boiling-point is reached, the picric acid is turned into the deep claret-red picramic acid. Half the quantity of solution of potash should be added to the urine, and half a drachm of saturated solution of picric acid added to this. The reaction is produced by kreatinin as well as glycose; but its chief practical drawback is that the change of colour is not nearly as plain as in Moore's test; still less in that by copper. The addition of caustic potash to a mixture of urine and picric acid always deepens the colour; when heated the tint becomes still darker, and it is far from easy to distinguish between this normal reaction and the production of a somewhat darker and redder tint, with or without turbidity, when a small amount of sugar is present.

(4) Of other tests for glycose, that which depends on the reduction of *bismuth* by carbonate of soda and heat (Böttger's test) was formerly much used in Germany. Like the cupric and picric tests, this depends on deoxidation, and therefore is liable to the same fallacy, that any other reducing agent than glycose may produce the same effect.

(5) The earliest chemical test was devised by Cruikshank, and published in Rollo's 'Treatise on Diabetes,' 1798. It consisted in converting the dextrose into oxalic acid by the oxidising action of nitric acid.

(6) Another test, Mulder's, has been recently advocated by Dr George Oliver. A solution of indigo-carmine, with enough carbonate of soda to make it alkaline, is boiled, while a drop or two of saccharine urine is added, and after a few seconds kept hot, but without boiling, by raising it above the flame for one minute. The blue colour of the solution will become violet, red, and finally pale yellow. On shaking the test-tube the blue colour returns.

(7) The *fermentation test* was first applied by Dobson, of Liverpool, in 1779, who thus showed the true chemical nature of the "honey urine" of Aylow, the sweet urine of Willis. A small quantity of yeast, which must be first thoroughly washed, so as to remove any adhering starch or sugar, is added to the urine, and this is set aside in a warm place with a control glass. When sugar is present, it is gradually decomposed into alcohol and carbon dioxide. The latter, if found in any quantity, is given off as a gas, and may be readily collected. For this purpose all that is needed is that the urine should be made to fill a test-tube, and that this should be then inverted in a saucer, and kept in position by a clamp. After some hours

it will be found that the liquid has receded from the upper or closed end of the tube, in consequence of the accumulation of carbonic acid gas. Ethylic alcohol is also produced by the same vinous fermentation. The great advantage of the fermentation test is that it proves, not the presence of a reducing agent, which may perhaps not be sugar, but the presence of a fermentable carbo-hydrate, which yields carbonic anhydride, and can be nothing but sugar.

(8) The determination of the presence and the amount of grape-sugar in urine by the *polariscope* is scientifically interesting and also exact; it depends on the property which a solution of glycose possesses of rotating the polarised ray of light to the right, whence its name *dextrose*. No other substance at all likely to be in the urine has this dextrogyrate property.

(9) An elegant test, due to von Jaksch, has been much used in our wards of late—the production of crystals of *phenyl-glycosazone*. Two grains of hydrochlorate of phenyl-hydrazine are added to three of acetate of soda, and dissolved in about three drachms of urine, aided by warmth or dilution with distilled water if needful. It is then kept boiling for half an hour in a water-bath, and on cooling in cold water a yellow precipitate falls, which under the microscope consists of radiating needles.

Quantitative analysis.—By the employment of any of these tests it is easy to determine whether a patient is or is not suffering from glycosuria. The copper test is the most used and the most useful, with that of fermentation in case of doubt.

For prognosis and treatment it is desirable to obtain an estimate of the total amount of sugar which is excreted by the kidneys in the twenty-four hours. With this object in view, all the urine which the patient passes in twenty-four hours must be carefully collected and measured, for the amount of sugar present is by no means uniform throughout the day. It is therefore from the mixed twenty-four hours' urine collected in a single vessel that a sample must be taken for analysis.

There are two or three methods by which the amount of sugar present in a certain quantity of urine can be accurately determined.

(1) One consists in ascertaining how many minims of urine are required to reduce the whole of the oxide of copper in 100 minims of Fehling's or Pavy's copper solution.

The solution is first measured by a pipette into a porcelain capsule. Into it is then dropped a fragment of caustic potass, of about twice the size of a pea, this having the effect of causing the reduced oxide afterwards to fall in a dense form, so that the colour of the remaining liquid can be more readily observed. The capsule is next heated by a spirit lamp until it boils steadily; a burette graduated to hold 100 minims, with subdivisions, is in the meanwhile charged with the urine, and this is now allowed to flow drop by drop into the boiling copper solution, which is kept constantly stirred with a glass rod. If sugar be present, the yellow or red oxide of copper gradually appears in greater quantity; but as soon as it is formed it settles, leaving the liquid still blue. At length, however, the blue colour is entirely removed; then the operation is suspended, and a glance at the burette shows how much urine has been used. The copper solution is of such a strength that exactly half a grain of sugar is required to decolourise 100 minims of it, so that there is half a grain of sugar in the quantity of urine that has been dropped from the burette. It is then easy to calculate the amount of sugar that must be contained in each ounce of urine or in the whole amount excreted daily. In his book on diabetes Dr Pavy gives a table by which the trouble of making this calculation may be saved. The process takes a very short time, and after a few trials any one can learn to do it with sufficient accuracy. If the urine be highly charged with sugar, it is advisable to dilute it with from two to four parts of healthy urine or of water before employing it for analysis, of course making the necessary correction afterwards.

An ammoniated cupric solution may also be conveniently used for volumetric analysis ('Lancet,' March 4th, 1884), and the results are, for comparison, very accurate, although there is some reason to believe that the presence of ammonia may somewhat alter the reducing power of a solution of glycose.

(2) Another plan is to ferment the urine with a little yeast, and next day to take its specific gravity and to compare it with that of the same urine in its unfermented state. For each grain of sugar per fluid ounce, one degree of density is lost by the process of fermentation. Roberts says that this method yields nearly accurate results, and its performance requires no technical skill; the only objection is that the result is obtained only after the lapse of twenty-four hours.

(3) The *picric acid* test has been used for a quantitative purpose by comparing the tint obtained with a standard solution made of a definite colour by mixing liquor ferri perchloridi with acetic acid and ammonia.

(4) An accurate and convenient quantitative test of grape-sugar in solution is the *polariscope*, or saccharometer, as it is called when so adapted in commerce, for estimating it in large quantity. The only clinical drawback, beside the expense of the apparatus, is the presence of albumen or of oxybutyric acid, which are lævogyrate, and might slightly diminish the amount of rotation due to the sugar. Lævulose is not known to appear in the urine.

The *amount of sugar* contained in the urine in diabetes varies from the smallest trace up to forty-eight grains in the ounce, and the total quantity of sugar excreted daily shows of course corresponding variations. Dr Pavy believes that this proportion is never exceeded, and that when it has been reached, any further increase in the quantity of sugar excreted leads at once to an augmented flow of urine.

Quantity of urine.—A diabetic patient, instead of passing two or three pints of urine in the twenty-four hours, or less, often passes as much as fifteen; and Dr Pavy has himself seen a case in which thirty-two pints were collected and measured in one day. Much larger quantities are recorded, some almost incredible.

Specific gravity, &c.—Since syrup is heavier than healthy urine, so in diabetes the specific gravity of the urine is almost always higher than normal. Instead of being 1015 and 1022, or lower, it is from 1028 to 1040 or more, but seldom rises to 1050. Occasionally, however, we meet with glycose in urine of low specific gravity. In ordinary cases the urinometer gives a rough estimate of the quantity of sugar it contains; but the relations are not constant. If, however, a diabetic patient's urine is diminishing in quantity and also in specific gravity, we may be sure that the amount of sugar is also diminishing.

Diabetic urine is generally pale, and the more so the greater the quantity that is passed. In some cases it looks as clear and colourless as water, but after standing it is apt to become opalescent from the presence of torula. It deposits no urates after it has cooled. Dr Prout used to ask his diabetic patients how long it was since the urine would become thick on cooling; and if such turbidity of the urine had previously been frequently observed, he dated the commencement of the diabetes from the time when it ceased to occur. As diabetic urine dries, it leaves a white crystalline deposit.

Urine containing sugar has a peculiar odour, which was compared by

Dr Prout to that of sweet hay or milk, by Sir Thomas Watson to the smell of an apple-loft. The *Torula cerevisiæ*, or yeast plant, forms in diabetic urine when it is left freely exposed to the air in a warm place; and the sporules of this fungus may be readily detected with the microscope.

Diabetic urine undergoes ammoniacal decomposition more quickly than that of health. *Urea* is not really deficient in the twenty-four hours, though less than normal is found in any given specimen.

When a patient suffering from diabetes is attacked by any intercurrent febrile disease, the urine often, but not always, becomes for the time free from sugar. This is a point of theoretical interest, as tending to the view that the disease depends upon a perversion of the glycogenic function of the liver; for it has been shown that when fever arises in healthy subjects glycogen disappears from the liver.

Glycosuria without diabetes.—If we make it a rule to examine the urine of every patient for sugar as well as for albumen, we seldom come unexpectedly on a case of diabetes, but we not infrequently find slight reduction of copper in otherwise normal urine, and in persons free from symptoms of diabetes. In most cases this is due to one of the causes above described as fallacies (p. 438), but occasionally glycose is undoubtedly present, though in small amount. Minute quantities occur normally in blood-serum, and from its crystalline character and diffusibility one would expect it to appear in minute quantities in normal urine: some authorities deny its presence even in traces; but Brücke and Bence Jones long ago asserted its presence, and Pavy has brought positive evidence to the same effect ('Croonian Lectures,' 1894, p. 10). Hence "physiological glycosuria" has been supposed to exist, like physiological albuminuria. It has also been asserted that "dietetic glycosuria" can be produced by eating too much sugar; and "dyspeptic glycosuria" has often been described, chiefly in gouty persons at or beyond middle life. This was taught by Sir Henry Marsh and by Graves, by Trousseau, who called a form of occasional glycosuria *glycosurie alternante chez les goutteux*, and by many other physicians.

But at present it is safest (as in the case of albumen) to regard the presence in the urine of sugar, if in quantities appreciable by the cupric test without concentration or extraction, as pathological; indeed, excluding the fallacies produced by excess of creatinin and otherwise, which are mentioned above, the conclusion is accurate.

If small quantities of sugar sometimes appear, and vanish again under dietetic treatment, it is not unlikely that these are incipient cases, which will develop into the slighter forms of diabetes often met with in persons who are past middle life.

However this may be, there are no other diseases than diabetes which produce glycosuria, as albuminuria is produced by cardiac lesions, fevers, and the other morbid conditions to be enumerated in the chapter on Bright's disease. The only partial and occasional exceptions to this are—(1) temporary glycosuria from inhalation of chloroform and ether, probably due to the presence of glyconuric acid; (2) "cyanotic" glycosuria after paroxysms of whooping-cough, asthma, or epilepsy; (3) during pregnancy; (4) after injuries to the head. The last group is of great physiological interest, as we shall presently find (p. 456); but, like the "artificial diabetes" which it resembles, the glycosuria is only temporary. Traces of sugar have sometimes been recorded in cases of fever and of dyspepsia, but they are transitory, and perhaps the reduction observed has not been always due to glycose. (5) Poisoning by *curare* produces glycosuria in animals.

In another class of cases the ordinary symptoms of diabetes are absent, although the presence of sugar in the urine is persistent. But there is no definite boundary line between such cases and those in which the most marked symptoms are present; indeed, the same patient may come in turn under each category.

The distinction, therefore, between mild and severe cases of diabetes, based on the presence or absence of symptoms, is not to be regarded as a natural or safe division; for, as a German writer who adopts it remarks, "fatal complications are not rare in slight cases." The same treatment is beneficial in both kinds of cases, and the same name should be given to both; in fact, persistent glycosuria is diabetes.

Symptoms.—The early signs of well-marked diabetes are as follows:—A man finds that his strength is failing him, he knows not why. His appetite is excellent, yet he loses flesh as well as muscular power. Soon he notices that he passes an unusually large quantity of water, and that he is always thirsty. His urine is tested, and it is found to contain sugar.

It is remarkable how well marked the symptoms often are from the beginning. Not unfrequently the patient can fix the beginning of the disorder to a single week. A gradual insidious onset is the exception with patients under sixty, and it is only in the latter half of life that we come accidentally upon diabetes.

The *muscular weakness* is often extreme, and is by no means a mere result of the wasting of the muscles which generally accompanies the disease. In a series of experiments which Dr Pavy made with various kinds of diet on a man affected with diabetes, he found that, as soon as the patient was allowed food which increased the quantity of sugar in the urine, he complained that he had no life or energy in him. Another patient when admitted was so weak that he could not stand alone; after about three weeks under treatment he had regained strength, so that he ran to the end of the ward and back to show what he could do.

Loss of virility is a frequent effect of the disease, and in women the suppression of the catamenia.

Thirst is another of the earliest and most persistent symptoms of diabetes. The patient generally drinks from eight to twelve pints a day, but sometimes as much as twenty-five or more. Yet even this does not satisfy the craving. The mouth and fauces are also the seat of a sensation of dryness, which causes great discomfort. Dr Pavy says that the way in which the patient keeps rolling the tongue about in the mouth, and the sound which it produces by sticking to the palate from time to time, may be recognised as signs of the disease. The tongue after a time becomes "raw," red, and unnaturally clean, and sometimes it is fissured. Occasionally a sensation is experienced of a sweet taste in the mouth.

Increased *appetite* for solid food is not so constant a symptom as thirst, and in the later stages of diabetes there may be a loathing of all kinds of food. The teeth generally become carious, and the gums swollen, loose, and inclined to bleed. Occasionally vomiting and symptoms of dyspepsia are present, but, as a rule, the digestive powers of persons suffering from diabetes are remarkably good.

Owing to the large amount of nitrogenous food taken, *urea* is formed in abundance. It was formerly supposed that the urine of diabetic patients contains much less urea than in health; but it has been shown that this was a mistake, for although each fluid ounce is poor in urea, there are so many ounces passed that the total amount of urea voided in the twenty-four hours is as great as, and often greater than in health. We shall presently see that, at least in some cases of diabetes, a part of the sugar is formed from the proteids of the food, or rather from the peptones into which they pass before they are absorbed; and these peptones split up so as to produce two series of substances, of which the one has its final term in sugar and the other in urea. A part of the urea excreted is no doubt derived from food, but from the nitrogenous tissues; but Dr Dickinson found that in at least one case of undoubted diabetes, although the actual daily quantities of urea and sugar varied greatly, yet the proportion of surplus urea to sugar was almost constantly as 1 to 6·1. Dr Ringer many years ago arrived at the conclusion that during abstinence, or under a non-nitrogenous diet, the average amount of urea and that of sugar excreted (whether by different patients or by the same patient at different times) had a constant ratio of about 1 to 2.

The daily amount of *uric* (lithic) *acid* which is excreted by diabetic patients does not appear to be increased under an animal diet.

Dr Ringer found that in diabetes the ingestion of *non*-nitrogenous food was followed by a marked increase of urea, as well as of sugar in the urine.

The *bowels* are usually constipated in diabetes, the fæces being dry and hard. But diarrhœa sometimes occurs, and it may lead to a state of prostration which is the immediate cause of death.

Diabetic patients often complain of chilliness; the *temperature* of the body is as a rule lowered, bearing some proportion to the severity of the disease. In one very severe case recorded by Dr Dickinson it varied from 93·6° to 94·8°, and when fatal pneumonia set in, the thermometer only rose to 97·8°. In another case the same disease was attended with a temperature of 103·2°. We shall presently see that in many cases the approach of death is preceded by a fall of temperature.

The *skin* is usually dry and harsh. The cuticle of the palm feels hard, and the furrows have a peculiar white appearance. As a rule there is no sensible perspiration through the whole course of the disease, but occasionally profuse sweats occur. The fact that the subcutaneous tissue sometimes becomes œdematous may be the result of anæmia, but when it occurs, the presence of albumen in the urine should be sought for.

One of the most marked symptoms of advanced diabetes is *emaciation*. The features acquire a peculiar drawn, pinched look, by which the disease may often be recognised.

On the other hand, many diabetics are fat, with smooth skin and pink faces, in striking contrast with the wasted and haggard aspect of the severer form and the latter stages of the disease. Roberts says that one of his patients weighed more than fifteen stone when he had been passing twelve pints of highly saccharine urine for some months, and that one of Prout's patients weighed twenty-three stone.

The patient's breath has a peculiar *sweet smell*. Dr Dickinson says that this is connected with a constipated state of the bowels, and often precedes the near approach of fatal coma.*

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* The late Dr B. G. Babington, when he came down to Guy's Hospital to take in

The *knee-jerk* is not unfrequently absent in diabetics, and this may occur without other signs of peripheral neuritis; but occasionally we see patches of anæsthesia, neuralgia, loss of power and trophic changes in the skin and nails. These symptoms most often affect the lower limbs, but in a patient under the writer's care (Philip Ward, No. 29, August, 1899) the arms were also affected, and on the right hand the nails were deformed and whitlows appeared.

Soon after the invention of the ophthalmoscope, "patches" in the fundus of the eye were recognised as pointing to something wrong with the urine, and they have now been recognised in diabetes as due to retinitis not unlike that of Bright's disease, or to retinal hæmorrhages.

Latency.—It is very rare for sugar to be discovered in the urine of persons who believe themselves to be perfectly well.

Of this Bence Jones met with an instance. A gentleman noticed some little white bodies in his urine, and consequently had it tested. They proved to consist of epithelium from the bladder, but there was sugar in the urine, and this continued to be the case whenever it was examined afterwards. He was a stout man, and remained in good health.

Another case, mentioned by Griesinger, is that of a medical student whose urine was saccharine during the whole of one winter, while he was living in a moist and foggy locality in Switzerland. He had no other symptom of diabetes, and both before and afterwards the urine was often tested and found normal.

Mild and severe cases.—Most of our cases of diabetes belong to one or other of these two categories, although there are many others of intermediate severity. Apparently mild cases may after months or years become severe, and develop one of the complications to be presently described; and, on the other hand, patients suffering from diabetic symptoms in their most marked form may, under judicious treatment, lose most of them, and remain for years in a state of comfort. There is no reason to suppose that the two forms have a different pathology; and it is most undesirable to separate the more favourable from the more serious cases of what is the same disease; for that would mean that "persistent glycosuria" is not "true" diabetes, and would make the prognosis of "diabetes" almost hopeless.

Subject to this caution, we may recognise the two forms of the disease above mentioned.

The milder cases occur for the most part after fifty, and their onset is less marked. The patients are well nourished, of pinkish complexion, with furred tongues, disposed to obesity, and sometimes to dyspepsia or to gout. With moderate dieting the sugar disappears from the urine, and the prognosis is favourable.

The severe cases are met with in children and young adults. They begin with well-marked symptoms, and run a comparatively short and malignant course. The patients soon become thin and meagre, with raw tongue, harsh dry skin, ferrety eyes, and insatiable thirst. Even strict diet does not prevent sugar appearing in the urine. The symptoms are ingravescent; and if acute phthisis does not intervene, they die by coma after a course measured only by months.

Complications.—One of the most distressing symptoms of diabetes is *pruritus vulvæ* in women, often attended with papular or weeping dermatitis. This pruriginous dermatitis is set up by the local irritation of the

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patients on a Wednesday morning, could tell at once whether there was a case of diabetes among the *applicants by the characteristic odour.—C. H. F.*

sugar in the urine; and the orifice of the urethra and the glans penis a sometimes excoriated in male diabetics, though this is comparatively rare.

A peculiar non-pruriginous form of papular eruption on the skin has been occasionally observed in diabetes. The first case was recorded by Addison and Gull among their cases of xanthelasma ('Guy's Hosp. Rep.,' 1851, p. 268); others have been called lichen. This rare complication will be discussed under the section on Xanthelasma in the second volume.

Carbuncles and *boils* are apt to arise in patients whose urine is saccharine. Prout stated that in his experience diabetes always accompanied carbuncles, but this was too sweeping a statement. The importance of remembering the liability of diabetic patients to carbuncular affections is well shown by a case of Sir William Gull's, which is related by Dr Pavy. A medical man was suffering from cerebral symptoms, for which he intended to apply a blister to the nape of the neck. His urine contained sugar, and on this account he was cautioned against doing so. However, the blister was employed, and a large carbuncle soon developed itself, which proved fatal.

Prout also believed that glycosuria may be a temporary result of affections of this kind. A middle-aged patient of his told him that for a long period he had been subject, at intervals of a year or two, to boils and carbuncles, and that during such attacks he always passed a quantity of saccharine urine, whereas at other times the secretion was natural. Later writers also have given cases in which patients have had sugar in the urine only while they suffered from carbuncles or boils.

Gangrene of one of the lower limbs, resembling senile gangrene, is also sometimes associated with a saccharine state of the urine. This is a fact which has been especially insisted upon by the surgeons of Dublin; several cases of the kind have occurred in Guy's Hospital, and have proved fatal.

Defective accommodation is another symptom which is common in diabetic patients, due to impairment of the power of the ciliary muscle; in such cases Dr Pavy has found that the application of Calabar bean to the conjunctiva is very beneficial.

Sometimes sight is affected by the formation of *cataract*. Many years ago Mr France published several cases of this kind in the Ophthalmic and the Guy's Hospital Reports for 1859 and 1860 respectively.* Diabeti cataract has acquired special interest from experiments made by Dr We Mitchell, in which frogs were immersed in a saccharine solution, with t result that the crystalline lens became opaque. This, however, seems to b only a curious coincidence, for the opacity of the lens in the frogs is due t exosmosis, and disappears in distilled water, whereas the cataract of diabete is due to the same anatomical change as in other cases.

Other occasional causes of impaired sight in diabetes are atrophy of th optic discs, or punctiform retinitis, or opaque patches from hæmorrhage.

Phthisis.—The most important complication of diabetes, and a frequen cause of death, is an acute form of phthisis. Of forty fatal cases diabetes in Guy's Hospital, in seventeen the immediate cause of deat was pulmonary tubercle. That this is the true nature of the lesion, ar not chronic pneumonia, as believed by Addison and by Fagge, is now a mitted. Tubercle bacilli have been repeatedly found in diabetic phthisis and apart from this evidence, the locality of the affection in both apices, t excavation, and the frequent presence of caseous material in the lungs and other organs, render it certain that the chronic affection of the lungs in diabetes is true phthisis. It runs a rapid course, and is seldom marked by hæmoptysis or high fever.

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* Lecorché's paper on the same subject appeared in the 'Archives générales de méd e' in May, 1861, but cases had been noticed before by von Gräfe.

Another frequent cause of death in diabetes is ordinary lobar or fibrinous *pneumonia.* This was present in ten out of the above forty cases. In four of them the hepatised parts were passing into a gangrenous state.

The present writer was indebted to Mr Kelbe's help in the analysis of the anatomical reports of seventeen cases of diabetes which consecutively proved fatal in Guy's Hospital by inflammation of the lungs between 1881 and 1887. The disease affected one or both apices in ten cases; with vomicæ but no other sign of tubercle in four, with miliary tubercles in the lungs or elsewhere in the other six. In two cases there was acute hepatisation of the base without any sign of phthisis. In the remaining five cases there was less acute pneumonic consolidation not affecting the apices; this was described as caseous in one case and as necrotic in three, in one of which it was caused by the presence of particles of food.

Diabetic coma.—In six of the forty fatal cases before mentioned the immediate cause of death was the supervention of *cerebral symptoms.* These generally began with drowsiness, and in a few hours passed into coma. Once or twice there was more or less well-marked delirium, or even convulsions; the pulse was often very feeble, and the temperature low. Indeed, in cases of this kind the state of the patient is often one of collapse quite as much as of coma.

This "diabetic coma" has been ascribed to the presence of *acetone* in the blood. But acetonæmia is not always present in diabetes, nor is it always, when present, accompanied by cerebral symptoms.* Moreover if, as is probable, it is derived from nitrogenous metabolism, it could not be present in sufficient amount to act as an intoxicant dose. The same negative view is taken by Dr Pavy, Prof. Karl Grube, and Dr Saundby.

During diabetic coma the following abnormal constituents have been discovered in the patient's urine:

(1) Acetone (C_3H_6O), dimethyl-ketone ($CO,2CH_3$).
(2) Aceto-acetic acid ($C_4H_6O_3$), which yields acetone and carbon dioxide.
(3) Crotonic acid ($C_4H_6O_2$).
(4) Oxybutyric acid ($C_4H_8O_3$).

The acetone, which imparts its odour to the breath, and which gives a crimson colour to the urine when treated with perchloride of iron, is the product of aceto-acetic acid, which may be extracted from the urine by ether after acidulation with dilute sulphuric acid.

Since acetonæmia is incapable of explaining diabetic coma, it has been proposed to account for it by oxybutyric, and perhaps other acids, diminishing the normal alkalinity of the blood. The phenomena would then be comparable to those produced by giving dilute mineral acids in large quantity to dogs (Walter and Schmiedeberg). This would prevent the blood dissolving carbon dioxide, which would therefore accumulate in the brain and other tissues.†

Recent observations have shown that the presence of β-oxybutyric acid is the most constant condition in diabetic coma and that its injection causes similar symptoms in dogs (Gruber, 'Journal of Physiology,' 1900).

1. Diabetic collapse, with coldness, lividity, and subnormal temperature. The pulse is rapid, and there is little dyspnœa. The patients are usually elderly, stout, and long dia-

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* See an excellent account of Acetonæmia and Lipæmia in Diabetes, in Dr Gamgee's 'Physiological Chemistry,' vol. i, pp. 168—172.

† Dr Dreschfeld, in his Bradshaw Lecture before the College of Physicians ('Lancet,' August, 1886), described three types of diabetic coma (not identical with those of Frerichs).

betic. The attack comes on after fatigue (as in Prout's cases), and proves fatal rapidly—within twenty-four hours as a rule. The heart is often found fatty, but no acetone or other abnormal constituent but glycose is found in the urine.

2. A rare form, closely resembling drunkenness, with staggering gait, incoherent speech, and disturbed mental faculties. Acetone is often present in the urine and the breath, and sometimes alcohol.

3. The most frequent form, with muscular weakness, drowsiness, rapid breathing, and at last coma. There is the acetone smell in the breath and urine, and not only aceto-acetic but also crotonic and oxybutyric acids can be demonstrated in the urine.

As described by Prout, and afterwards by Kussmaul, the condition of coma (as distinct from collapse) agrees closely with the third of these varieties. Breathlessness without dyspnœa (" air-hunger "), occasional convulsions, and a subnormal temperature are its leading features. It occurs mostly in young patients, and early in the disease.

In addition to the six cases mentioned above, in which death was preceded by cerebral symptoms, there were two in which it was quite sudden, but probably due to a similar cause. The fatal symptoms developed themselves very shortly after the admission of the patient into the hospital; in five of the eight death took place within five days of admission, and in three it occurred either on the day of admission or on the following day. The cause of the sudden fatal termination was, no doubt, the fatigue and excitement which the patients underwent in coming to the hospital. The same thing was noticed long ago by Prout, who says that four of his private patients sank almost immediately after coming to London from the country to consult him, and one of them was very near dying in Dr Prout's own house.

Pavy has observed that those cases of diabetes in which the disease has been kept under control by treatment are particularly apt to end at last in convulsions, collapse, or coma; whereas when the disease is allowed to run on unchecked, the supervention of phthisis is more likely.

Experience has shown that the cause of death in diabetes is very uncertain unless an autopsy is made. Two diabetic patients in Guy's Hospital died with cerebral symptoms, but in one case the pelves of both kidneys were dilated and inflamed, with commencing renal suppuration;[*] and in the other there was extensive hepatisation of the left lung.

As above stated, of the forty fatal cases at Guy's Hospital between 1860 and 1874, seventeen patients died of phthisis, ten of acute pneumonia, and six of coma ('Reports,' 1875); in thirty-six reported by Dr Frederick Taylor from the same hospital between 1875 and 1882 (*ibid.*, 1881, p. 152, note; and 'Path. Trans.,' 1883, p. 371) twenty-eight deaths were from coma, three others probably from uræmic coma, and eleven from phthisis or pneumonia. Dr Mackenzie reported, among thirty-seven fatal cases of diabetes at the London Hospital (1874-83), nineteen deaths with coma, five of these and ten others with phthisis, and eight from accidental causes

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[*] The inflammation of the kidneys in the former of these two cases was so exactly like what occurs in cases of stricture and other diseases of the urethra or bladder, that these parts were very carefully examined. The urethra was perfectly healthy; the *bladder*, on the other hand, was greatly hypertrophied. This led me to consider whether the increased thickness of the coats of this viscus could be due to the augmented work it had had to perform in consequence of the over-secretion of urine. In the second case, therefore, we looked at the bladder with much interest, and found that it also was markedly hypertrophied, and that its mucous coat protruded between the muscular fasciculi, so as to form numerous sacculi. Probably hypertrophy of the bladder may be found to be frequently *present in* diabetes.—C. H. F., 1882.

Morbid anatomy.—The viscera of those who have died of diabetes have a decidedly sweet smell, resembling that observable during life in the urine and breath; in one case this odour was still perceived, although the patient died of typhus, and his urine (which also retained the sweet smell) had been free from sugar some days before death.

In some cases of diabetes *the blood* has been found creamy from the presence of fatty molecules, and fat-embolism has been found in the lungs (Sanders and Hamilton, 'Edin. Med. Journ.,' July, 1879). But *lipæmia* (as this condition of the blood has been called) is certainly not constant. It was first noticed by Dr Rollo in 1778, and afterwards by Dr Babington the younger ('Cycl. Anat. and Phys.,' i, 422). Dr Pavy regards it as a physiological effect of the abundant food that is taken by diabetics.

The *liver* is usually large, and fatty—sometimes cirrhotic, but this is no doubt an accidental coincidence.

The *pancreas* is generally found atrophied or diseased—a fact of great pathological interest. Most often there is induration from interstitial inflammation, and sometimes carcinoma, or cystic degeneration, or abscess, but the pancreas is often found perfectly healthy in patients who have died of diabetes.

The *brain* is unaffected: in two of our cases dilated perivascular spaces were seen, as Dr Dickinson has described, around the blood-vessels of the bulb and pons. These dilated perivascular spaces are not, however, characteristic of diabetes, and are probably not even present during life, as seems proved by the observations of Drs Taylor and Goodhart (see 'Guy's Hosp. Rep.' for 1877, and 'Path. Trans.,' vol. xxxiv, pp. 328—396, and the report, p. 397).

Dr Hale White (*ibid.*, vol. xxxvi) has shown that the pigmentation, induration, and other changes of the *semilunar ganglia* reported in diabetes are present in other cases ('Journ. of Phys.,' vol. viii, p. 70).

The *kidneys* are not infrequently large, soft, and fatty; they are occasionally affected with chronic tubal nephritis or some other form of morbus Brightii, or, still more rarely, with suppurative inflammation.

In 44 autopsies on diabetes at Guy's Hospital, reported by Dr H. J. Campbell ('Guy's Hosp. Reports,' vol. xliv, p. 207), the *kidneys* were usually large, sometimes "coarse" in aspect, and occasionally fatty. The presence of glycogen in the looped tubes, as asserted by Frerichs and Ehrlich, was not clearly demonstrable. Sections stained with iodine, logwood and eosin, silver or osmic acid, showed very often the necrotic degeneration of the epithelium described by Ziegler. The *bladder* was hypertrophied in 13 cases as described by Dr Fagge (see foot-note on p. 448).

The *lungs* were affected in 23 cases; in 18 there was ordinary phthisis of the apex, in 1 gangrene, and in 4 acute œdema or pneumonia.

In a series of autopsies on diabetic patients collected by Dr Windle ('Dublin Medical Journal,' September, 1883) the *liver* was reported normal in 84 cases, enlarged in 57, congested in 40, fatty in 15, and tubercular in 2.

The *kidneys* were normal in 115, enlarged in 88, fatty in 35, affected with tubal nephritis in 6, cirrhotic in 10, and lardaceous in only a single case. The *bladder* was normal in 20, and hypertrophied in 13 cases.

The *lungs* were normal in 81 cases, and congested or œdematous in 37 more, making 118 in which they were free from organic disease. In 136 they were phthisical ("tubercular" in 109, "pneumonic" in 27), to which

17 cases with cheesy masses, 12 with vomicæ, 3 with caseous bronchi glands, and 8 with miliary tubercle may be added, making a total of 178 In 24 cases there was acute lobar or catarrhal pneumonia, and in 3 gangrene, while in another 3 fat-emboli were discovered. The *heart* wa normal in 70, large in 4, small in 9. The *brain* was normal in 91, and perivascular changes were found in 11. The sympathetic ganglia were normal in 10 cases, and "cirrhotic" in 5.*

Physiology of glycose.—In 1848 Claude Bernard published the discovery that a substance which he called glycogen is constantly present in the liver of man and other animals, and that abundance of grape-sugar may be obtained from the liver and the hepatic vein after death. He also stated that there was a much larger amount of this sugar in the hepatic than in the portal and other veins. Next he ascertained that though the glycogen of the liver is greatly diminished by starvation, yet that its presence does not wholly depend upon that of carbohydrates in the food. Lastly, he showed that when the liver of a recently killed animal was washed out by the injection of a stream of water through the portal vein until its sugar was carried away, the same glycose reappeared in abundance after a few hours.

Glycogen was isolated as a white powder by Bernard in 1857, and its composition and reactions place it in close relation to vegetable starch. His theory was that the sugar which is absorbed from the stomach and intestines in the form of glycose, as the product of all digestible carbohydrates—whether vegetable starch from bread, or animal starch (glycogen) from meat, whether cane-sugar, fruit-sugar, malt-sugar, or milk-sugar—is conveyed by the portal vein to the liver, and is there changed by dehydration into glycogen, and stored in this indiffusible form; and finally, that it is gradually reconverted into glycose by the action of a special ferment, carried through the hepatic veins to the right side of the heart, and thence conveyed to the lungs, where it is burnt off.

The last statement has been modified by increasing knowledge; neither glycose nor any other combustible food is oxidised in the lungs. Th process of oxidation and heat-making takes place neither in the lungs no in the blood, but in the tissues, and particularly in the most active tissues the muscles and the glands.

Moreover it was ascertained by Pavy (and afterwards confirmed b the late Robert McDonnell, of Dublin, and by Ritter and others abroa that the large amount of sugar found soon after death in the liver i formed *after* its removal from the body. If immediately on an anim being killed the liver is cut up and thrown into boiling water, so as destroy the ferment, only traces, and sometimes not even traces, of sug can be found. This is, in fact, the method on which is based the ordina preparation of glycogen. Pavy also showed, by passing a catheter dow the jugular vein into the right auricle, that blood obtained straight fro the hepatic veins of a living dog or rabbit was approximately free fro sugar. These experiments certainly established the conclusion that there is no sugar in the liver during life at all comparable to the large quantity obtained a few hours after death; in fact, the active stream of blood which flows through the portal capillaries would immediately wash out the sugar if present, like the stream of water which Bernard used in the dead liver.

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* Valuable information on the morbid anatomy of diabetes will also be found Frerichs' treatise, where he describes and tabulates (pp. 134—183) 55 cases under his o care; in Dr Saundby's "Bradshawe Lecture" ('Brit. Med. Journ.,' Aug. 23rd, 189 reprinted in his monograph; in Dr Finlay's report of 20 cases from the Middlesex H *pital* ('Path. Trans.,' xxxiv, pp. 336, 341); and in Dr. R. T. Williamson's article in t '*Medical* Chronicle,' May, 1897.

We may, however, doubt whether Pavy's experiments, skilful and long continued as they have been, have established the further conclusion which he has drawn from them, namely, that glycogen is not reconverted into sugar during life, but undergoes transformation into fat. The varying amount of glycogen in the liver, its increase after food and diminution after fasting, seems to show that it is constantly undergoing some transformation, and the storage of fat by the liver is in direct, not inverse ration to that of glycogen. Moreover Pavy agrees with other physiologists in admitting the normal presence of glycose, though in small quantity, in the blood; and, as in the case of urea or the alkaline urates, we must remember that with the considerable mass and rapid circulation of the blood, large renewal and equally large abstraction of its constituents are continually taking place. Fat, sugar, urea, urates and kreatinin, bilirubin and tyrosin, may be excreted so rapidly as to form a large amount in a short time, and are thus rightly regarded as of great importance in the economy, although it is only possible to demonstrate their presence from any single specimen of blood in quantities which seem at first insignificant.

It is certain that the glycose absorbed after every meal is, for the most part at least, stored as glycogen in the liver; otherwise it would be found periodically increased in the blood, which experiments on animals prove is not the case; and if present in the blood it would, from its high diffusibility, equally certainly appear in the urine, so that glycosuria would be a normal and not a pathological event.

Most physiologists, therefore, believe that the glycogen stored in the liver is slowly converted into glycose, and gradually carried off in the blood, to be oxidised in the tissues; and that this process is so complete in health that nothing but the merest trace appears in the urine.

The pathology of glycosuria.—We have next to inquire what leads to the enormous excretion of glycose in diabetes, which may amount to two pounds in the twenty-four hours.

The first and natural hypothesis was that the kidneys secreted the abnormal constituent glycose, as they were supposed to secrete the other constituents of healthy or morbid urine. But it is now ascertained that the kidneys do not secrete, in the sense of manufacturing, the urea, nor probably the uric acid and kreatinin, any more than the chlorides and phosphates; they only secrete in the sense of separating them from the blood of the renal artery. And it is well established by observation and experiment that the same is true of the excretion of leucin and tyrosin, of oxalates, and of bilirubin, as well as of sugar—in acute yellow atrophy, in the formation of calculi, in jaundice, and in diabetes respectively. The first point, then, established in the natural history of diabetes was that it is not a disease of the kidneys at all, but depends upon some antecedent derangement of the chemistry of the body. In the case of so diffusible a body as glycose, glycosuria is the natural and inevitable result of preceding glycosæmia.

We next inquire to what cause is due the excess of sugar in the blood.

Obviously it must depend upon one or more of the following causes: either the production of sugar must be increased, or it must be saved from being stored away as glycogen, or its destruction by oxidation in the tissues must be diminished.

(1) We shall see that in most cases of diabetes it is possible greatly to reduce the amount of sugar in the urine, and therefore, we infer, of sugar in the blood, by cutting off the supply of carbohydrates in the food; but in many cases sugar, though diminished, is still present, even under the strictest diet. Animals, when fed on nitrogenous food alone, are capable of forming glycogen in the liver, just as they are capable of forming fat, by splitting up the complex proteid molecule into a nitrogenous and a non-nitrogenous moiety; and no doubt the glycose found in the severe cases of diabetes is formed from the nitrogenous diet. No increase in the amount of carbohydrates eaten will produce glycosuria in a healthy animal or man.

We can thus see why, in the worst cases of diabetes, it is of little service to limit the ingestion of food. Whatever carbohydrates are taken appear at once in the blood and run off by the urine; and if all such food is rigidly abstained from, the proteids themselves furnish glycogen to be excreted; starvation would be the only physiological cure in such cases. Hence a diabetic, though eating with a good appetite and digesting without discomfort, is nĕvertheless being slowly starved.

(2) Defective power of converting glycose into the indiffusible glycogen by the liver is theoretically a sufficient cause for glycosuria; and we have an attractive explanation of the way in which this might occur. After Bernard had established the glycogenic function of the liver, he made the equally brilliant discovery that by puncturing a definite spot in the floor of the fourth cerebral ventricle we can produce glycosuria, or, as it is called, artificial diabetes. The explanation is that the vaso-motor centre of the liver being paralysed, the hepatic artery dilates, the flow of blood through the liver is more rapid, and hence the passage of glycose into the circulation is augmented. One objection to the theory is that sugar is carried to the liver by the portal vein, not by the hepatic artery; while the distribution of the portal branches, as well as their greater size and number, makes it probable that it is from the portal rather than the arterial blood that glycogen is formed. Another is that the duration of artificial diabetes is very limited, and that injury or disease affecting the fourth ventricle appears to produce transitory glycosuria rather than permanent diabetes. Again, Pavy has shown experimental ground for supposing that the glycosuria which follows Bernard's puncture is not due to the mere dilatation of the hepatic artery which follows, but to the increased presence of oxygen, for he has obtained glycosuria by causing arterial blood to flow through the portal vein.*

* A broad ground of objection has been taken to any theory which ascribes glycosuria to increased production of sugar; namely, that even if all the glycose which can be produced by digestion of the proteids taken in twenty-four hours by a diabetic patient under strict diet were supposed to pass through the liver without any of it being stored as glycogen, it would not account for the enormous amounts which are passed in the urine. This conclusion seems established by observations upon dogs with artificial diabetes; and in human subjects under strict diet it is certainly true of the most severe cases, when much more sugar is passed by the urine than could be the product of the proteids digested, unless we add to them the fixed proteids of the tissues. But this argument, though used by several writers of repute, seems to prove too much. For if a man or a dog with diabetes really passes a greater weight of sugar in the twenty-four hours than is accounted for by the food he eats, the fact is just as conclusive against the theory next to be considered, that of diminished destruction of sugar. The only explanation, if the facts are as stated, must be

Two facts, moreover, prove that increased change of glycogen into glycose is not the only cause of glycosuria. Firstly, the examination of the liver immediately after death in artificial diabetes in animals, and soon after death in the diabetes of man, does not show such constant diminution of the amount of glycogen as we should expect.

Secondly, when there is extreme obstruction to the portal circulation in the liver, the blood must needs go in great part by other channels. Very little glycose would then be transformed into glycogen, the large amount absorbed after digestion would be excreted by the kidneys, and thus glycosuria would result. Bernard himself tied the portal vein in a dog, and thus produced artificial *glycosurie alimentaire*. Dr Lépine (following Couturier) observed the same result in three patients with cirrhosis of the liver (verified after death) who were fed on starchy and saccharine diet, and he did not obtain it in cases of cancer of the liver and of fatty liver in phthisis. This result, however, is far from constant; it failed when tried several years ago by the present writer at Guy's Hospital, and sugar ought far more frequently to appear in the urine in cases of cirrhosis, adhesive pylephlebitis, and portal thrombosis. The same negative result was obtained by Frerichs.

A third objection to glycosuria depending upon the diminished power of the liver to store the sugar which reaches it in the form of glycogen was thus put by Dr Fagge:—"If a patient, whose urine is kept free from sugar by a restricted diet, one day breaks through the dietetic rules laid down for his guidance, and eats an apple or drinks a glass of sweet beer, it will generally be found that the secretion again becomes saccharine. This is no more than might be expected; but now comes a circumstance which is remarkable, and for which some further explanation is evidently necessary. The quantity of sugar that is voided is altogether disproportionate to the amount contained in the apple or the beer; and sugar often continues to be excreted for a long time afterwards. Dr Pavy mentions the case of a patient in whom the disease had been kept under control by strict dieting, and who drank about a pint of cider. His urine thereupon became loaded with sugar, and remained so for a period of two months, before it again became normal. Now the only hypothesis which seems capable of explaining such facts as these is that saccharine or amylaceous food exerts some directly injurious influence, so as to cause the blood to contain an excess of sugar for long afterwards; and, if we believe that the liver is the organ principally concerned in supplying the blood with sugar, we can hardly help inferring that it is the liver on which this injurious influence is exerted. It seems as if saccharine food were a *poison* to a patient who is affected in this way."

In any case, if we assume that in health the glycose which reaches the liver is stored as glycogen, while in diabetes it runs through the portal circulation unchanged, we must find some other normal destination for glycogen, or else all the sugar absorbed from the food will sooner or later be returned to the blood. Dr Pavy finds this destination in fat, but at present his hypothesis cannot be proved, either chemically or physiologically.

that from the fatty elements of food, in some unknown way, more carbohydrates enter the blood and urine as glycose than are ingested as food. After all, the facts are very doubtful. Zülz found that there is never so much sugar excreted in diabetes as answers to the amount of carbohydrates ingested; some is always oxidised and burnt off.—C. H. F.

(3) Let us now see what may be said for the hypothesis of glychæmia, and so glycosuria, being the result of diminished destruction of sugar by oxidation. In health the glycose, which is the product of the digestion of carbohydrates, undergoes an imperfectly ascertained chemical change in the tissues, where it is oxidised into carbon dioxide and water. It is thus eliminated from the body, while the chemical energy of the combination is liberated, and reappears as animal heat and the work of muscles, glands, and other less active tissues. That retention of sugar is not the direct and immediate result of deficient oxidation is shown by the absence of glycosuria, unless in very small amount and under exceptional circumstances, in cases of cyanosis, of laryngeal dyspnœa, bronchitis, and emphysema, cardiac disease and anæmia. In all these cases the antecedent process, whatever it is, which fits glycose for oxidation, must have been performed, and we must suppose that it is in a different molecular condition that a great part of the glycose of the blood remains unoxidised in diabetes.

Another argument that it is not lack of oxidation which keeps the proportion of glycose in the blood so high, is the fact that other oxidations go on as usual in natural or artificial diabetes. Fatty food is perfectly assimilated, and excreted as carbon dioxide and water; Nencke found by experiment that benzole is changed into carbolic acid, Schulzen recovered vegetable salts as carbonates, and Külz showed that even certain carbohydrates were oxidised, as lævulose and inosite. Schulzen has found that in cases of phosphorus poisoning, when oxidation is much hindered, glycose is still split up ready for oxidation, and appears as lactic acid in the urine.

Again, if as each fresh accretion of glycose reached the liver, it were storing its glycogen normally, the diminished destruction of sugar in the tissues would keep the amount in the blood and urine at a pretty uniform quantity, but in diabetics it is well known that the sugar excreted by the kidneys is increased after each meal.

A diabetic patient, Joseph North, under Dr Pavy's care, was placed in succession upon different kinds of food, and his urine was collected and analysed every four hours. As a rule, the amount of sugar excreted in the urine was considerably greater between 5 and 9 p.m. than at any other part of the day; while it was commonly at its lowest point during the night and early morning. These variations were evidently due to the food taken during the day. On one occasion he departed from his instructions, and at 4 p.m. drank some cocoa sweetened with sugar. Between 5 and 9 p.m. of that day he passed 1311 grains of sugar, whereas in the twenty-four hours previously the quantity in equal periods had ranged between 166 and 468 grains; and from 9 to 1 a.m. the same evening it was again only 483 grains.

On the whole, we must admit that the riddle of glycosuria has not yet been solved. It would, of course, be easy to assume, as some have done, several kinds of diabetes: one due to increased production of sugar and diminished deposit of glycogen, another to increased production of sugar with abundant glycogen, a third due to diminished oxidation, and a fourth to diminished preliminary changes of the sugar in the blood or tissues, before oxidation takes place.

But the distribution to each category would be very arbitrary, and in the case of so well-marked and peculiar a clinical condition as that of diabetes it seems more probable that it has a constant origin.

Thus much may perhaps be said. The liver, when rendered hyperæmic by the "diabetes-puncture" or other nervous lesions, manufactures sugar rapidly and abundantly from its glycogen in a well-fed animal or a man, and thus produces a temporary glycosuria, but not the permanent condition of diabetes. There is something—whether a ferment or a want of a ferment in the blood, or some molecular difference in the arrangement of the CHO atoms in diabetic sugar—which prevents its being oxidised, as ordinary dextrose is in health, and thus it accumulates in the blood in spite of free respiration.

Artificial diabetes.—Before quitting this part of our subject we must briefly refer to certain conditions known as "artificial diabetes," or rather artificial glycosuria.

The first of these conditions is the result of Claude Bernard's famous experiment of puncturing the "diabetic centre" in the bulb. *Traumatic* glycosuria is produced, as it is also by other injuries of the nervous system; but the experiment fails if from starvation or other cause the liver of the animal contains no glycogen. The sugar derived from its food apparently does not pass on into the circulation, but is stored as glycogen; while the sugar formed from glycogen in the liver is washed out abundantly by the augmented stream of blood—an argument, as far as it goes, for Bernard's rather than Pavy's view. But this glycosuria is temporary only, as indeed we should expect, for it is clear that after the puncture, in proportion as the amount of glycogen in the liver becomes reduced by the excessive demands made upon it, the glycosuria must pass off; and, in fact, after a few hours sugar can no longer be detected in the urine of animals on which this experiment has been performed. It is true that Schiff found that by dividing the anterior columns of the cord in rats he could make the animals diabetic, and that this condition would last for two or three weeks. But these results scarcely warrant our attributing ordinary persistent diabetes in the human subject to paralysis of the vaso-motor nerves. Division of the splanchnic nerves does not cause glycosuria, though it produces hyperæmia, while poisoning by strychnia does, though it causes constriction of the hepatic vessels. And it seems impossible that the excretion of such large quantities of sugar as are passed by diabetic patients at all hours of the day, and in amounts influenced only partially by the meals, can be due to a mere increased rapidity of the conversion into sugar of the glycogen naturally formed by the liver.

The second artificial kind of glycosuria is *toxic*. It has been observed after poisoning by chloral, morphia, or curare, and after inhalation of chloroform and carbonic oxide gas; but, except in the last case, the presence of sugar in the urine is far from constant.

A physiologically interesting form of glycosuria is that observed by von Mering, as the result of feeding dogs on *phlorizin* ($C_{21}H_{24}O_{10}$), glucoside prepared from the bark of apple, pear, and cherry trees.*

The third kind of artificial diabetes is that caused by destruction of the *pancreas*. Lancereaux first drew attention in 1887 to the co-existence of lesions of the pancreas with severe diabetes; and Minkowski, confirmed by von Mering and other trustworthy observers, has shown that glycosuria can be produced in dogs by destruction of the pancreas, while this effect is prevented if a small portion of the gland is left behind (cf. p. 450).

There is no doubt that the pancreas, like the testis and the kidney, is not a mere secreting gland, but has other important functions. Perhaps, like the thyroid and supra-renal bodies, it keeps in check the occurrence of remote changes in nutrition, and so causes glychæmia.

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* That this is not the result of transformation of phlorizin (or phloridzin) into glycose is shown by the fact that the same glycosuria is produced by giving its derivative phloretin, which is not a glycoside.

But diabetes is, at least in most cases, due neither to injuries of the nervous system, nor to ingestion of poison, nor to disease of the pancreas.

Theory of the other symptoms.—Given glycosæmia, the other pathological effects seem to follow naturally. With a diffusible crystalloid like dextrose glycosuria will at once follow. That in itself leads to polyuria, and then the flow of urine produces dryness of the tissues, constipation, and thirst; the loss of oxidisable food leads to hunger, emaciation, muscular weakness, lowered temperature; while dextrose, or rather some concomitant products of deranged metabolism—as aceto-acetic or oxybutyric acids—act as poison in producing local gangrenous inflammation in the skin, or causing cerebral coma, or preparing a suitable soil for the growth of the tubercle bacillus in the lungs.

Clinical ætiology of diabetes.—Some interesting cases have been recorded in which the urine has been found to contain sugar after *the brain* has been injured, or when it was diseased. As far back as 1854 Dr Goolden published in the 'Lancet' a series of instances of glycosuria following blows or falls upon the head; but in most of them the presence of sugar in the urine was transitory, like that in dogs and rabbits after Bernard's puncture. Dr Pavy, however, mentions the case of a cadet at Woolwich who was attacked with strongly marked diabetes a few days after being stunned by a blow upon the head; and he also observed two instances of diabetes after an attack of hemiplegia. Frerichs recorded a large number of similar cases. Many years ago (about 1867) a little girl was brought into Guy's Hospital dead from fracture of the skull and other injuries in being run over; a patch of hæmorrhage was found by the writer in the floor of the fourth ventricle, and some urine was then collected from the bladder and gave decided evidence of sugar. A striking case in which disease of the fourth ventricle has led to glycosuria in man is one quoted by Trousseau, in which there was a tumour in the floor of this cavity. Similar cases have been since recorded; but, interesting as they are from a physiological point of view, they lend small support to a nervous theory of diabetes.

Like most other diseases, diabetes has been attributed to mental anxiety and "overwork." Sir Hermann Weber met with the case of a gentleman who became diabetic on two separate occasions, at an interval of nine years, under the pressure of anxiety from impending commercia ruin. In other cases recorded by Rayer and Frerichs, diabetes has fol lowed a violent fit of grief or of anger, and Dr Dickinson mentions severa similar cases.

It has been stated by Maudsley that diabetic parents often have insan children, and Dickinson found glycosuria in 17 per cent. of insane persons But Hale White found it in only 4 per cent. of the inmates of Bethlem and in 2·6 at the Surrey County Asylum ('Path. Trans.,' 1883, p. 353) and Savage also believes that insanity and diabetes are unconnecte ('Brit. Med. Journ.,' 1885, vol. ii, p. 1054).

An *inherited tendency* is clearly present in some cases of diabetes Pavy gives numerous instances of this: in one family two sisters and tw brothers; in another a son, his father, and an aunt; in a third a fathe and his two daughters; in a fourth a father and a son; in a fifth two brothers; in a sixth three brothers; in a seventh a brother and a sister; and in an eighth the father, mother, and three daughters; and (to mention only one other case) a mother, grandmother, and four out of five children. Roberts knew a family of eight children, all of whom became diabetic, although the parents were healthy. Frerichs found 39 hereditary cases among 400.

One hundred and twenty-two cases were collected for the writer by Mr J. E. Nevins from the records of Guy's Hospital, and in eight of these there was a history of diabetes in one or more members of the patient's family (6·55 per cent.). Adding cases obtained by the courtesy of the registrars of the three other large hospitals, St Bartholomew's, St Thomas's, and the London, and those recorded by Schmitz and Griesinger, he found 35 hereditary cases in a total of 537 (6·51 per cent.). In one remarkable case the mother and seven of the mother's brothers had died of diabetes.

Many writers, particularly on the Continent, where true *gout* is rare, ascribe a large share to that disease in the production of diabetes. It is said that gouty diabetes is marked by the patient being stout and ruddy and suffering little, as well as by his being elderly. But this means little more than that gout does not exclude diabetes, and that patients over forty, florid and dyspeptic, are by many physicians called gouty without more ado. If we inquire how many diabetics have suffered from inflammation of the great toe, or from chalkstones, we find the number not above that of the accidental coincidence of two not uncommon diseases, both of which affect men rather than women, and those over forty rather than younger persons. It is instructive to observe that some physicians have believed in the antagonism of gout and diabetes.

Distribution.—Diabetes is said to be nearly twice as common in Paris as in London, less common in Ireland and Scotland than in England,* more common in Europe than in America, and in Saxony or Thüringen than in Berlin. It is more common in Ceylon and parts of India than in China, Mauritius, or Berbice. Jews are particularly liable to it,† and negroes are almost exempt. It is said to occur frequently among Parsees and wealthy Hindoos.

It is more frequent in private than in hospital practice, and as a rule private patients are older than those in hospital. Trousseau used to say that when there came into his consulting room a sleek, plump, pink-faced patient, dressed in black with a white neckcloth, he was sure to be a notary and diabetic.

Age and sex.—Diabetes rarely attacks young children, and is most frequent in the latter half of life. Among 1360 private patients who came to Pavy, diabetes began under ten years old in 8, between ten and twenty in 57, between twenty and thirty in 97, between thirty and forty in 224, between forty and fifty in 339, and between fifty and sixty in 418. Even between sixty and seventy the number was 182—very large considering the smaller number of persons living over sixty compared with those between twenty and forty. Above seventy there were 35 patients, one in whom the disease began when he was over eighty. The youngest patient he ever saw was an infant twelve months old ('Brit. Med. Journ.,' Dec. 5th, 1885).

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* Notwithstanding Sir Charles Scudamore's statement as to the greater frequency of diabetes in Scotland, which misled Dr Fagge, and is, as Dr Saundby remarks, refuted by the Registrar-General's returns.

† Frerichs, for instance, had 102 Jews among his 400 diabetic patients.

Diabetes is far more common in men than in women: in the 537 cases mentioned above, by more than two to one. In Pavy's private cases the proportion was 24·5 to one, and the total was 966 to 394. In every decade (excepting the children under ten) the number of males was greater than that of females. Diabetes is particularly rare in old women, and the predominance of the male sex in this, as in some other diseases, does not appear during childhood.

Prognosis.—In its well-marked forms diabetes is always a serious disease, and is often rapidly fatal if the patient be under middle age.

Cases are sometimes met with in which death follows a few weeks after the first symptoms of the disease appeared. Sir George Paget had a patient, a Cambridge undergraduate, supposed to be in perfect health, who took part in athletic sports, and came in second in a foot-race, within twelve days of his death from diabetes. The writer had once a young man under his care who died from diabetic coma after only two months' illness. Such cases of acute diabetes are, however, rare; the duration of diabetes is seldom under three years, and often much longer. Prout said in 1848 that, among nearly seven hundred patients whom he had seen within thirty years, he then knew of but two in whom the disease had been fully developed ten years before. Dr Dickinson, however, mentions the case of one patient in whom the urine was constantly saccharine for fifteen years, and Dr Pavy a similar case which lasted twenty-five years.

The *age* of the patient has a greater influence on the prognosis than any other condition. Children who are attacked by it never live to grow up. In young men it is still a dangerous disease. After forty years of age our patients may be treated with hope, and at fifty with a probability of improvement or even recovery. Old people sometimes pass saccharine urine for years without appearing to suffer from it; but in such patients the urine is seldom excessive in quantity, nor other symptoms severe.

At a middle period of life, say between forty and sixty, the prognosis of a case of diabetes, when it first comes under observation, must mainly be based upon the degree of severity of the symptoms, particularly emaciation. It is always a favourable sign if a diabetic patient is stout and well nourished.

If sugar is present without much increase in quantity of the urine the prognosis is better than when there is polyuria.

It is generally supposed that the detection of albumen in the urine in addition to the sugar is a serious indication, but Dr Pavy says that he has known a small quantity of albumen to be present for years without apparent harm. It seems doubtful whether there is more than fortuitous connection between glycosuria and albuminuria.

The next ground for prognosis is the effect of diet. If under restricted diet the amount of sugar rapidly diminishes, and the patient's discomforts diminish also, the prognosis is favourable, and particularly if he makes flesh. But if in spite of his faithfully following out advice as to diet, the output of sugar is still large, and the other symptoms are not materially *relieved*, the outlook is almost hopeless.

Treatment by diet.—Theory and experience agree in teaching that by diminishing the amount of glycose in the urine we diminish it in the blood, and that by diminishing it in the blood we relieve the symptoms of diabetes. They equally teach that we can in all cases limit, and in most prevent the excretion of sugar by removing carbohydrates from the food of diabetic patients. Thus, as Prout long ago laid down, "diet is the first and chief point to be attended to."

Dr John Rollo, at the end of the last century, appears to have first prescribed abstention from starch and sugar; he proposed to confine diabetic patients entirely to animal food. But although the inhabitants of arctic regions, the Caffres, and the trappers of North America live for months together without the chance of obtaining anything else, experience shows that when all kinds of food are within reach, there is very great difficulty in keeping patients exclusively to meat; and the more so since in diabetes the appetite is voracious, and the craving for forbidden food all the greater. We therefore try to include among permitted viands as many of vegetable origin as possible, and the result has been the construction of a tolerably copious diet-table.

Almost all *animal food*—flesh, fish, and fowl—may be eaten by persons suffering from diabetes, notwithstanding the small amount of glycogen it contains. But soups must not be thickened, or joints basted with flour. The liver of calves or pigs, and the edible molluscs—oysters, cockles, and mussels—all contain glycogen; but in the case of Joseph North (p. 455), to whom Dr Pavy twice gave four dozen oysters for two days running, they caused no decided increase in the amount of sugar excreted. *Milk* (which yields dextrose and galactose) is not so injurious as would be thought from the amount of lactose it contains.[*]

Cream and butter form important articles of diet in diabetes. Cod-liver oil is useful for loss of flesh. *Honey* (yielding dextrose and lævulose) is of course injurious. Glycerine is harmless, since its composition, $CH_5(OH)_3$ is different from that of any of the sugars. Indeed, when administered to animals it increases the storage of glycogen in the liver and diminishes the sugar in the blood. Another substitute for sugar is Saccharin (Benzoyl-sulphonic-imide). It sweetens far more powerfully than cane-sugar, and of course does not yield glycose; but it renders the saliva unpleasantly sweet.

The plan of treating diabetic patients by a diet of *skimmed milk*, six or eight pints daily, is not theoretically justified, for the lactose is taken and the cream left behind, and the practical results are unsatisfactory.[†] Much more likely to be useful is a preparation of milk which Dr A. E. Wright[‡] made by precipitating the casein with dilute acetic acid, and then redissolving the curd in an alkaline solution of the phosphates and other salts of milk. The result is a not unpalatable drink, which can be sweetened if desired with a little saccharin, and is entirely free from sugar. Practically, however, most diabetics do without much milk, and a small amount is very rarely found injurious.

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[*] Dr Pavy, indeed, found that in North's case the administration of three pints of milk daily not only caused a marked increase in the amount of sugar, but also brought back the uneasy sensations which the man had experienced when the disease had been uncontrolled by treatment. But Sir William Roberts mentions the case of a girl who (her diet being restricted in other respects) continued to gain strength and to improve in health when she was allowed to drink three pints of milk daily.

[†] Dr Greenhow recorded a case in which a patient took from four to six quarts of skimmed milk daily, with the effect of removing his symptoms and of freeing his urine from sugar, and two months afterwards he remained well. But Roberts refers to the cases of several persons treated with skimmed milk, and says that few of them could tolerate it for more than a few days, and then were rapidly reduced, while one case was brought to a fatal termination in three months.

[‡] In the Grocers' Lecture at the University of London, which was published in the '*British Medical Journal.*' *April 11th. 1891.*

Most *vegetables* are injurious. The worst are those richest in starch, as potatoes and artichokes; but carrots, parsnips, turnips, beans, Brussels sprouts, cauliflower, broccoli, and sea-kale also contain either starch or sugar, and can only be allowed with caution.

Cabbages, spinach, celery, lettuces, watercresses, or mustard and cress may all be eaten. By boiling in a large quantity of water, even the forbidden kinds of vegetables, if they contain sugar only, and not starch, may be rendered much less injurious. As Prout long ago remarked, there is a direct advantage in the use of such green vegetables as are harmless; for their indigestible part, cellulose or lignin, tends to correct the constipation which is often so troublesome in diabetes.

All kinds of sweet fruits are harmful, although currants and raspberries and other acid fruits are sometimes allowed. Nuts are harmless; but chestnuts abound in starch.

The greatest difficulty is for the diabetic patient to do without *bread*, and there have been many attempts to invent a substitute. Bouchardat in 1841 prescribed a kind of bread made from flour, after removal of all the starch by washing. But this gluten bread is by no means perfectly free from starch, for it at once turns blue if a little iodine be dropped on it; and patients complain that it is very unpleasant to chew. Gluten biscuits are less distasteful, and gluten may be used for the *boules de soupe* recommended by Dr Palmer, of Nayland. A second substitute for ordinary bread is a sort of cake from bran. Prout suggested this; and Dr Camplin, who himself suffered from diabetes, greatly improved the method by which it is prepared, so that bran cakes are still made according to his directions. A third substance which may be used by diabetic patients instead of bread is the almond cake which was introduced by Dr Pavy; its only fault is its high price. Cocoa-nut cakes are also manufactured with the same object, and bread made of the soya bean; but the latter, according to Keveli's analysis, contains 24 per cent. of carbohydrates, according to König's nearly 30. If inulin, the starch of dahlias and several other roots, could be produced in abundance, it might be used instead of flour, for it only yields lævulose on digestion instead of dextrose (Hale White).

Wheaten flour, as well as that of other kinds of corn, is of course injurious; and so are rice, arrowroot, sago, tapioca, macaroni, and vermicelli. In all but the most severe cases we may allow a little toast, not for the dextrine it contains, but because less is taken.

As to the *drink* of diabetic patients, restriction of the amount of water taken is followed by no advantage, and would add greatly to the patient's discomfort. Other allowable beverages are tea, coffee, cocoa made from nibs, claret, burgundy, chablis, hock; also brandy or other unsweetened spirits, with mineral waters. Chocolate, sweet ales, porter and stout, cider, all sweet and sparkling wines, are of course forbidden.

Dr Pavy says that the patient often at first complains that his food is not bulky enough to satisfy him; but after persevering for a few days he finds that his appetite becomes less. Johnson's dictum that it is easier to abstain than to be abstemious is very applicable to the diabetic.

Before a patient suffering from this disease adopts the restricted diet he should be weighed, the amount of urine passed each day and its specific gravity should be carefully registered, and the quantity of sugar secreted in the twenty-four hours should also be determined.

After such observation for two days (or in mild cases longer) we know how the patient's organism deals with an ordinary mixed diet, and have thus a standard by which we can judge of the effect of treatment. In all but intermittent or doubtful cases we should then begin the diabetic diet, at first excluding all saccharine and all starchy food, except milk and a little bread. If the sugar disappears and the symptoms improve, we may be satisfied with the result, and continue this modified restriction. If more than a trace of sugar is passed, and if the thirst and polyuria continue, we must then persuade the patient to give up bread for almond biscuits, and prescribe opium. When the sugar steadily diminishes or disappears, when the distressing symptoms are relieved and the patient begins to make weight, we may indulge him with bread and the less saccharine fruits or vegetables, and allow him to take as much milk as he pleases. In elderly persons free from serious discomfort, the less rigidly restricted diet should be allowed; but if a considerable and constant excretion of glycose goes on, we should advise stricter abstinence from starch and sugar.

In severe cases, and even in mild ones during the first week of treatment, it is necessary to analyse the urine daily, noting the amount, specific gravity, and the daily excretion of urea, as well as that of dextrose. Afterwards once a week, or in the slighter cases once a month, a sample of the urine should be examined, until on an ordinary mixed diet it has continued free from sugar for two or three months, and the other symptoms of the disease have disappeared.

One object of a daily analysis is to tell whether the patient is strictly following the prescribed rules. Those who suffer from diabetes often wilfully deceive their medical advisers, and are detected in having surreptitiously eaten bread, apples, or other forbidden food, by the discovery that the urine has on some one day been found to contain an increase of sugar.

When the restricted diet is conscientiously adhered to, we sometimes see the most rapid and striking benefit ensue.

A man aged thirty-nine was passing eight pints of urine daily, containing 5680 grains of sugar. Under a restricted diet the mean daily flow of urine went down within a week to 60 ounces; the sugar fell on the third day to 134 grains, at the end of a week to 48 grains, and after a fortnight only a trace of it could be detected. The patient lost all his symptoms, and gained flesh at the rate of three pounds a week. In about eight months all the sugar had disappeared, and nearly four years afterwards he was quite well.—C. H. F.

The present writer once met with a somewhat similar case. The patient, a stout florid man of forty-three, had begun rapidly to lose strength and weight, and to suffer from thirst and frequent micturition. The symptoms dated from early in December, 1890, and sugar was detected soon after. When first seen (January 1st, 1891) he had the raw tongue, constipation, and all the other discomforts of acute and somewhat advanced diabetes. The urine was of sp. gr. 1032, and contained a large amount of glycose. He was put on restricted diet with a grain of opium three times a day. He had greatly improved in a week, and the sugar had diminished to about a third of its previous amount. He persevered with the treatment, and three weeks after it was begun he felt perfectly well, and the urine was moderate in amount of sp. gr. 1021, and nearly free from sugar. On February 26th the urine was perfectly normal, and there was no return of symptoms two months later.

Unfortunately, in the majority of cases treatment is much less successful. The patient's symptoms may be greatly benefited, or altogether removed; the amount of urine which he passes may be much diminished, or reduced to the normal quantity; it may even cease to contain sugar, so that for the time all signs of the disease are absent; and yet, if he now attempts to return to his previous way of living, the urine again becomes saccharine, and one by one all the symptoms of diabetes reappear.

Even when the patient under strict diet loses his symptoms, and the quantity of urine which he passes falls to the normal amount, it by no means always ceases to contain sugar.*

Sometimes the most rigidly restricted diet entirely fails. There are cases, especially in young patients, in which all treatment appears to be useless. Other patients, again, are at first benefited by dieting; but after a time it ceases to be useful, and sometimes the disease appears to advance less instead of more rapidly when all restrictions are withdrawn.

Drugs.—When dieting fails, or as adjuvants to dietetic treatment, various drugs have been recommended. Pepsin and rennet have been tested by Parkes, Griesinger, and Roberts, are found useless or injurious. Ammonia, and other alkalies, strychnia, belladonna, physostigma, uranium, and lactic acid have all been tried and abandoned. Arsenic diminishes the amount of glycogen in an animal's liver, but has no obvious effect in diabetes.

The only drug that has stood the test of experience in diabetes is *opium*. It seems to have been first used by Rollo, its value was known to Prout, and Sir Thomas Watson spoke of it as a treasure. It is well known that patients suffering from this disease can take much larger doses of opium than healthy persons. Dr Pavy gives it in increasing doses, until he finds either that the disease yields or that the patient is unable to take it. We do not know how opium acts in diabetes. Roberts—who says that, without any restriction as to diet, daily doses of six to twenty grains always reduce the quantity of urine by about one half—believes that in patients who are dieted it is useless, except as a sedative. This, however, is inconsistent with Pavy's accurate observations; and, from a much smaller experience, the present writer would urge the great benefit of opium in full doses, particularly in severe cases, and when from poverty or carelessness the patient is unable or unwilling to submit to a serious restriction of diet. In such cases avoidance of sugar, potatoes, and pudding can generally be secured, and opium seems to make up for the bread that is taken.

In some cases opium disagrees, usually causing constipation, or, occasionally, diarrhœa. When this is the case we may employ *codeia*, as recommended by Pavy. Half a grain three times a day should be given at first, but the daily quantity may afterwards be gradually raised to as much as thirty grains. Such large doses, however, do no more good than moderate ones of three or four grains thrice a day. Often codeia may be given without any obvious effect beyond diminishing the sugar excreted; the tongue remains clean and the appetite good. Sometimes, however, codeia causes headache, and morphia may then be used; in fact, from the observations of Dr Mitchell Bruce and Prof. Fraser, it appears that in some cases it is more effectual than codeia ('Pract.,' Jan., 1887, and July, 1888; and 'Brit. Med. Journ.,' 1889, i, p. 118).

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* Whenever dieting does good, it appears always to bring the quantity of urine down to normal before seriously affecting the specific gravity or the amount of sugar. But whenever dieting readily lowers the specific gravity, it may be assumed that the quantity of urine passed by the patient is not excessive.—C. H. F.

General treatment.—The waters of Vichy have a great reputation in France, and those of Neuenahr in Germany; both of them contain sodium carbonate. The well-known Carlsbad waters are chiefly useful, if at all, from the presence of sodium sulphate (Cheltenham salts) when laxatives are needed. Some pathologists believe that the blood of diabetics is habitually less alkaline than normal, and it is said to have been occasionally found neutral. Careful experiments by Dr A. E. Wright showed in one case that the sugar steadily diminished under alkaline treatment.

Diabetic patients are generally advised to avoid exertion beyond their limited powers, unless they belong to the fat and plethoric group, in which all the symptoms are milder. Bouchardat, however, advised that active exercise should be taken, and Trousseau recommended it in the strongest terms. " Un diabétique, qui chaque jour fait à pied un exercise violent, peut, sans changer rien de son régime, retrouver temporairement la santé qu'il avait perdue," etc. ('Leçons,' tome ii, p. 764). It is curious to note that Celsus wrote, " At cum urina, super potionum modum etiam sine dolore profluens, maciem et periculum facit, si tenuis est [*i. e.* clear and pale] opus est exercitatione," etc. (lib. iv, cap. xx). Prolonged and active muscular exertion is now prescribed by several German physicians on theoretical grounds, as tending to oxidation of the glycose. In England we generally believe it to be injurious.

Complications.—The intolerable *thirst* of diabetes may be relieved by dilute phosphoric acid, or solution of bitartrate of potash. Constipation should be treated with Ol. Ricini or Conf. Sennæ or extract of aloes, or with saline purgatives, of which sulphate of magnesia well diluted is probably the best.

If there is *phthisis* present, one can only give opium more freely than in other cases of pulmonary disease, and urge as much cod-liver oil, butter, and cream as the patient is able to digest.

When *diabetic coma* comes on, intra-venous injection of saline solution in order to dilute the thickened blood has been repeatedly tried, unfortunately with even less frequent or more temporary success than in the collapse of cholera (cf. *supra*, p. 290).

In the 'Guy's Hospital Reports' for 1873-4 (vol. xix, p. 173) Dr Fagge recorded a case of injection in which the pulse was scarcely perceptible, and the body and limbs were cold. Twenty-six ounces of a solution of phosphate of soda and chloride of sodium, of sp. gr. 1020, were injected into the right cephalic vein, with the effect of restoring the patient to consciousness for a time. He sat up, answered questions, took nourishment well, and even asked for it; his pulse was 80. Thirty-two hours later, however, he again became drowsy and died. This patient's condition, before the solution was injected into his veins, was strikingly like that of a man in the collapse of cholera: only a few drops of thick dark blood escaped from the wound in the arm.

A few months afterwards Dr Frederick Taylor had a similar case, in which he employed the same treatment, but with scarcely any good result (*ibid.*, vol. xix, p. 521, and xxv, p. 169). Dr Dickinson has since put on record a remarkable case in which very free intra-venous injection of saline solution had a decided though only temporary effect ('Clin. Trans.,' 1890,

p. 130). In a case reported in the 'Lancet' for 1898 (vol. i, p. 401), Dr Thomas Oliver, of Newcastle, had as striking success, and the coma had not returned after four weeks.

Probably the best chance of averting threatening diabetic coma is active purging, but no method can be depended on.

GENERAL DISEASES AFFECTING THE JOINTS

GOUT

'Ω στυγνὸν οὔνομ', ὦ θεοῖς στυγούμενον,
Ποδάγρα, πολυστένακτε, Κωκυτοῦ τέκνον.

Πόδα, γόνυ, κοτύλην, ἀστραγάλους, ἰσχία, μηρούς
Χεῖρας, ὠμοπλάτας, βραχίονας, κορώνα, καρπούς
'Εσθίει, νέμεται, φλέγει, κρατεῖ, πυροῖ, μαλάσσει.

LUCIAN, *Podagrotragædia.**

History—Onset and symptoms of a first attack—Subsequent course—The affected joints—lithate of soda—tophi—Pathology of gout—Ætiology—hereditary gout—age and sex—diet—climate—saturnine gout—Diagnosis—Prognosis—Gouty nephritis—other complications—Treatment by drugs, diet, and baths.

Synonyms.—Gr. Πυδάγρα. The terms χειράγρα, ὠμάγρα, and similar compounds were also used.—*Lat.* Podagra: in Mediæval Latin *Gutta*, a drop, *i. e.* of distillation of morbid humours into a joint; whence *Gout*, *Goutte*, and *Gicht*. "Arthritis," ἀρθρῖτις, sc. νόσος, is sometimes used as a synonym by classical and by modern writers.

Definition.—A disease characterised by deposition of insoluble urates in the cartilage of the joints and in the subcutaneous tissues.

History.—This remarkable disease was well known to the ancients. The Hippocratic writings show that it was common in Greece, and it is frequently alluded to by Cicero, Horace, Juvenal, and Martial. Celsus, Galen, and Aretæus describe the disease very much as we see it now. Alexander of Tralles, in the sixth century, recommends hermodactyl, and this drug was praised by the Byzantine and Arabian physicians; it was probably the corm of a species of colchicum. Lucian wrote a burlesque tragedy, of which four lines are quoted at the head of this chapter, describing the power of the goddess *Podagra*.

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* *Oh!* hateful name, abhorred by the gods, most lamentable, hell-born gout!
Foot, knee, hip, ankle, haunch, and thigh,
Hands, *shoulders, arms, elbows, and wrists.*
and scorches and

In modern times the medical literature of gout dates from Sydenha (1683), who was very competent to write about this disease, having himsel suffered from it for thirty-four years. His masterly description was copie by Cullen, and has formed the basis of almost all that has since bee written upon the subject.

At the beginning of the last century Sir Richard Blackmore, the court physician, whose poems live in Dryden's satire, wrote a worthless discourse on the Gout (1726), and Dr George Cheyne an essay on the same subject (1722), better written, but with no other merit. Heberden's chapter on the subject is one of his best (1782). Early in the present century Sir Charles Scudamore published a treatise on Gout and Gravel, which went rapidly through many editions (1817 to 1823); and here, for the first time, we find the modern method of collection of facts, anatomical observations, and statistics. A great advance was made by the publication of Sir Alfred Garrod's well-known and original researches (1859), by those of the late Sir William Roberts (1882), and of Dr Luff (1898).

Abroad, gout has always been comparatively rare, but Trousseau and Charcot in France, and recently Professor Ebstein in Germany, have made valuable contributions, both clinical and pathological, to its study.

The fit of gout.—Probably most cases of gout begin insidiously, but the first onset of the disease is sometimes dramatically sudden and severe. The following is taken from Sydenham's clinical description of such a case, drawn, no doubt, from his personal experience:*

"The patient goes to bed and to sleep in apparent health, but is awakened about two o'clock by a pain in one of his feet, generally in the ball of the great toe, but sometimes in the heel, ankle, or leg. The parts feel as if tepid water were poured over them. Then follow chills and rigors and a little fever. The pain, which was at first moderate, becomes more intense. It is a grinding, wrenching pain, like that of dislocation of a joint; or as if a dog were gnawing it; or, again, like a violent compression of the foot. The patient keeps changing the position of his foot, in the vain hope of finding a place in which to lie in comfort. He cannot bear the bedclothes to touch it, and the least vibration of the floor causes him extreme distress, so that those about him have to tread the room with the lightest possible step." For, as Sydenham elsewhere puts it, "a fit of gout is a fit of bad temper."

"Towards the morning of the second night the patient has a sudden and slight respite, which he imputes to having at length found a comfortable position. He perspires gently and falls asleep. He wakes freer from pain, and then finds that the part is swollen. Till then the only visible swelling had been that of the veins round the painful joints. For the next two or three days the pain returns towards evening, and again abates in the morning. A few days after, however, the other foot begins to swell, and the whole tragedy," as Sydenham calls it, "is acted over again."

The affected joints, besides being exquisitely painful, are of a deep red colour, swollen, tense, and shining. As soon as the patient can bear the pressure of the finger he finds that the skin pits, from inflammatory œdema. Then follow desquamation and itching.

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* The passage occurs in the 'Tractatus de Podagra,' § 5, beginning "*Sanus lecto somnoque committitur.*" Another abridged account appears in the 61st chapter of the '*Processus* integri de morbis curandis.'

The amount of fever depends on the local inflammation. Even in the most severe attacks, it is not so high as in Rheumatism.

A first attack is made up of a series of minor fits, which gradually ecome milder, and altogether last about a fortnight. Later attacks are ss severe and more continuous.

Before a fit of the gout the patient often appears to be in his usual ealth. Sydenham, however, observed that indigestion and flatulence ometimes precede the attack; and Sir Alfred Garrod says that such premonitory symptoms may be very distressing. Trousseau remarks that he patient is often "bilious," with a capricious appetite, preferring acids nd highly seasoned dishes.

After an attack of gout a man often feels much better than before. He is more active, and free from the uncomfortable feelings that may have roubled him. Sooner or later he experiences a second seizure; sometimes not for two or three years after the first; but often the interval is not more than a twelvemonth, and it may be less. The second attack is in its turn succeeded by others, and always at shorter intervals; until at last the disease becomes chronic, and the patient is scarcely ever free from it.

Probably in consequence of Sydenham's graphic and accurate description of a first attack of acute gout, it has been too often assumed that such is its usual or even constant mode of onset. But in many cases, perhaps in the majority, the first attack is far less acute, severe, and dramatic: the tragedy is more slowly played out. And in not a few patients gout begins in no acute or sudden form, but stealthily and almost imperceptibly. A man about fifty complains of his boots being tight, or sprains his ankle while shooting, or feels a twinge in one knee as if it were strained. There is no pain at night and no redness or swelling to be seen, but a moderate walk is more and more apt to be followed by a "sprain," and at last the great toe or instep or ankle is found to be decidedly swollen, tender, and slightly reddened, so that the suspicion, which the patient had long silenced, becomes a certainty,—he has the gout.

Locality.—The ball of the great toe is the joint far most frequently attacked by gout. Sir Charles Scudamore found that this joint was affected on one side or the other in 373 out of 512 first seizures. Perhaps its liability to strains and blows and pressure may explain this predilection; for if another joint is the first to suffer, it is often found that it has previously been injured. Garrod has seen the knee attacked first when the patient has previously hurt it by a fall. Next to the great toe, the joint most obnoxious to a first attack of gout is the metacarpo-phalangeal articulation of the index finger; not that of the thumb. Both the great toe and the root of the index are more exposed to pressure and strain than the other joints of the foot or hand.

Taking first and later attacks together, we see gouty arthritis most often in the first metatarso-phalangeal joint, next in the ankle and heel, next in the fingers and wrist, and next in the knees. All these situations are common, and correspond to the old recognised names of podagra, chiragra, and gonagra. Less frequently is the elbow attacked, and very rarely the shoulder-joint, the hip, the clavicular, mandibular, or vertebral articulations.

Whatever may be the part first affected, other joints afterwards suffer, and even in the first seizure *two or three* may be attacked at once.

acute patients after death. Dr

Norman Moore ('St Barth. Rep.,' 1887) found the same order in the presence of biurate of sodium in the joints, the least often affected being the elbows, shoulders, hips, jaw, and clavicle.

Anatomy.—The result of repeated attacks of gout is to cause more or less deformity of the affected joints. The fingers, which suffer more than the toes, become bent in all directions—sometimes inwards, sometimes outwards. A common condition is for the metacarpo-phalangeal and the second phalangeal joints of a finger to be stiffly flexed, while between them the first phalangeal joint is over-extended, and its knuckle represented by a deep hollow.

All the phalangeal joints are in some cases uniformly enlarged. Sydenham compares their appearance to that of a bunch of parsnips. But as a rule the deformities caused by true gout are unsymmetrical.

Lithate of soda.—In long-standing cases of gout there are found around the joints masses of a white material, sometimes as soft as putty or mortar, sometimes as hard and dry as chalk. These masses are called *tophi*[*] or "chalk-stones." They do not, however, consist of carbonate of lime, but mainly of urate (lithate) of soda, as was first proved by Wollaston in 1797. This salt is also found deposited in the articular cartilages of the affected joints. It looks as if it were upon the surface of the cartilages, covering them more or less completely, just as if it had been laid on by a brush. But on making a section of the cartilage one finds that the deposit is really embedded in its substance. Examined by the microscope, it proves to consist entirely of crystals. The bundles of slender needles (according to Cornil and Ranvier) commonly occupy the centre of the cartilage cells, but the writer has also seen them in the intercellular matrix. It is in the superficial part of the cartilage that the crystals are most densely crowded, and often make it opaque to transmitted light. Towards the articular surface of the bone they are more thinly scattered; here they often traverse the whole thickness of a cartilage cell, which is, as it were, impaled on them.

The synovial membrane may contain similar deposits of the urate; but they are more often found in the surrounding ligaments and tendons. Sir Alfred Garrod says that he has never been able to find lithate of soda deposited in the bones.[†]

Bony ankylosis is unknown, and it is very rare to find the cartilage absent in a gouty joint.

Urate of soda is deposited in many cases at a distance from the joints. In 1886 and 1887 a patient was in Philip Ward who had tophi of lithate of soda in the skin of his legs and arms, as well as about the joints. These deposits, however, are rare, except near the joints, or in the external ear, generally in the helix. Garrod remarks that these deposits in the pinna of the ear are at first fluid, the skin over them forming a vesicle of a milk-white appearance. He says that some months elapse before they become the white, or yellowish, hard, bead-like little nodules which are commonly seen.

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[*] *Tophus* or *tofus*, the Greek τόφος, seems to have been applied to rough crumbling rock, the disintegrated volcanic *tufa*. Virgil associates it with chalk:

Et tophus scaber, et nigris exesa chelydris
Creta. 'Georg.,' ii, 214.

[†] Cornil and Ranvier teach that it is not uncommon in the cancellous tissue. They mention one case in which they observed it, and in which the bones forming the metatarso-phalangeal joint of the great toe had entirely lost their cartilages and were ankylosed together, and say that absorption of the articular cartilages is a common remote effect of urate of soda in a joint. This, however, is not in accordance with our experience in *England*.

The bursæ are very liable to receive deposits of urate of soda, particularly those over the olecranon, which may become enlarged until they are almost as big as oranges.

A man used to attend the present writer's out-patient room with numerous tophi in the skin of the scrotum; a second much earlier case is figured in a drawing made for the Guy's Museum; and a third is cited by Duckworth from Mr Butlin's practice at St Bartholomew's Hospital. They have also been found, though very rarely, in the dura mater, the sclerotic, and the perineurium of nerve-trunks. In the kidneys they are so frequently present, along with granular degeneration, that Todd spoke of this form of disease as the "gouty kidney."

When the tophi become very large, the skin gradually wears through. Nodules in the ears may thus be cast off, and Watson tells the story of a gentleman who had tophi exposed on his hands, and who used, when playing at cards, to score the game upon the table with his gouty knuckles.

Although a joint affected with acute gout often looks so inflamed that suppuration seems imminent, yet as a matter of fact this never happens.

It has even been questioned whether the arthritis of gout is inflammation at all. But apart from the pain, heat, redness, and swelling, with pyrexia, there is evidence of exudation also. So that the classical phrase may still be applied; but the inflammation is gouty not suppurative, irritative not pyogenic (cf. *supra*, pp. 56, 60). Abscesses around the extra-articular tophi in chronic gout are not uncommon, and they discharge lithate of soda, mixed with pus. Garrod speaks of as many as five or six abscesses of this kind being open at one time on each hand, and others on the feet, and he remarks that they give rise to little pain or constitutional disturbance.

It was once supposed that deposits of urate of soda were to be found only in advanced cases of gout, and Scudamore asserted that they did not occur in one gouty case in ten. This is true only of masses of the urate deposited outside the joints, and large enough to be seen and felt. Within every gouty joint urate of soda is present in greater or less quantity. Garrod mentions two patients, of whom one had only a single attack of gout thirteen years before his death, while the other had had two attacks within the last two years of his life. In each case a small quantity of urate of soda was found, as a white deposit, in the cartilage of the joints which had undergone gouty inflammation.

Pathology.—In considering the theory of gout, the primary fact to bear in mind is that the blood in this disease contains an excess of uric acid, in the form of urates of soda, potass, and lime.

Garrod has not only shown that excess of uric acid can be detected in the blood in gout by an elaborate chemical analysis, but also that for clinical purposes its presence may be ascertained. About two drachms of the serum of blood are put into a flat glass dish, somewhat larger than a watch-glass, and acetic acid is added so as to give a slight acid reaction. A fibre from a piece of linen cloth is then placed in the fluid, and the dish is set aside until its contents have acquired a gelatinous consistence by evaporation. If the blood contains uric acid in excessive quantity it is deposited upon the fibre, and this becomes studded with crystals, the characteristic appearance of which can readily be identified with a pocket lens. The serum from a blister will also yield crystals of uric acid when examined in this way, but only when the blister is placed at a distance from a joint affected at the time with gouty inflammation; and hence Garrod infers that gouty inflammation causes a local destruction of uric acid. This agrees with the fact that an attack of gout has the effect of clearing the blood from urates. In patients who were recovering from the fit Garrod found a marked decrease in its quantity, and in the intervals between early attacks of gout he found no appreciable amount of urates in the blood, restlessness, depression, and dyspepsia and flatulence, headache and neuralgia, which are supposed to depend on excess of urates circulating in the blood-serum. This result of gouty inflammation in freeing the blood from accumulated lithic acid accounts for a fact which appears to be well established, that an attack of acute gout often leads to the rapid disappearance of dyspepsia, flatulence, irritability of body and mind, and other symptoms which used to be called "irregular" gout.

Garrod believes that lithate of soda is invariably present in a gouty joint; and he supposes that the salt is deposited not as the result, but the cause, of the inflammation.

Gouty concretions form in the pinna of the ear, without causing much local inflammation. In some cases the patient experiences sensations of heat and pricking, and the part is tender; but more often he is quite unconscious of their presence. So also in the joints we may find the articular cartilages of the great toe encrusted with urate of soda in many cases in which no mention of gout had been made during life. But so we may find rheumatic endocarditis without history of rheumatism, obsolete tubercle without history of phthisis, and gummata without history of syphilis.

Garrod states that among a large number of autopsies on persons who were not known to have gout, there were only two in which even a trace of urate of soda was found in the great toe-joints; and he supposes that the deposition of the salt in a joint is always followed by a fit of the gout.

There are two conditions which may be supposed to cause a rapid increase in the lithate of soda in the blood, and so lead to its deposition in the joints and provoke an attack of the gout; namely, gastric disturbance, chiefly from errors of diet, and failure of the kidneys to excrete lithic acid.

The former of these conditions is well known to be a frequent exciting cause of a gouty seizure. Scudamore mentions the case of a gentleman, free from hereditary tendency, and with no reason to suspect that he would be attacked, who was seized with gout for the first time after three or four days of "excessive conviviality." Another striking case is that of a gentleman who had never had gout in the summer, and who, persuaded of his security, drank six or seven glasses of champagne; in twelve hours he was laid up with a fit. In three instances patients had sat down to dinner with scarcely the sensation of gout, but when rising to leave the table found themselves completely disabled. Similar cases are familiar in modern practice in London.

It was Garrod who drew attention to the fact that in many cases the condition of the kidneys is the exciting cause of a fit of gout. He found that in severe cases of acute gout the amount of uric acid contained in the urine was on an average less than four grains daily—the normal amount being eight or ten grains. It is true that in such cases the urine as it cools often deposits lithates, which are of a bright pink or red colour, and are supposed to indicate an excessive excretion of uric acid. But this conclusion is fallacious: for, first, the quantity of urine passed in the twenty-four hours is diminished; and secondly, its acidity is much increased, so that the whole of the lithates are deposited. As gout becomes more and more chronic, uric acid is excreted by the kidneys in diminished amount even in the intervals between the attacks, and in advanced stages of the disease it may be entirely absent from the urine. But the *blood* in chronic gout always contains excess of uric acid in the form of urate of soda.

Sir William Roberts ascertained that the condition in which lithic acid normally circulates in the blood is that of sodic quadrurate ($NaH,H_2\bar{U}_2$, as in the urine); but in the gouty state, whether from renal inadequacy or some other reason, the quadrurate lingers too long in the blood and so becomes gradually transformed into sodic biurate ($NaH,\bar{U}$), a far less soluble salt.*

This process can be imitated out of the body by mixing a solution of the biurate of soda with one of bicarbonate of the same base.

When the accumulation of the biurate has reached a certain point of saturation, it is suddenly precipitated as a crystalline deposit in the joints; its locality being determined by the fact that the tissues are less alkaline than the blood, the cartilages nearly neutral, and that blood, synovia, and cartilage are all less alkaline in gout than in health.

Moreover the crystals are most readily formed in those parts which are not vascular, or in which the circulation is sluggish. This applies both to the pinna of the ear and to the articular cartilages, while in the knee-joints, the parts in contact with the vascular fringes remain free. Sir William Roberts by careful experiment showed that soluble urates are apt to break up into insoluble salts, and contrasts the solvent effect of rapidly circulating blood with that of stagnant synovia.

Ebstein's theory of local necrosis as the result of poisoning by soluble urates and the occasion of the deposit of the crystals has been, it appears to the writer, refuted by the arguments of Roberts, of Duckworth, and of Luff ('Goulstonian Lectures,' 1897, and p. 11 of the monograph, 1898).

Accepting the strictly chemical or (it used to be called) humoral origin of gout in excess of uric acid in the blood, we naturally suppose that this depends on some error in the metabolism of the proteid elements of the food. To explain this faulty metabolism the ready influence of the nervous system has been invoked, or direct trophic nerves which determine the arthritis have been assumed, and even articular centres in the cerebrum.

Irregular gout.—For some reason it has become common to ascribe bronchitis, dyspepsia, gastralgia, iritis, cystitis and urethritis, phlebitis, eczema, and even psoriasis to a gouty diathesis.† But the evidence is very slight, and the "gout" to which such evidence applies is the distillation of morbid humours which belong to a bygone pathology, not deposit of urate of soda in the tissues.

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* The neutral urate ($N_2\bar{U}$) does not exist in the body at all, either in health or in disease, either in blood, urine, or joints.

† Crabbe satirises this weakness of our art as follows:

> "One to the gout contracts all human pain;
> He views it raging in the frantick brain,
> Finds it in fevers all his efforts mar,
> And sees it lurking in the cold catarrh."

Whether excess of urates in the blood is ever the cause of acid dyspepsia, neuralgia, headache, lumbago, sciatica, ill-temper, and sudden death, we do not know. It certainly must not be assumed without evidence. Long before Wollaston discovered uric acid it was believed that these and many other symptoms were caused by "gout," wandering about the body, until it ended its migration and settled down in one of the joints to a regular and orthodox attack. Patients were therefore described as the subjects of an "irregular" form of gout, which was further stigmatised as atonic, masked, latent, lurking, suppressed, bastard, and sneaking. This goutiness was not a clinical condition like Murchison's lithæmia, but a disease, a "morbid entity." But "explanation" is always easier than investigation; and explanation is what many of our patients require even more than cure; and to be told that his symptoms are "gouty" is always acceptable to him who looks on the disease as a patent of gentility. If we put aside the baseless theories of an arthritic diathesis, and the medical casuistry supplied by advertising druggists, we find assumption and little evidence of gout in the modern sense being the cause of the irregular symptoms ascribed to it.

The diseases which do frequently accompany gout are plumbism, chronic interstitial nephritis, atheroma, diabetes, dyspepsia, and flatulence. No one pretends that gout causes dropped wrist, but lead causes granular kidneys, and inadequate renal action helps to bring on gout. Atheroma belongs to the later period of life, as does gout, and the chances are that a man of sixty who has an attack of gout has also an atheromatous aorta; but the calcareous plates there found are not made of urate of soda, but phosphate of calcium.

The coincidence of gout and diabetes is probably more than accidental (cf. *supra*, p. 457); but no one supposes that diabetes causes gout. Probably gout does not cause diabetes either, but both diseases are in certain cases the results of common causes.

Over-indulgence in eating and drinking causes dyspepsia, and also causes gout; and when a man frequently suffers from a painful complaint his temper is apt to suffer.

The only disease of the skin which, in the writer's experience, has any more than accidental relation to gout is a certain form of chronic, usually dry and very irritable, eczema.

There is no reason to believe that gout ever "flies to the stomach," but over-indulgence at the table may produce acute gastralgia as well as inflammation of the great toe. Elderly people are liable to bronchitis, from which they are not protected by gout; and while drink may lead to chronic Bright's disease, cirrhotic kidneys favour an attack of gout.

It is doubtless possible that the deposition of lithate of soda causes inflammation in other tissues beside the joints; as neuritis of the sciatic nerve, or inflammation of a varicose vein, of the sclerotic, or the iris. The proof, however, must be given in each case.

Ætiology.—Accepting the deposit of crystalline biurates in cartilage and other tissues from uratosis, or excess of urates in the blood, which is present in leuchæmia and other states unallied with gout—the inquiry still remains under what general conditions this uratosis is observed.

Here we leave theory, and appeal to experience concerning the incidence of true gout (not of hypothetical "goutiness") on a large scale.

In the first place, gout is without question an *hereditary* disease.* Garrod found that more than half of all his gouty patients could prove an inherited disposition to this disease; and he says that among persons belonging to the upper classes the proportion is considerably greater. Roberts estimates it as two thirds. Even among hospital patients the present writer found 21 out of 61 cases with a clear hereditary history.

Sir Charles Scudamore found, among 522 patients suffering from gout, 190 in whom no hereditary taint could be traced. Of the remaining 332 patients, 181 inherited it from the father, 59 from the mother, and 24 from both parents, while the other 68 had grandparents (44 cases), or uncles (21) or aunts (3), who had suffered from the same disease.

The popular idea that the disease often misses a generation only applies to the case of transmission from a grandfather through his daughter to his grandson. Hutchinson ('Med. Times and Gaz.,' 1876) believes that the younger children in a family are more likely than the elder to suffer, and to suffer severely, and Roberts agreed in this statement so far as it refers to acquired gout.

The *age* at which a first attack of gout is most apt to occur is between thirty and fifty-five. In persons less than twenty years old the disease is very rare. In early cases there is always a strong hereditary predisposition. Garrod has seen it in the great toe of youths of sixteen. Scudamore mentions two cases in boys of eight and twelve, but neither was seen by him, and they were possibly not genuine: his own youngest patient was fifteen. Sydenham said, "I have not hitherto found children or very young persons affected with the true gout." The aphorism of Hippocrates is still true: *παῖς οὐ ποδαγριᾷ πρὸ τοῦ ἀφροδισιασμοῦ* (vi, 30).

Beyond the age of sixty-five a first seizure seldom occurs: Scudamore had never seen a case, but Garrod records one at eighty and another in a woman nearly ninety. Among sixty-one cases collected by the writer, in one the first attack occurred at seventy, and one of the worst cases of (hereditary) gout was in an abstemious young woman of twenty-one, who was under his care when her father, who was an intemperate man, was also a patient with gout.

The disease is much more common *in men* than in women, perhaps as much as eight or nine times. This doubtless depends mainly upon the fact that their habits more frequently tend to develop the disease. It was said that the intemperance of the Roman ladies under the Empire frequently led to gout. Even where favoured by inheritance it seldom attacks women until after the cessation of the menstrual function.† Among seven female patients with gout, in three it was hereditary and in three it was caused by drink.

High living is generally and probably rightly believed to be a cause of gout. Whether eating much meat is worse than other kinds of indulgence at the table is not so certain.

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* The hereditary character of gout seems to have been unknown to the classical writers, but it is said to be mentioned by Aëtius in the sixth century. It is clearly stated in Pirkheimer's 'Apologia seu Podagræ laus,' published at Nürnberg in 1522, and afterwards translated into German (1577) and into English ('Praise of the Gout,' translated by W. Est, Lond., 1617). Heredity was made part of the definition of true gout by Cullen.

† Here again the aphorism of Hippocrates holds: *Γυνὴ οὐ ποδαγριᾷ ἢν μὴ τὰ καταμήνια αὐτῇ ἐκλίπῃ* (vi, 29).

As regards the power of *alcohol* to produce gout, there is no doubt whatever; but all liquor does not act equally to this effect. The fact that malt liquors are more apt than ardent spirits to produce gout is well shown by comparing the working men of London with those of Glasgow; the former drink beer and porter, as well as spirits, and are very liable to the disease; the latter drink little but whisky, and much of this, and they scarcely ever have gout. The rarity of gout in many of the cities on the Continent, where spirits form the chief intoxicating beverage, is another proof that they have little tendency to produce it. The beer of Munich and Strassburg has not the same effect as London porter. Perhaps the quantity drunk, and its powerful diuretic effect, may account for this as much as its weaker alcoholic strength.

As to different kinds of wine, gouty patients well know the danger of indulgence in port, champagne, Burgundy, or Madeira. Gout is more common at Dijon than on the Rhine. Garrod gives a caution against sherry also—however dry and pure,—and thinks that cider, when sweet and partially fermented, is apt to cause gout, although rough cider is comparatively harmless. Why one kind of alcoholic drink is more "gouty" than another is hard to say. Their comparative effects in causing dyspepsia, and on the other hand in leading to free diuresis, are probably important.

It is generally believed that sedentary habits favour, and active exercise keeps off, gout; but this is contrary to its rarity in women, and many men who suffer from it are of vigorous habits and constantly in the open air. Gout, as Roberts says, is rare among drunkards.

Sydenham comforted himself under the pains of gout by remembering that wise men, not fools, philosophers, monarchs, generals, and admirals are its chosen victims.[*] In England it has been called the statesman's disease, and abroad Charles V and Richelieu were martyrs to gout. Both these men were abstemious, although the Emperor was a great eater; Walpole, Chatham, and Pitt were wine-drinkers; but Milton, the sober son of a sober father, would seem to have earned the gout by intellect alone, for his "hands and fingers were disabled by chalk-stones." Even among hospital patients those afflicted with gout are, as a rule, skilled artisans, bodily and mentally above the majority of those who throng the out-patient rooms of London.

Sydenham observed, in his description of gout, that the first attack generally occurs in the *winter*, towards the end of January or the beginning of February. Why this should be the case is not very clear. In many patients the disease returns for two or three years in the spring only; after a time a second attack occurs in the autumn, and at length the seizures occur quite independently of the season.

The influence of cold in the development of gout is shown by the effects of change of *climate*. Garrod says that a gouty man may often escape his accustomed winter attacks by going to Malta or Egypt. In hot countries this disease appears to be very rare, or unknown. Even in the south of Europe it is much less frequent than in England; but we must remember that the habits of the people of different countries are very different.

Certainly the disease seems to have been more common in ancient Greece and Rome than in modern Italy, Spain, and France. Pliny says that gout was not so common before his time in Italy, and to prove that it was no native of Italy argues that it had no Latin name (lib. vi, cap. x). To the same effect Sir Wm. Temple writes, "Among all the diseases to which the intemperance of this age disposes it, at least in these northern climates, I have observed none increase so quickly within the compass of my memory and recollection as the gout." Among sixty-four autopsies at Vienna, the present writer, when studying there under Rokitansky in 1865, only once saw a case of gout, and the deposit in the joints was not recognised by anyone but Rokitansky himself. It is believed to be more common in Paris and Berlin than it used to be, and also in the United States. Gout is rare in Russia and Scandinavia, notwithstanding cold and spirit drinking; and is more frequent in beer-drinking Holland and Flanders. It is undoubtedly less common in Scotland and Ireland than in England. Pagenstecher speaks of gout with respectful envy as the heritage of "die reichen Söhne des gesegneten Albions."

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[*] 'Tractatus de Podagra,' § 15: "Ita vixerunt atque ita tandem mortem obierunt magni reges, dynastæ, exercituum classiumque duces, philosophi aliique his similes haud pauci. Verbo dicam articularis hicce morbus (quod vix de quovis alis affirmaveris) divites plures interemit quam pauperes, plures sapientes quam fatuos."

A remarkable cause of gout is chronic *poisoning by lead*. Garrod found that about 30 per cent. of the gouty patients in his hospital practice had been exposed to the influence of lead.* Some of them were painters and plumbers, others workers in lead mills, and others "composite-doll" makers. A careful inquiry into the habits of the men failed to show that they had been less temperate in their habits than others of the same class. Duckworth, among out-patients, found 25 cases of saturnine gout among 136 patients.

That this is a true cause is sufficiently established by everyday hospital experience in London. The way in which lead produces gout is more doubtful. Garrod believes that persons who are already gouty are more susceptible than others to be affected by lead. He says that in several instances he has found those patients to be of gouty habit, or to have already had severe attacks of gout, in whom the medicinal administration of the preparations of lead has produced colic or a blue line upon the gums with unusual rapidity. Possibly the excretion of lead in the urine leads to irritation of the kidneys, and thus to chronic Bright's disease and retention of uric acid. Saturnine gout is rare in the north of England, and almost unknown abroad (Dr Thomas Oliver, Goulstonian Lectures, 'Brit. Med. Journ.,' March, 1891). Dr Lorimer, of Buxton, has published an account of 107 cases, most of them occurring in visitors (*ibid.*, July 24th, 1886). He finds that it usually befalls patients earlier than hereditary or than alcoholic gout, that the fits are less severe but more lingering, and that albuminuria is almost constantly present.

An occasional exciting cause of gout is *mental fatigue or anxiety*. Sir Charles Scudamore mentions the case of two female patients, in each of whom a severe first attack of gout was brought on by sitting up for several nights in succession, nursing a sick relation. Severe intellectual labour, want of sleep, sudden passion, and sexual excess may each be the occasion of a fit of the gout. The ancients laid stress on the last exciting cause:

> *"Λυσιμελοῦς Βάκχου καὶ λυσιμελοῦς Ἀφροδίτης*
> *Γεννᾶται θυγάτηρ, λυσιμελής ποδάγρα."*

Diagnosis.—This is seldom difficult, if we keep to our definition of gout as the effect of deposit of crystalline biurate of sodium in the joints and other organs; if we extend the term to cover any symptom for which a patient can be induced to visit Vichy or Carlsbad, the diagnosis is impossible, since it can never be verified.

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* *Arthralgie saturnine* described by Tanguerel des Planches in his treatise on Plumbism does not refer to gout, but to neuralgia. See Wilks in the 'Guy's Hosp. Rep.' for 1870, vol. xv, p. 40.

(*a*) When *arthritis* is actually present, and we can exclude traumatic disease, the diagnosis is limited to gout, rheumatism, gonorrhœal synovitis, pyæmia, tuberculous and syphilitic disease, and osteo-arthritis.

A difficulty has been supposed, from the existence of a form of arthritis intermediate between gout and rheumatism. But there is no evidence of such a *tertium quid*. Either the affected joint contains urate of soda or it does not: in the former case the disease is gout, in the latter it is not. Doubts never arise in the deadhouse as to the gouty nature of disease in a joint. As for the simultaneous presence of gout and rheumatism in the same patient, its theoretical possibility must be admitted, but practically it does not occur oftener than coincidence of acute rheumatism and enteric fever, or of scabies and scarlatina, or of syphilis and psoriasis. The term "rheumatic gout" is either a bad name for osteo-arthritis, or is a mere excuse for shirking a difficult diagnosis.

The following characters are those most useful in deciding a case to be gout:—That the small joints are affected, and particularly the great toe-joint; that the attack began suddenly in the night, especially if it be a first attack; that the skin over the affected joint is tense, shining, and red, after a day or two œdematous, and finally desquamating; that the febrile disturbance is moderate, and in proportion to the local inflammation. The sex, age, and family history of the patient have also to be taken into account; and the state of the heart and kidneys must be investigated. All those parts which are apt to be the seat of tophi should be carefully examined; for if a deposit of urate of soda can be found, it settles the question. But care must be taken not to mistake other kinds of enlargement of the finger-joints for those caused by gout, and it is needful to distinguish from tophi in the auricle comedones of the sebaceous glands, and that occasional nodule in the edge of the helix which Darwin described.

In exceptional cases other forms of multiple arthritis beside rheumatism may be mistaken for gout. Garrod mentions a case in which the great toe was swollen, tense, red, and hot; in which, in fact, the joint looked exactly as though it were affected with severe gouty inflammation; but the disease turned out to be pyæmia. The diagnosis from gonorrhœal synovitis and osteo-arthritis will be considered in the following chapters.

(*b*) In cases where *no articular inflammation* is present the recognition of gout is a matter of far greater difficulty. If the patient has suffered from unmistakable gouty arthritis before, or if his father or elder brothers have suffered severely or frequently from genuine articular gout, we may then reasonably ascribe wandering pains, headache, lumbago, or sciatica to this cause, and may anticipate an attack of arthritis.* But in the absence of such evidence the assertion that dyspepsia, bronchitis, cutaneous diseases, cardiac palpitation, asthma, gonorrhœa, iritis, or any other disease is of a gouty origin, cannot be called anything but arbitrary, and it has the fatal objection to all such easy "explanations" that they can be neither proved nor disproved; they only check investigation. An "arthritic diathesis" is often made to play the same part as "a sluggish liver," "nervous debility," a remote "syphilitic taint," a "strumous cachexia," or a "scorbutic habit of body"—the discredited commonplaces of a bygone pathology, which, when abandoned by the physician, still linger among the laity. As the existence of an arthritic diathesis cannot be proved, so it cannot be refuted; for if the patient dies and no lithate of soda is found in his joints, "one must not be bound by too narrow and mechanical a theory of the disease;" if an inflamed joint can be clearly traced to a traumatic origin, it is argued that the gout was only thus determined to a particular spot; if the patient has lived freely, the diagnosis is clear; if he is abstemious, he suffers from the excesses of his forefathers; if the urine deposits lithates on a cold morning, then lithiasis, lithæmia, and a gouty disposition are easy steps in reasoning; if the urine is albuminous, he has a gouty kidney; if it is pale and free from deposit, that is a proof that uric acid is not properly eliminated, and must be accumulating in the blood. Diabetes is "notoriously" gouty, so is dyspepsia, gravel, bronchitis, and asthma, and "skin diseases" in general. If any doubts remain, they must yield before the voluminous testimony which is laid before us every year of the effects of the fashionable waters of the Continent where cures are still wrought.*

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* The late Professor Hebra used to relate how a patient, who had gone the round of "baths" and "cures" for *Gicht*, came for his advice, and was found when the feet were examined to suffer from nothing but corns. He did not, however, anticipate that corns themselves would one day be regarded as a proof of the gouty diathesis!

We must learn to explain less and to investigate more; and by keeping to the narrow path of terms which admit of strict definition, of ætiology which is logically demonstrable, and of diagnosis which can be verified or refuted, slower but surer progress will be made, and our successors will not regard our pathology as we do that of Eugalenus.

Event and prognosis.—Acute gouty arthritis is never directly fatal; the prognosis relates to the ultimate effect on the health of the patient.

It was once deemed rather an honour than a misfortune to have the gout; it showed that not only the patient himself, but perhaps his father and grandfather before him, had been able to afford good living. Sydenham, as we have seen, took ironical comfort in the fact that gout, unlike any other disease, kills more rich men than poor, more wise men than simple. Such consolatory reflections may now be checked by the consideration that insurance offices charge an additional premium to anyone who has had an attack of gout, for experience shows that on the whole the disease tends to shorten life. However slight it may have been, a seizure of gout is always an admonition that the patient's habits of life are incompatible with prolonged maintenance of sound health.

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* The following is extracted from an advertisement of a certain mineral water:—"I am very much pleased to be able to inform you that, according to the advice of my friend ——, M.D., medical officer of the staff, I used your mineral water against the arthritic affection from which I have been suffering the last ten years with such success, that I think it my duty and in the interest of suffering humanity to relate to you the history of my illness. Already ten years ago I had an attack of an acute rheumatic fever, which developed itself to such a severe gout that I had to keep in bed, and was suffering from the most awful pains for weeks every spring. I tried different cures and visited watering-places, but without any effect. In the month of August last year I commenced to drink, and after having consumed 150 bottles I was delivered from all sufferings, and was able to get out of bed. The inflammatory alterations in the joints and the chronic sediments of uric acid disappeared, and I can say to be totally cured. It promotes the secretion of urine, and acts also very favourably on digestion, and is a very pleasant refreshing drink.—28th May, 1887. F— B—, curate in the Austrian army."

Sir Thomas Watson says that "in not a few instances men of good sense, and masters of themselves, having been warned by one visitation of the gout, have thenceforward resolutely abstained from rich living and from wine and strong drinks of all kinds, and they have been rewarded by complete immunity from any return of the disease; or, at any rate, its future assaults upon them have been few and feeble." "I am sure," he adds, "it is worth any young man's while, who has had the gout, to become a teetotaler." For the earlier the age at which a first seizure occurs the worse is the prognosis; and particularly if there be an inherited predisposition. Garrod says that he has known thirty-five years elapse between a regular attack of gout in the great toe and the patient's death, which latter event took place when he was seventy years old. He has also seen several cases in which the disease, after having recurred periodically for many years, gradually declined in intensity and duration, and at last altogether disappeared.

The repetition of attacks of gout is a serious matter, not only on account of the severe pain, but of the ultimate crippling it leads to. But a more serious risk is its liability to induce *chronic interstitial nephritis*, or renal cirrhosis, so that Dr Todd called the "small, red contracted kidney" the "gouty" kidney. When the disease is caused by gout, white streaks are often seen, running in the course of the straight tubes in the pyramids; these white streaks consist of prismatic crystals of biurate of soda and of amorphous masses, blocking up the tubes, and embedded in their walls.

The signs by which this affection of the kidneys may be detected will be fully discussed in the chapter on Bright's disease. A prolonged examination of the urine, both chemical and microscopical, is often necessary. But merely looking at the secretion may be enough to excite suspicion. Sydenham remarked that in cases of long-standing gout the urine, "no longer high-coloured, is pale and copious, like the urine of diabetes." This pale copious urine, unlike that of diabetes in having a low specific gravity, points, as we now know, to renal cirrhosis. We also know that hypertrophy of the heart follows this form of Bright's disease, and that apoplexy frequently results.

Evidence of Bright's disease was found in a third of the writer's sixty-one cases of gout. In many of them atheroma was also present. In ten consecutive cases of patients who died after repeated attacks of gout, two died of cancer, and in the other eight the writer found granular kidneys. The immediate causes of death were renal pericarditis, pleurisy or pneumonia, uræmic coma, and cerebral hæmorrhage or softening, with hypertrophied left ventricle and diseased arteries.

Of diseases of the skin the only one which has any probable relation to gout is *eczema*. This is so common that few cases of eczema are gouty, but those affected with chronic gout are more subject to eczema than other persons of the same age, and the disease is apt to be dry and particularly irritable. Its cure is also helped by alkalies and colchicum.

In a gouty patient bronchitis may be renal, or senile; not infrequently it proves the immediate cause of death in long-standing cases.

It has been said of gout, as of cancer, that it is incompatible with *phthisis*. This, however, is not the case. The fact that they affect different periods of life, so that few have gout who die under thirty, explains their seldom being met with together, but in the small number of sixty-one cases above quoted the ordinary symptoms of phthisis occurred three times in patients with well-marked gout. In a fourth case of gout tubercles were found after death in the lungs, and in February, 1888, one of the writer's patients of Philip Ward with unmistakable tophi was also suffering from tuberculous disease of the larynx and lungs.

Treatment.—The pain of a fit of the gout is so great that it demands every effort to relieve it. The great Dr Harvey was one of the eminent men subject to this complaint, and he used to cut short a fit by plunging his feet into cold water. This and like attempts to stop the local inflammation were always condemned on the supposition that they "drove in the peccant humour," and would lead to gout of the stomach or the brain But no recent and carefully observed cases of such treatment seem to be on record.* The judicious Heberden remarks, "I do not recommend Harvey's example (of which I have been told by some of his relations) as proper to be imitated, though it is known he lived to a good old age; but I am not warranted by any experience to condemn the practice of endeavouring, by exercising the limb, to prevent the gout from settling there." There are numerous instances of persons subject to gout who, on the first intimations of a fresh attack, have begun walking, riding, or some other prolonged and violent exercise, and have thus escaped the threatened invasion.

Hot fomentations sometimes give ease; more often evaporating lotions of chloroform, ether, or alcohol. Lint soaked in lead and opium, infusion of poppy-heads, or laudanum, applied to the inflamed joint and lightly covered with cotton wool, will do good in different cases. Local applications of colchicum are useless, blistering and iodine are hurtful, and leeches do not relieve the pain as one would have hoped. Scudamore's favourite lotion was made of one part of alcohol and three of camphor water, made lukewarm and applied on thick linen compresses. A local anodyne recommended by Dr Fagge was a solution of one grain of atropine and eight grains of hydrochlorate of morphia to the fluid ounce, applied on lint beneath oil-silk. Garrod also advises that oil-silk or gutta-percha sheeting be carefully applied, so as to keep the skin in a kind of vapour-bath. He has seen irremediable injury from the application of leeches to joints affected with gout.

The foot should be kept higher than the hip, and well protected from accident by light bandages of cotton wool, and under them lint soaked in belladonna liniment or warm lead lotion.

Sir William Temple gives an admirable description of how he cured his first fit of the gout when, in February, 1674, he was forty-seven years of age and ambassador at the Hague, by applying "a certain kind of moss that grew in the East Indies, and is called a moxa," to his foot and setting it alight. This kind of actual cautery was afterwards much employed in sciatica and other painful disorders, but it is now almost forgotten. It more than once proved effectual in Temple's case, and also in one of toothache.

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* Dr Parry, of Bath, had, as Watson tells us, at one time two patients under his care, each of whom had attempted to cut short or to ease a paroxysm of gout by plunging the affected part into cold water. This gave instant relief to the pain, and the inflammation presently abated, but in each case hemiplegia occurred a few hours afterwards.

Trousseau relates a case that occurred to Dr Demarquay of a gentleman who applied cold water compresses to his foot, which was affected with severe gout. The pain was almost immediately relieved, but a few hours later the patient fell into a state of apoplectic semi-stupor, which disappeared under the use of sinapisms to the foot, and consequent return of the articular inflammation.

In Parry's patients the cerebral symptoms were probably due to rupture of a cerebral artery, and in Demarquay's to uræmia. In none is there evidence of the supposed causal *relation.*

He narrates of Prince Maurice of Nassau, that though often attacked with gout he laughed at it, and was sure he could always cure it with one remedy; this was to boil in water a good quantity of horse-dung from a stone-horse of the Hermelinne colour, and set his leg in a pailful of it as hot as he could bear it. Temple adds that "he even had a set of such Hermelinne horses in his coach, which he told me was on purpose that he might never want this remedy." He mentions other treatment, including carbonate of ammonia internally, and shampooing and urtication locally, and concludes that exercise and temperance are better than them all ('Works,' vol. iii, pp. 238—265).

Happily the *internal treatment* is more efficient, and gout is one of the few diseases for which modern medicine has found a specific remedy. When Sydenham wrote, he spoke of the possibility of such a discovery, which he said would delight him above all other physicians; but he knew of no specific for gout.

The ancient writers recommended hellebore and hermodactyl. The latter plant has been identified with *Iris tuberosa;* it was more probably a species of colchicum, but not that which is now officinal. The Portland powder was a famous nostrum for gout in the last century, until it was eclipsed by the *eau médicinale de Husson,* which owed its virtues to meadow saffron (*Colchicum autumnale*). But it was long before colchicum was used as it deserved; perhaps it bore a taint from its introduction as a quack nostrum, for many writers have depreciated its value. Some asserted that it only does good by purging; others, that when it removes the local symptoms it leaves "the disposition to the disease much stronger in the system." This was the opinion of Scudamore. Cullen advised patience and flannel alone, and Trousseau advised his pupils to cross their arms and look on, doing absolutely nothing to subdue an attack of acute gout.

Very different from this is modern experience. Watson prescribed forty minims or a drachm of the colchicum wine in a saline draught at bedtime, and half a drachm more in a warm draught the next morning, repeating the sequence if the disease continued. Garrod gave about twenty minims of the wine every six hours; and both say that the effect is marvellous. Roberts declared that for controlling the inflammation and shortening the attack there is no remedy comparable to colchicum. He advised 10—25 drops of the wine two or three times a day.

It is certain that the curative action of colchicum is not dependent upon its purgative operation, for it is often effectual when it does not act upon the bowels at all. Under its use the stools acquire a green colour.

In the severity of a gouty attack it is well to add twenty drops of laudanum to forty of colchicum wine: but the freedom of the kidneys from disease must first be ascertained. It is a common and probably a good practice to give a blue pill or colocynth and calomel purge at the same time.

Many French physicians advise the administration of salicylate of soda in acute gout, but experience in England shows this to be inferior to colchicum in immediate effect, and the results are sometimes negative, if not injurious. It is right to add that Haig and also Fawcett have found the output of lithic acid increased under the influence of salicylates; but see Luff's remarks (*loc. cit.*, p. 193). In cases where colchicum causes too much cardiac depression to be safely used in full doses, it is probably better to give a blue pill and black draught, a laxative dose of sulphate of magnesia every succeeding morning, and ten or fifteen grains of Dover's powder every night,—always supposing that the urine is healthy.

The *diet* in an attack of acute gout should for the first few days consist of milk, arrowroot, sago, tapioca, and the like, with barley-water, water, or toast and water. The less alcohol taken in any form the better, but there is no reason to forbid tea or coffee.[*] Sometimes, however, we see gout in a patient so broken down by previous attacks that he requires as much nourishment as the stomach will digest,—beef-tea, soup, and eggs, with abundance of milk, and even a little brandy.

The *treatment of chronic gout* must be less energetic than in an acute attack. Colchicum is almost always useful, even in inveterate cases.[†]

Alkalies are indicated in gout, and they may often be advantageously given with colchicum; a traditional prescription is to add ten or fifteen grains of carbonate of magnesia to fifteen or twenty of colchicum wine in a draught of camphor, peppermint, or chloroform water, taken thrice daily; another way in which an alkali can be given is in the form of potassium salts of citric, tartaric, or acetic acid, in the hope that the base may combine with uric acid, and help its elimination by the kidneys. The urate of potass is more soluble than that of soda, and potass also possesses greater powers as a diuretic. Sir William Roberts believed that soda salts are decidedly injurious, and was often disappointed with those of potash. The salts of lithia were introduced by Garrod as possessing some advantage over those of potass in the still greater solubility of the urate of that base; but perhaps this gain does not outweigh the disadvantage of their higher price. The carbonate of lithia may be given in doses of five to ten grains dissolved in aërated water, or the citrate in doses of eight to twelve grains or more. Whether potass or lithia salts are prescribed, they should be largely diluted with water, and taken at least an hour before meals, when the stomach is empty.

Haig believes that alkalies increase the excretion of uric acid, while acids decidedly diminish it, but Luff gives reason to doubt this. Sarcosin, which diminishes the excretion of uric acid, has been recommended on theoretical grounds as a remedy for gout by Schultzen.

Guaiacum is very useful in many cases of chronic gout. Garrod strongly recommends it, especially for old subjects; a patient may go on taking it for a whole year at a time. Bark or quinine is also useful in some cases of what is called atonic gout, *i. e.* when the attacks have become more frequent, short, and slight, while the patient's digestive powers, his strength, and his appetite fail. In these cases opiates are often more useful than colchicum during a subacute attack; and stimulants, particularly old port wine or brandy, and even good champagne, are not only admissible, but therapeutically valuable.

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[*] "Potus theæ et caffeæ inter reliqua remedia calculosis et podagricis excellunt" (Baglivi, 'Prax. Med.,' lib. i, p. 117).

[†] I remember a patient who lay in bed week after week with deformed and crippled joints until I prescribed colchicum, which I had at first supposed to be little likely to be of service to him. In a very short time he was free from pain and about the ward.—C. H. F.

But as a rule a patient subject to gout should live an abstemious life, taking no stimulants, and meat only once a day, eating freely of vegetables and fruit, and taking occasional saline laxatives. He should also drink water on rising and going to bed. Alkaline and diuretic waters are no doubt often valuable aids to treatment and prophylaxis. Even more desirable are active habits and habitual exercise in the open air.

Sir William Roberts advised that the amount of sodium chloride taken with food should be diminished, and found that patients can be induced to substitute potassium chloride as a condiment with benefit.

Piperazine has been strongly recommended and largely advertised as a specific in gout. It is an alkali, and as such a solvent of uric acid, but has no chemical action to recommend it as preventing the deposit of biurates (see Sir William Roberts's Croonian Lectures for 1892, and Dr Fawcett's paper on the subject in the 'Guy's Hospital Reports' for 1894-5.

Certain *baths and mineral waters* have so great a reputation that most patients, among the richer classes, who are supposed to be "gouty," sooner or later visit one of them. Of such resorts, Vichy was the most in vogue under the second Empire; the main ingredient of its springs is carbonate of soda in the proportion of about forty grains to the pint. Another fashionable water is that of Vals, and a third (indifferent) Contrexéville. Many persons who are robust and of full habit appear to derive benefit from these springs; but to feeble patients they often do harm. Garrod says that in very chronic cases the formation of tophi appears in his experience to be hastened by the use of Vichy water. Trousseau advised that as a general rule alkaline water should not be taken for more than ten or twelve days at a time, and only in small quantities, for that not a year passed without his seeing evil consequences from their prolonged use. Mineral waters should not be taken when an acute attack is present or threatening; nor by patients who have organic disease of heart or kidneys. It is worthy of remark that, according to Heberden, the Bath was valued during the eighteenth century for the efficacy of its waters in *bringing out* an attack of gout.

Since 1870 the waters of Carlsbad are perhaps the most fashionable; next come those of Marienbad, which also are purgative; Wiesbaden, which contain chloride of sodium; and Aix-les-Bains, saline or sulphurous. Old or infirm persons may be sent to Bath or Buxton, where the waters are hot and the saline ingredients small in quantity; or Teplitz or Gastein may be chosen if more distant places are preferred.

Sulphate of magnesia is often valuable as a laxative, particularly in those who are called "gouty" without ever having had gout; but the sulphate and other salts of soda are, so far as chemistry is a safe indicator, injurious in true gout.

No reasonable person will pretend that it makes any difference whether a patient takes the salts contained in natural springs at their source, or the same chemical compounds at home. The benefit unquestionably derived by some patients from "taking the waters" may be stated thus:—First, they drink a great deal more water than they otherwise would, and this is a solvent for quadriurate of sodium and most other salts, as well as a potent diuretic and diaphoretic. Secondly, they drink much less wine, beer, and spirits than they do at home. Lastly, they rise early, they eat less, and they walk more.

RHEUMATISM

"Joint-racking rheums."—MILTON.

"The throttling Quinsey 'tis my Star appoints,
And Rheumatisms descend to rack my joints."—DRYDEN.

History and Definition—Onset and symptoms—the inflamed joints—their anatomy —the pyrexia, sweating, &c.—erythema—cardiac complications—pleurisy—tonsillitis—nodules—Event—Relapses—Prognosis: fatal cases—Rheumatic hyperpyrexia—Rheumatism in children—Diagnosis, particularly from pyæmia—Ætiology and pathology of rheumatism.

Treatment by older methods—Observation of the natural course of the disease —Treatment by salicin and salicylates—Treatment of hyperpyrexia.

Chronic articular rheumatism—Muscular rheumatism.

Synonyms.—Rheumatic fever—Acute and subacute Articular Rheumatism—Arthritis vaga—Polyarthritis rheumatica acuta—Rheumarthritis.

Definition.—An acute febrile disease, running an undefined course, with implication of the joints and often of the heart.

History and nomenclature.—The name of rheumatism, like that of gout, carries with it the impress of humoral pathology. The words *rheuma* and *catarrh* are used by the Greek writers from Hippocrates downwards, with similar meaning; and their etymology is also alike, for the one term was derived from ῥέω, and the other from καταῤῥέω. The notion was that of an acrid humour, generated in the brain and distributed over the body. *Destillatio* was a term applied in common to catarrh, rheuma, and gutta. But in course of time, diseases of the mucous membranes became known as catarrhs, while the name of rheumatism was confined to painful affections of the joints, bones, or muscles. Baillou, or Ballonius,[*] is said by Bright and Addison to have given it this restricted meaning; and he also distinguished it from gout or *gutta*, the third name witnessing to the humoral doctrines of Galen. Holland, in his translation of 'Pliny's Natural History' (1600), speaks of the "sharp and eager flux of phlegm which the Greeks call rheumatism" (lib. xxii, cap. 18 and 25). The quotations at the head of the chapter show that articular rheumatism was recognised in literature in the second half of the seventeenth century.

The conception of rheumatism became modified in some countries so as to include the notion of its production by external cold. All sorts of complaints have accordingly been termed rheumatic,—some, of which the cause was unknown, because they were painful; others, although painless, because cold was the supposed cause.[*]

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[*] He died in 1616, and his posthumous work was only published in 1642.

But there is a very common, definite, and important disease, which was first accurately described by Sydenham in 1670, in the sixth section of his 'Observationes Medicæ circa morborum acutorum historiam et curationem' (cap. v). He says it was then usually called "Arthritis," and was confounded with gout: "unde forsan petenda est ratio cur tam sicco illum pede transiverint scriptores medici." For this common and important disease we have no other name than Rheumatism, *i. e.* acute rheumatism, or rheumatic fever. The pyrexia, the multiple but transient synovitis, and the frequent implication of the membranes of the heart sufficiently distinguish it. We will include under the same name all genuine cases, though their course may be "subacute."

As to what is called "chronic rheumatism" there is more difficulty. The term used often to be applied, and still is by many eminent surgeons,[†] to the disease now known as *osteo-arthritis* or *arthritis deformans*, which bears no relation whatever to acute rheumatism. But there are exceptional cases in which a chronic affection of a joint can only be described as a sequel of the acute disease. The question will be resumed at the close of this chapter.

The use of the adjective "rheumatic" ought, of course, to be limited in precisely the same manner as that of the substantive; but it is often employed loosely for various affections of the muscles and of other parts, of which the only common character is that they are caused (or believed to be caused) by cold. It is much better to speak of "myalgia" than of muscular rheumatism, and of specific periostitis than of syphilitic rheumatism; for the common vague use of the term implies a connection between totally different conditions.

Many years ago Dr Frederick Chambers, of St George's Hospital, and Dr Francis Hawkins endeavoured to draw a distinction between two varieties of acute rheumatism, one of which they termed "fibrous," the other "synovial," and their views were adopted in Sir Thomas Watson's 'Lectures.' They were, however, altogether mistaken in supposing that true rheumatism ever attacks the ligaments outside a joint and not the synovial cavity itself, and the clinical distinction they drew, so far as it is real, corresponds to that between two different diseases—true rheumatism (fibrous) and osteo-arthritis (synovial).

The local affection, characteristic of rheumatism, is a synovitis, acute or subacute, with effusion of serum and coagulable lymph, comparable to pleurisy, but not leading to adhesions, nor ending in suppuration or destruction of the tissues.

Onset and course.—Most attacks of rheumatism begin somewhat gradually. For a day or two the patient feels uncomfortable, and perhaps complains of sore throat or of pains in the limbs. There is seldom headache, and still more seldom rigors or vomiting. Pyrexia sets in, and has often reached its full height on the second day, when one of the joints, generally a large joint, is found to be swollen as well as painful. But in many cases inflammation of one or more joints is the first thing noticed.

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[*] This confusion is still common with good German writers, so that it is often as difficult to know what is meant by "rheumatic" as to tell the meaning of "typhus."

[†] Volkmann, for example, and Hutchinson. See a judicious paper by Prof. Bäumler on "Chronischer Gelenk-Rheumatismus," in which he concludes with some reservation that there is no such thing ('Verh. d. xv Congr. f. inner. Med.,' Berlin, 1897).

It is not always easy to determine clinically the fact that fluid is effused into the shoulder-joint, the elbow, or the ankle; but in the knee very small quantities of fluid may be detected with certainty. One has only to grasp the joint gently between the two hands, pressing the sides of the synovial cavity upwards; a slight tap upon the patella with one forefinger will then bring it down upon the condyles, giving a sensation which is unmistakable; or a finger may be placed on each side of the ligamentum patellæ and fluctuation felt.

In those rare cases which prove fatal, all the affected joints show distinct signs of inflammation at the autopsy. The synovial membrane is not, indeed, always injected, for in this, as in so many other tissues, redness may subside after death; but fluid is almost always present, often cloudy and with shreds of fibrin. Not infrequently a separable layer of coagulated exudation lines the synovial membrane, or covers the surface of the cartilages. Leucocytes are present in the exudation, but not in large numbers. In one case, examined by Dr Moxon, the sheaths of the extensor tendons of the wrist were full of opaque serum and of masses of greenish-yellow lymph.

That the anatomical changes in the joints are but slight might be inferred from the clinical history of rheumatism. One of its most striking features is the rapidity with which it flies from one part to another. Thus a joint may be extremely swollen one day and normal on the next. There is not always any obvious reddening of the skin; generally some ill-defined pink patches are seen, but they are often near the articulation rather than over it. Only rarely is the skin so red as to look like gout.

The different joints are sometimes attacked in regular order: the right ankle, then the left; the right knee, then the left; and so on. But more often the distribution of the disease is quite irregular. All four limbs usually suffer more or less.

Of all the joints, the knees are most often affected in rheumatic fever, next come the shoulders and ankles, then the wrists and hands, and then the elbows. The hips are less frequently attacked and the toes often escape, even in severe cases. The mandibular joint has almost complete immunity, and probably the same is true of the articulations of the vertebræ and pelvis, but they are too deeply seated to be examined during life, and probably no traces would be left if they were dissected after death. It is said that acute rheumatism sometimes attacks the synchondroses of the pelvis.

This distribution differs from that of gout, gonorrhœal synovitis, and osteo-arthritis; moreover, in no other kind of arthritis does the inflammation shift rapidly from joint to joint.

In some exceptional cases rheumatic synovitis, instead of subsiding, passes on into a chronic stage, and ends in hydrops articuli, most likely of the knee. Ankylosis or disorganisation of the joint is so extremely rare that its occurrence throws doubt on the diagnosis of rheumatism.

There is no evidence whatever that rheumatic inflammation attacks any but synovial and serous membranes. It has no predilection for fibrous tissues, whether the fibres be white, elastic, or muscular.

The patient dreads the gentlest touch or the jar of a passing footstep; with his head and limbs immoveably fixed, he turns his eyes with anxiety as a stranger approaches his bed. He lies perfectly helpless, unable to feed himself, or even to turn in bed, paralysed by the dread of pain; so that hospital patients often speak of having lost the use of their limbs. The pain is usually worse by night than during the day. The face is pallid, the tongue thickly furred, and the face and body covered with sweat.

The *pyrexia* of rheumatism in most cases corresponds to the number of joints involved, and the severity of the inflammation. But to this rule there are many exceptions even in adults. Sometimes the articular affection is well marked while the temperature is scarcely raised. The converse may also occur,—an attack of pyrexia, with pericarditis or endocarditis, or perhaps with pleurisy alone, may be followed by a relapse which is attended with synovitis, and thus proved to be rheumatism. Graves cited a case of relapse, the patient having previously passed through two attacks of the usual kind, to prove that it is possible for the fever to occur alone without synovitis.

Acute rheumatism is a general disease with localisation in the joints, not a local disease of the joints with symptomatic fever; but Wunderlich was unable to discover any typical course of temperature. The maximum, which is usually about 104° F., sometimes occurs as early as the fourth, third, or the second day, but more often not until the end of the first or the beginning of the second week. The *pulse* is often extremely rapid, large, strong, and short; and sometimes it is markedly dicrotic. The breathing is not proportionately hurried. The *tongue* is generally flabby, marked by the teeth at its sides, and coated with a thick white fur. There is usually thirst and anorexia, but the patient can sleep if not kept awake by the pain. He is not apathetic as in other fevers, and is seldom delirious unless his temperature runs very high; if, however, he has been a drunkard, a form of delirium tremens is often developed. There is no tendency for the fever to assume a "typhoid" character.

The *urine* is characteristically febrile: scanty, dense, high-coloured, acid, depositing pink lithates in abundance, and often crystals of lithic acid; occasionally it contains a trace of albumen for a day or two. It does not contain sugar, but often reduces copper when salicin or salicylic acid is being taken.

One of the striking symptoms of untreated rheumatism is *sweating*. The patient lies bathed in water, so as to make even the blankets damp. This must, of course, carry away much heat by evaporation from the surface of the body, but it has no obvious influence in lowering the pyrexia or in relieving the articular pains. The sour smell of the sweat seems to be due to its quantity rather than to its containing an unusual quantity of free acid. Sir William Gull used to show that the reaction to litmus-paper often varies upon different parts of the skin, being acid, alkaline, and neutral in the same patient. Sweat when first secreted is not acid, and only becomes so by decomposition of the sebaceous compounds mingled with it. Senator believes that the sweat may become alkaline by conversion of urea into carbonate of ammonia, particularly between the toes and in the armpits.

There is frequently developed a copious eruption of *sudamina*—minute transparent vesicles, containing an acid fluid which is no doubt sweat. They glisten when a bright light falls upon them, but they are colourless, so that they often can more easily be felt than seen. After a while their bases become slightly reddened, and their contents opalescent and alkaline:

by a somewhat pedantic change of name these inflamed sudamina have had the old synonym *miliaria* reserved for them. This eruption bears no special relation to rheumatism, for it occurs in other diseases attended with sweating, and also in healthy people during hot weather.*

In some cases of rheumatism there is another remarkable eruption, which assumes the form of erythema, urticaria, or purpura. It has often been described under the name of *Peliosis rheumatica*, originally applied by Schönlein. Varieties of the same rash have been named Erythema tuberculatum, Roseola marginata, Erythema nodosum, and Purpura urticans. These are all varieties of *Erythema multiforme*, and will be described under that name in the section on diseases of the skin.

That this is a true rheumatic eruption, and not a cutaneous hæmorrhage with secondary effusion into the joints, as in scurvy and hæmophilia, is certain, for it occurs in those who have had rheumatic fever before, in those who have had chorea, and in cases complicated with endo- and pericarditis. Moreover there is no bleeding from the gums or other mucous membranes.

Cardiac inflammation.—The important relation of rheumatism to disease of the heart was not fully recognised until after the discovery of auscultation, and was unnoticed by Laennec and his immediate successors in France. It is a mistake to suppose that it was first recognised by Bouillaud in 1835; in 'Outlines of a Course of Lectures' delivered at Guy's Hospital by Babington and Curry in 1811 it is stated (p. 87), "Acute carditis is of more frequent occurrence than generally believed, but perhaps oftener a metastatic or misplaced form of Rheumatitis (acute rheumatism of authors) than a primary disease allied to Pleuritis and Pneumonia." Hope writes, "I have not the slightest pretension to originality in this idea, since at the time I wrote [1831, in the first edition of his book, candidly quoted by Bouillaud] there was not a better established doctrine in the London schools." The connection of rheumatic fever with cardiac disease was known to Alex. Pitcairn and published in 1794 by Baillie, whose words are ('Morbid Anat.,' p. 51, fifth edition), "Dr Pitcairn observed this in several cases [cardiac hypertrophy as the result of rheumatism], and is to be considered the first person who made this important observation." Corvisart (*c.* 1810) ascribed adherence of the pericardium to "gout or rheumatism." The relation of heart disease to rheumatic fever was well known to Scudamore, Bright, Elliotson, and Latham. Here, as elsewhere, no sudden and complete discovery was made by anyone. Our present knowledge is the result of many independent observations, and even now is far from perfect.

In every case of rheumatism we must watch day by day with the stethoscope for indications of inflammation of the pericardium or the cardiac valves, an inflammation which often causes neither pain nor any other symptom.

The frequency of inflammation of the heart in rheumatism cannot be stated with numerical accuracy. The results of autopsies are not available for the purpose, because fatal cases are not average ones; and auscultatory signs during life are open to sources of fallacy, particularly the difficulty of distinguishing between organic and functional murmurs.

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* The "febris miliaris" which occurred as an epidemic on the Continent, and especially in France, throughout the eighteenth century and down to the year 1856, may very likely have included cases of rheumatic fever, and it was attended with a red papular or vesicular rash. The name is still used in Italy, but probably for scarlatina, enterica, and other febrile diseases attended by sweating and sudamina. In the "sweating sickness," of which there were five terrible epidemics between 1485 and 1551 in this country, there seems to be no record of such an eruption (cf. *supra*, p. 268).

Among forty-five cases of rheumatic fever which ended fatally at Guy's Hospital, and in which there had been no previous chronic disease of the valves, Dr Fagge found the heart to be healthy in eight only; both pericarditis and endocarditis existed in nineteen, pericarditis alone in ten, endocarditis alone in eight.

Turning to clinical records, we find that Bouillaud estimated the occurrence of cardiac lesions at 80 per cent., but that was in patients who were bled and leeched without remorse. Budd put their proportion at 48 per cent., Fagge at 50 to 60 per cent., Fuller at only 23 per cent., and the same proportion, 23 per cent., was independently arrived at by Lebert at Breslau, and by Wunderlich at Leipzig.

Dr Sibson found in 326 cases observed at St Mary's Hospital, between 1851 and 1866 inclusive, 63 of pericarditis, 108 of endocarditis, and 54 of both together, beside 76 in which the physical signs were doubtful, leaving only 79 cases certainly free, or about one fourth of the whole ('Reynolds' System'). At St Bartholomew's Hospital, out of Dr Church's 574 cases in which the state of the heart was ascertained, it was affected in 371, about 66 per cent. ('Allbutt's System'). Dr Peacock recorded cardiac murmurs in only a third of his cases ('St Thos. Hosp. Rep.,' 1873).

The present writer collected 400 cases for his paper in the Guy's 'Reports' for 1874 (vol. xix, p. 311), and Mr Shadwell added 100 more for the second edition of the present work. Of these 500 cases from the clinical records of Guy's Hospital, only 221 were free from cardiac murmurs; while in the remaining 279 cases, 110 bruits were believed to be pericardial and 169 endocardial, making 56 per cent. together.

There is little doubt that acute inflammation of the heart is occasionally the sole indication of a rheumatic attack, and that it is often attended with only slight and fugitive articular pains. Nevertheless, as a rule. there is a distinct relation between the degree of severity of the synovitis and the frequency of cardiac complications. Sibson found that the affection of the joints was severe in only one fourth of those cases in which the heart showed no sign of inflammation, but in two fifths of those in which endocarditis was present, and in three fifths of those in which there was pericarditis, with or without endocarditis.

The frequency of inflammation of the heart in rheumatism depends also on the age, and the younger the patient, the more liable to it he is. This is strikingly shown by the third table in Dr Church's paper in the 'St Barth. Hosp. Rep.,' vol. xxiii, p. 273, where the percentage of cardiac affections in successive decades from under ten to fifty runs thus:—83, 69, 51, 30, 21. In young female servants, who make up a large proportion of the cases of rheumatism seen in hospital, the heart scarcely ever, according to Sibson, escapes. Latham also said that maid-servants with rheumatic fever in hospital always get pericarditis. This, however, is not in accordance with present experience.

Older patients escape more often; and here a curious difference is found to exist between men and women. Pericarditis seems to be much commoner in girls between fifteen and twenty-five than in young men of that age; and to be three times as frequent in men above the age of twenty-five as in women above that age. The reason for this is probably that early rest in bed lessens the likelihood of cardiac complications in rheumatism; most women can give up work sooner than men of the same age, but the girls of the lower classes are very apt to have their strength overtaxed.

There can be little doubt that while rheumatic endocarditis is apparently neither more nor less common than before, pericarditis is far less so. Comparing his recent experience with that recorded by Bouillaud, Latham, Todd, and even the practice of Guy's Hospital in 1860-70, the present writer is convinced that pericarditis in rheumatism is now comparatively rare. It is probable that, taking all cases of acute cardiac implication in rheumatism, only the minority affect the pericardium, either alone or with the endocardium; although this cannot be certainly affirmed, because pericarditis is often transient, and leaves no trace behind it. Adherent pericardium is much less commonly found after death than valvular lesions—excluding those which in later life are due to atheroma; and a pericardial rub is certainly less frequent than an endocardial murmur as estimated by auscultation during life.

Judging by the same clinical method, the mitral valve is much more often affected in rheumatic endocarditis than the aortic, and mitral incompetence is more common than mitral stenosis. When a presystolic bruit or other signs lead to the diagnosis of the latter lesion, we may be almost sure that there have been one or more previous attacks of rheumatic endocarditis.

Other complications of rheumatism are comparatively few and unimportant. Pleurisy, affecting chiefly the left pleura, is most frequent. It often accompanies pericarditis.

Bronchitis is only occasionally present; the lungs are often œdematous; and, in fatal cases, the microscope may show them to be affected with slight catarrhal pneumonia. Lobar pneumonia is of somewhat rare occurrence, but when present commonly leads to death.

Rheumatic peritonitis and iritis are both excessively rare; and rheumatic meningitis, cerebral or spinal, may be said not to exist.

Acute tonsillitis sometimes begins an attack of rheumatism, but it has no features to distinguish it from an accidental sore throat.

Chorea is doubtless closely allied to rheumatism, but as a sequel rather than a complication.

Rheumatic nodules.—An effect of rheumatic fever, of both pathological and diagnostic interest, consists in the formation of small, subcutaneous, fibrous nodules, usually but not always in the neighbourhood of joints, often upon prominent points of bone, like the knuckles, the olecranon, the tibia, and the acromion, or on the superior curved line of the occipital bone. They were first described by the late Dr Hillier in his 'Diseases of Children' (1868), and afterwards by Meynet, of Lyons, in 1875, by Rehn in Germany (1878), and by Hirschsprung in Denmark (1881). A full account of them by Dr Barlow and Dr Warner will be found in the 'Transactions of the International Congress of 1881,' vol. iv, p. 116, with twenty-seven cases.*

These nodules are rarely seen in adults, but are frequent in children. They are not very tender, and usually disappear soon after the attack of rheumatism is over. They have been found in the pericardium after death; and when numerous on the surface of the body during life are in Dr Cheadle's experience indicative of severe inflammation of the pericardium, and of a fatal result from that or from endocardial lesions. Occasionally these rheumatic nodules are much larger than usual, the size of a hazel- or even a walnut.

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* Numerous cases were recorded by Dr Cavafy ('Path. Trans.,' xxxiv, p. 41), Drs Lees, Duckworth, Money, Drewitt, Stephen Mackenzie, and Fowler in the 'Transactions' of the Pathological Society for 1883, and of the Clinical Society for 1883-4. A remarkable case in a woman of thirty-nine is described and figured by Dr G. S. Middleton, of Glasgow, in the 'American Journ. of Med. Sci.,' October, 1887.

Event.—In the great majority of cases an attack of rheumatism when left to itself slowly and gradually improves, the pains diminish, the joints recover, and the fever subsides. This takes place in a variable time, occasionally in a week or ten days, often not until three weeks have elapsed, and sometimes only after six or eight weeks. The duration as well as the symptoms of rheumatism, however, is now happily so much modified by treatment, that we scarcely ever see the disease in its natural severity or persistence.

Relapses.—Of all acute diseases (except spirillum fever and ague), rheumatism is the most prone to relapse. In a large proportion of cases—varying in different times and places and under different modes of treatment from a sixth to a fourth or even a third of the whole number—only a few days after the first attack has subsided, a second comes on; this runs a similar though usually a milder and a shorter course, and the heart, if it has escaped before, is seldom affected now. A second relapse is common, and a third and a fourth are sometimes seen. These are less severe and prolonged than the original attack, but may be repeated again and again, until the disease becomes chronic in duration though still acute or subacute in symptoms.

Dr Church has shown that relapses are much more common after second than after first, and still more so after third attacks. The question of how far they can be prevented by regimen and drugs will be presently considered, for it is one important object of treatment. There is no doubt that prolonged care and continued treatment during convalescence are influential in preventing relapses. Hence they are comparatively rare among patients who can afford a long period of easy convalescence; while it is one of the disappointments of hospital practice to see a patient, who has been sent out perfectly well after an attack of rheumatism, return to the ward a week or two later with all his symptoms renewed.

Protection and results.—So far from protecting against recurrence, one attack of rheumatic fever is a predisposing cause of another. Apart from relapses, the interval is usually from three to five years, and sometimes much longer.

The joints are very seldom permanently injured. Occasionally hydrops articuli remains behind, and after many attacks the joints may undergo the changes described in a subsequent chapter as osteo-arthritic. As a rule, however, when the rheumatic synovitis is once cured, no local deformity results. In this particular true rheumatism differs remarkably from gout and from gonorrhœal synovitis.

Anæmia is a marked effect, in proportion to the severity of the attack. It is scarcely ever absent, and return of colour and of muscular strength is often very slow.

The most serious results of rheumatism are the increased liability to a fresh attack, and the frequent damage to the peri- and endocardium, and the muscular walls of the heart.

Mortality.—A fatal result of rheumatism is very rare. When not due to acute pericarditis, or to some accidental cause, death usually results from hyperpyrexia, a remarkable condition to be presently considered, or occasionally from pneumonia or ulcerative endocarditis. Dr Church ('Allbutt's System,' vol. iii, p. 1) and Dr Goodhart ('Clin. Trans.,' vol. xiii, p. 123) have each recorded a case of death in acute rheumatism, with only very slight and early endo- or pericarditis.

Senator puts the average mortality at from 3 to 3·7 per cent. of those who are attacked, and at Guy's Hospital in the 400 cases collected by the present writer it was nearly the same, 3·75. So also in the 655 cases of the Collective Investigation Report ('Brit. Med. Journ.,' February 25th, 1888), the mortality was 3·3 per cent. But at St Bartholomew's Hospital Dr Church found only ten deaths in 693 cases (1·4 per cent.).

But the number of deaths in our wards varied widely in different years. From 1855 to 1867 (thirteen years) only *ten* fatal cases altogether appear in the records. From 1868 to 1884 (seventeen years) there were *fifty* fatal cases, and the numbers in the several years were as follows:—Two in 1868, three in 1869, five in 1870, two in 1871, seven in 1872, two in 1873, seven in 1874, six in 1875, three in 1876, six in 1877, one in 1878, three in 1879, one in 1880, two in 1881, and none in 1882-3-4.

In three of these *fifty* cases the fatal termination was due to stenosis of the mitral valve, which had resulted from previous attacks of acute rheumatism. In only four cases was there found a severe and recent lesion of the valves as the direct result of the rheumatism.

In eighteen instances death was attributed to pericarditis, which was sometimes severe, the heart being covered with lymph, and there being from ten to sixteen ounces of fluid, often stained with blood. In several of these cases there was also effusion into one or both of the pleural cavities; twice there was inflammation of the mediastinum; in seven cases the muscular substance of the heart was obviously involved in the inflammatory process. Each of the complications in question may be supposed to have helped in bringing about the patient's death; and it is not improbable that myocarditis was sometimes present when it was not noticed.

Although recent endocarditis existed in eleven of the eighteen cases, it was so slight that it could not be considered to have affected the issue.

In twenty-seven fatal cases of rheumatism collected by the writer, subsequently to the last series, death occurred from hyperpyrexia with delirium in five, the ages of the patients ranging from twenty-three to thirty-one. One patient died from delirium tremens with acute pneumonia, four from pneumonia with cardiac lesions, thirteen from severe pericarditis or valvular disease or both, the immediate cause of death in two being ulcerative endocarditis and embolism, complicated in both cases by chorea. The remaining three died from accidental causes (diphtheria, enteric fever, and epilepsy) which supervened while under treatment for rheumatism.

Rheumatic hyperpyrexia.—That cerebral symptoms sometimes interrupt the course of rheumatic fever, and prove rapidly fatal, has long been known. By Sir Thomas Watson and Dr Latham it was thought that they depended on cardiac complications, and particularly on pericarditis; but in many cases the heart is found free from all signs of inflammation. The existence of meningitis has also been disproved.

In 1867 Dr Ringer related three cases of rheumatic fever in which extremely high temperatures were observed, the thermometer having risen from 104°—105° to 109·2° or even 110·8° Fahr. Dr Kreuser had noticed the same fact in Würtemberg about a year previously. Many similar cases have been since recorded, both in England and abroad; at Guy's Hospital in seven years fourteen patients died of hyperpyrexia in acute rheumatism.

The most probable pathology of this terrible complication is that while the high temperature is consequent upon an exhaustion of the heat-regulating centre in the bulb, the cerebral symptoms in their turn result from the action of the over-heated blood upon the brain.

Unlike the occasional high temperatures observed in severe cases of pneumonia or typhus shortly before death, the hyperpyrexia of acute rheumatism may suddenly supervene in an apparently favourable case. One of Dr Ringer's patients seemed to have recovered, and was to leave the hospital the next day, when cerebral symptoms set in, and he died in two hours, with a temperature of 110°.

A frequent indication of the onset of hyperpyrexia is that the patient loses his pains and finds that he can move his limbs freely. Unless the temperature falls at the same time, this is a warning of impending danger, and should lead to the use of the thermometer at regular intervals of ten, twenty, or thirty minutes. Another warning symptom is that the skin ceases to perspire.

The symptoms of rheumatic hyperpyrexia vary in character in different cases. Sometimes the patient becomes drowsy, appears to fall asleep, and rapidly passes into a state of unconsciousness, with contraction of the pupils, and dies comatose. Sometimes he grows violently maniacal, jumping out of bed, and fighting with nurses and attendants. Sometimes he is seized with convulsions, or with tonic spasms and opisthotonos. His pulse becomes very frequent, from 140 to 136, and towards the last it may be imperceptible. His breathing is much accelerated, and his face shows the dusky purple flush of venous congestion.

The interval which elapses from the commencement of the hyperpyrexia to the fatal termination is very variable, as is well shown by comparison of twenty-two cases made by Dr Wilson Fox. In one instance the thermometer rose from 103·5° to 109° in two hours, while in two others a period of twenty-four hours passed before a similar point was reached. Even when the temperature is 107° or 108° the patient may now and then continue to live for some hours; and at 110° there has been an interval of from one to two hours before death took place.

Dr Fox thought that hyperpyrexia is more frequent in first attacks of rheumatic fever than in subsequent ones; and subsequent experience has confirmed this belief. As shown in a report based on 67 cases, rheumatic hyperpyrexia is nearly twice as common in men as in women ('Clin. Trans.,' vol. xv, pp. 261—312). It very seldom occurs under puberty.

Of late years rheumatic hyperpyrexia seems to be decidedly less frequent than before 1880, and this we may hope is the result of improved treatment.

Rheumatism in children.—In the case of patients under puberty, rheumatic fever assumes somewhat special characters. It is often subacute without severe pain or synovitis, and with only moderate fever. Moreover gout, gonorrhœal synovitis, and osteo-arthritis are unknown at that age, so that if multiple arthritis with pyrexia is present we have only to consider the possibility of pyæmia or of tuberculous disease—pulpy synovitis or caries

—affecting more than one joint. The articular inflammation is sometimes so slight that pain and swelling have to be inquired for. The heart is, however, extremely apt to become affected; there is a bruit to be heard in far more than half, some writers believe in nearly all cases of rheumatism in children. Hyperpyrexia is very rare. Tonsillitis is common, and erythema is not infrequent. Cases of rheumatic pericarditis or endocarditis or pleurisy, with only slight pyrexia and no discoverable synovitis, are very rare in adults, but not uncommon in children. Chorea is a frequent complication or sequel. Lastly, rheumatic nodules are far more frequent and numerous than in adults. An hereditary origin is more often present, or more easily detected, and one from cold or exposure much less frequent.

Diagnosis.—Acute rheumatism is, as a rule, very easy to recognise. But there are not a few diseases which may be mistaken for it, and in some exceptional instances it may be impossible to decide at once.

Dr Fagge was called into the hospital one Sunday by the house physician to see a girl who had been just admitted for rheumatic fever, and whose temperature was very high. The peculiarity of her case was that she was covered all over with a bright scarlet rash (p. 190). She had had acute rheumatism on a former occasion, and the real nature of her illness was not suspected until the following day, when the papular eruption of *smallpox* was found upon her.

In several instances the pains in the limbs produced by the growth of *multiple sarcomata*, especially about the vertebræ, so as to compress the spinal nerves, have been supposed to be due to acute or to subacute rheumatism. In a case of multiple sarcoma of the skin, secondary to a growth in the cæcum, under the writer's care, there were first pains and swelling about the joints which he mistook for rheumatism, and when the sarcomata appeared he supposed them to be purpuric erythema—peliosis rheumatica.

Affections of the spinal cord in the early stages have been regarded as rheumatic on account of the pains to which they gave rise, until paralysis supervened; and it must not be forgotten that in some spinal cases, and notably in tabes, the joints may actually suffer.

The pains and synovitis produced by *syphilis* have before now been mistaken for rheumatism. *Scorbutus*, *purpura*, and *hæmophilia* must also be remembered as diseases in which articular effusion is by no means rare.

The diagnosis from an acute attack of *gout* is occasionally difficult when the latter occurs for the first time in a young subject, but such cases are mostly hereditary, and normal cardiac sounds would be unusual in rheumatism at that age. The degree of fever, the locality of the arthritis, and, above all, its shifting from joint to joint without leaving traces behind, are the chief additional marks which distinguish rheumatism from gout. The diagnosis from *gonorrhœal synovitis* and from *osteo-arthritis* will be given hereafter (pp. 516 and 527).

A useful means of diagnosis of doubtful cases in children is afforded by the presence of the "rheumatic nodules" described above (p. 489).

The writer had once a man of about fifty under his care, who appeared to be suffering from rheumatic fever; but soon diarrhœa and then a rose-rash appeared, and the diagnosis was altered to that of enteric fever. He died at an early stage of the disease from perforation, and after death it was clear, from the condition of the joints, the heart, and the intestines, that the two diseases had co-existed.

Perhaps the most common and serious error is mistaking for rheumatic fever one of those forms of *pyæmia* which come particularly before physicians because they are not secondary to a wound or obvious source of suppuration. The most frequent primary lesion is caries of the petrous bone, which may be recognised by otorrhœa, or by fœtor of a fragment of cotton wool left in the meatus, even when there is no obvious discharge of pus, by perforation of the membrana tympani, and by tenderness over the mastoid process or the jugular vein. Sometimes the pyæmia is due to acute osteomyelitis of a long bone; or acute periostitis with suppuration and secondary synovitis may in like manner simulate rheumatism. Occasionally, in spite of all care, diagnosis is impossible till after death, and difficult even then.

Cases.—(1) In 1879 Dr Goodhart made an autopsy in the case of a boy aged sixteen, who had been lying for five days in a medical ward in a typhoid condition. He had a systolic murmur; his temperature was 103·8° on admission, his pulse 132, his respirations 48. He had been attacked with pains and with chilliness five days before his admission. Numerous abscesses were found in the lungs and in the kidneys. Evidently, therefore, the case was one of pyæmia; but it was not until after nearly every bone had been examined that Dr Goodhart discovered suppuration beneath the periosteum of the lower end of the right fibula. On the tricuspid valve there was a vegetation of the size of a pea, with a little ulcer beneath, which had torn through some of the chordæ.

(2, 3) Two such cases occurred under the writer's care in 1897. Both patients were boys; both had pyrexia with synovitis, and were admitted for rheumatic fever, but the absence of cardiac symptoms, and the pain being limited to one hip and to one shoulder, as well as the high but intermittent temperature, and in one case rigors, pointed to pyæmia. This was traced to the neck of the femur in one case, and to the scapula in the other; but though active surgical treatment was carried out, both patients died.

(4) A youth was admitted under Dr Moxon in 1877, who had nine days before been attacked with headache, sickness, and rigors, followed by profuse sweating and by pains in the joints. Salicylic acid was prescribed, but the temperature rose and delirium set in, so that cold baths were employed on several occasions. The question whether the disease could be pyæmia was formally discussed, and negatived in favour of the diagnosis of acute rheumatism. He made no complaint of the thigh, as being more painful than other parts, when he was being moved into or out of the bath. Yet at the autopsy not only was there osteitis of the lower part of the shaft of the right femur and of the adjacent epiphysis, but the bone was denuded of its periosteum, and there was a large collection of pus beneath the muscles. There was also suppuration about the shoulder, and pyæmic abscesses in the lungs and heart.

An important distinction between acute rheumatism and pyæmia is that in rheumatism the pain and swelling are transient in one joint. But that this criterion is not perfect is proved by the case of a boy who died of pyæmia in a surgical ward, and who for a time had flying pains in the joints.

Most of the cases of pyæmia from osteomyelitis or periostitis seem to occur in youths between ten and twenty years of age. As a rule, the diagnosis is rendered comparatively easy by the severe constitutional disturbance, by the presence of rigors, and often by the skin being sallow and dry instead of sweating; the joints, too, show a deeper blush, they are more frequently hot, and the temperature is more irregular than in rheumatism.

Certain eruptions, which are sometimes associated with pyæmia, may simulate rheumatic erythema. Beside the scarlet rash which is comparatively common in surgical wards, and is really scarlatina, there are cases in which the skin presents more unusual appearances.

In 1861 a boy of thirteen was admitted into hospital who had for a week been treated r rheumatic fever, but whose disease was at once recognised to be pyæmia, there being a rge abscess of the thigh. Towards the last his body became covered with "a purplish sh, resembling the mottled rash of typhus, partly consisting of petechiæ (probably flea-ts), but also of papules, which became vesicular at the apices and slightly scabbed."

In another patient, in 1874, pustules appeared on the back and on the abdomen two ys before death. Pustular eruptions are also mentioned in two instances of "sponta-ous pyæmia," collected by the Pathological Society's Committee in 1879.

In regard to all such cases, one must be careful not to overlook the esence of *glanders*. That disease at its commencement has been more an once mistaken for acute rheumatism.

Another class of cases which are likely to be set down to acute rheu-tism are those of *pyæmia from gonorrhœa.*

In 1872 a young woman in advanced pregnancy was admitted into Guy's Hospital with t was supposed to be rheumatic fever. An hour later she was delivered of a child, ch survived for some days. She was now seen to be suffering from pyæmia, and four s afterwards she died. At the autopsy an abscess was found in the subserous tissue near right ovary, and there were softening thrombi in the adjacent veins, no doubt starting yosalpinx from a vaginal, probably gonorrhœal discharge.

Ætiology.—Acute rheumatism is generally attributed to cold. But s is open to question.* Exposure to cold winds, or travelling in winter in cold climates, or insufficient clothing does not cause it. The only icient kind of cold is getting wet through. The patient is sometimes acked within a few hours after being out in the rain; but when there s been an interval of a week or more, the causal relation of the events more doubtful. Careful inquiry as to previous exposure to wet and cold s led to a negative statement in probably half of the writer's patients; d there is no question that many, and perhaps the majority, of cases of ute rheumatism are not due to any unusual exposure. The general belief the contrary probably depends on the unlucky ambiguity of the word. uscular "rheumatism" is no doubt caused by wet and chill.

Season.—Rheumatic fever is supposed to be more common in the winter d spring than at other seasons of the year. Haygarth, who in 1805 st published exact statistical observations of cases of rheumatism 70 in number), found as many of them to have begun in the five cold nths (December to April) as in the seven warmer months—not more, haps, than an accidental difference. The number of cases of rheumatism nitted into the wards of hospitals in London at different times is liable wide variations which are not attributable to changes in the weather; for eral weeks hardly a case may present itself, and then a great many y appear within a few days.

A review of the statistics at Guy's Hospital during ten years, collected the writer by Mr Capes, shows that the largest numbers of admissions re in September (158) and November (136); and the next largest in tober, January, and April (127 each). There were not so many in May, ne, and August (108—112); fewer in December, February, and July out 100); and fewest in March (69). Dr Archibald Garrod gives a series similar observations by nine different writers in the fifth chapter of his rk on 'Rheumatism,' and finds great discrepancy in the results. Still, the whole, Dr Gabbet's 2000 cases from the London Hospital, and Dr urch's 2000 all but 6 from St Bartholomew's, show, as those published the present writer, that September, October, and November are the favourite months for Rheumatic Fever, so that we may repeat Sydenham's original statement, "*Nullo non tempore incessit hic morbus, maxime autumno.*"

Climate.—The geographical distribution of the disease is as yet too imperfectly known to enable one to state positively its bearings upon the ætiology of rheumatic fever. Hirsch gives a large amount of information; but with regard to many of the statements which he cites, it is difficult to tell whether acute rheumatism is meant, to the exclusion of the vague affections which are so often labelled "rheumatic." * In Cornwall and South Devon rheumatic fever is comparatively infrequent; in a dispensary practice during four and a half years in Cornwall, only four cases occurred, and medical men practising in Exeter say that it is seldom seen in that city. The Isle of Wight and Guernsey are said to enjoy a similar degree of immunity from the disease. In the United States "rheumatism" appears to be far more frequent in the Southern than in the North-eastern States, where the climate is comparatively cold and changeable. It is said to be rare in Canada and in Australia, both dry climates, though very different in temperature, and to be common in Mexico, in Egypt, and at the Cape, also dry climates. Palgrave found both rheumatism and rheumatic disease of the heart common in the pure, keen, and dry atmosphere of the Nejid in Central Arabia. Both are rare in India and the Tropics generally.

Fatigue is, according to some authors, an exciting cause of rheumatism. It not infrequently sets in immediately after a long march, and its great frequency in servant-girls is supposed to be due to their often being sadly overworked. Some of the occupations which furnish a large number of cases involve hard muscular work. Rheumatic fever is certainly more common among hospital patients than in private practice.

There can be no doubt that certain families are particularly liable to suffer from rheumatic fever; several children of the same parents are often affected by it, and others have chorea. Garrod traced an *hereditary predisposition* in about a quarter of his patients, Fuller in 27, and Chomel in 33 per cent. Among 400 hospital patients the writer found it in 68, *i. e.* in 17 per cent. Dr Archibald Garrod reports that, among 500 cases of patients at St Bartholomew's Hospital who had never had rheumatism, 105, or 21 per cent., gave an account of rheumatic fever in near relatives; while, compared with this general coincidence, he found that, of 500 patients with acute rheumatism themselves, more than 150 (30—35 per cent.) could give a history of the same disease in near relatives.

Whether the disease is more apt to occur in persons of one *complexion* than in those of another is very doubtful. Mr Hutchinson (following Bazin) is convinced that there is a "diathesis" which is termed by him arthritic, and which is common to gout, rheumatism, and arthritis deformans. A very general impression is that most patients with rheumatic fever have light hair and grey eyes. But this applies to all diseases in England, because our population is chiefly xanthochroic.

Rheumatism not infrequently follows *scarlet fever*, usually during the stage of desquamation † (cf. *supra*, p. 176). A similar affection sometimes occurs as a sequel of dysentery, but probably this is not true rheumatism. The puerperal state is also believed to dispose to rheumatic fever.

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* The same remark applies to the statistical tables i, ii, and iii, appendix to Dr Church's valuable article in 'Allbutt's System,' pp. 32-4.

† See papers by Dr Thos. Barlow and by Dr Ashby, 'Brit. Med. Journ.,' Sept. 15, 1883.

Sex.—Men are somewhat more liable than women to rheumatic fever, in about the proportion of 5 to 4; a difference which is probably too small to be significant, though it has been ascribed to the harder work of men and their greater exposure to weather. Of Dr Goodhart's 69 cases in children below fourteen, 27 occurred in boys and 42 in girls.

Among 400 cases at Guy's Hospital the numbers were 223 men to 177 women. Of 654 cases recorded by the Collective Investigation Committee ('Brit. Med. Journ.,' Feb. 25th, 1888) were 375 men to 279 women.

Age.—Rheumatism is a disease of *youth:* a large proportion of first attacks take place in children and in young adults; and it seldom occurs for the first time after the age of fifty:—"Hic morbus, præ cæteris, annis florentes (incessit) et *οἷς γόνυ χλωρόν*" (Sydenham). In infants under two or three years old it is very uncommon; only a few cases have been recorded, one at the age of twenty-three days by Widerhofer, another at four weeks by Stäger, and one at ten months by Henoch (all quoted by Senator).

About four fifths of first attacks occur between eleven and thirty. At Guy's Hospital, out of 365 first cases, 22 fell between five and ten, 179 between ten and twenty, 118 between twenty and thirty, 34 between thirty and forty, and only 12 above forty. Even second and third attacks become less frequent as the patient grows older. The writer has met with one case in a woman of sixty-one, and one in a man of seventy-three.

Putting together 683 cases (whether first or later) from St Bartholomew's Hospital (Dr Garrod) and 620 from Guy's Hospital:—Out of the total 1303, 48 occurred under ten, 521 between ten and twenty, 441 between twenty and thirty, 195 between thirty and forty, 69 between forty and fifty, and 30 above fifty.

On the whole, we must confess that the conditions of rheumatism are obscure. Practically all that can be said is that those who are young, those who are poor, and those who have suffered from it already, are the most likely to be attacked by the disease.

Pathology.—The pathological allies of rheumatic fever are Chorea and Erythema. Difficult as it may be to distinguish it in certain cases from gout, or osteo-arthritis, or even from gonorrhœal synovitis or pyæmia, this is no evidence of the diseases being related, any more than the same or greater difficulty of diagnosis in cases of fracture or dislocation, tubercle or typhoid fever, intestinal obstruction or peritonitis. As for mixed or hybrid forms of disease, not only is their existence unproved, but the notion of a hybrid between two diseases is equally absurd, whether we regard them as due to a chemical poison, or a spore-bearing microphyte, or as an abstract expression for a constantly recurring combination of symptoms.

The essential nature of the disease is quite unknown. It has been ascribed with equal confidence to a destillation of diseased humours, to a disturbance of nervous centres or of trophic nerves, to the presence of a bacillus, and to an organic poison in the blood.

Dr Fagge, like other physicians, was attracted to the view, originally suggested by Prout, that the poison of rheumatic fever is lactic acid. Analogy, a fallacious guide, argues that rheumatism, like gout, may be due to some acid in the blood; but, in spite of its peculiar sour smell, we have seen that the sweat of rheumatism is not more acid than usual. In 1853 Dr B. W. Richardson published a series of experiments upon cats, which were believed by him to show that the injection of lactic acid into the peritoneal cavity was capable of setting up endocarditis; but eight years later Reyher, in 'Virchow's Archiv,' pointed out that appearances precisely similar are constantly seen in the cardiac valves of healthy cats. After this the lactic acid theory languished, until, in 1871, Dr Balthazar Foster, of Birmingham, recorded in the 'British Medical Journal' two cases in each of which the administration of this acid (in doses of ♏xv to ♏lxxv), with a view to check diabetes, was followed by the occurrence of painful swellings of the joints like acute rheumatism. Külz, in his 'Beiträge zur Path. u. Ther. d. Diabetes,' related a case in which lactic acid set up pains called "rheumatic" in the hip and thigh. But the same drug has been repeatedly administered to other patients and to healthy persons without result. Lastly, a plausible hypothesis is that under the influence of cold the lactic acid which is always formed as the result of muscular exertion fails to be destroyed by oxidation, as it should be, and that when so accumulated it acts as an irritant to the joints. Until better evidence than imperfect observation, hypothetical chemistry, and vague conjecture is given, we may defer acceptance of the last of the humoral theories.*

It has often been suggested that rheumatism is a *specific fever*, and within the last few years the discovery of its infective microbe has been repeatedly announced. But none of the criteria stated above (p. 18) have been satisfied; and, apart from the evidence of the laboratory, the natural history of the disease is strikingly unlike that of a specific fever. It is not contagious, it does not run a definite course, and it does not protect against future attacks.†

A relation between *malaria* and rheumatism was a pardonable guess at the beginning of the present century, but there is not the least evidence in its favour as a serious hypothesis.

Treatment.—Formerly the treatment of rheumatism was not satisfactory. A variety of medicines were prescribed, each of which appeared to be highly successful in some cases, while it utterly failed in others. Some physicians, among them the late Dr Fuller, of St George's Hospital, recommended the administration of alkalies, in such doses as to maintain an alkaline reaction of the urine; others, following Dr Rees and Prof. Lebert, trusted to lemon-juice. Some gave bark (as Haygarth recommended), or quinine, others the tincture of iron, others colchicum, others ergot, others propylamine (or rather trimethylamine). The late Dr Herbert Davies advocated the employment of blisters to all the affected joints; surrounding the knees, for example, with strips of emplastrum cantharidis three inches wide. It seems to be certain that the blistering plan is often followed by a rapid subsidence of pain, but there is the disadvantage that when carried out fully it is apt to produce strangury, and (as Senator has shown in vol. lx of 'Virchow's Archiv') it sometimes leads to inflammatory exudation in the urine, which may not only contain flakes when voided, but even coagulate afterwards. Moreover it may produce local sloughing; and, after all, the local synovitis is not the disease.

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* As ingenious hypotheses on the chemical origin of rheumatism should be mentioned Dr Latham's 'Croonian Lectures' of 1886, and Dr Haig's more recent papers. The inhalation of sewer gas as a cause of rheumatic fever has been put forward by Dr B. N. Dalton, in a practical paper in the 'British Medical Journal' for 1890.

† For all that can be at present brought forward in favour of rheumatic fever being an infective specific disease the reader is referred to Dr Newsholme's interesting Milroy Lectures for 1895 ('Lancet'). Achalme in 1891 and Thiroloix in 1897 ('Comptes rendus de la Soc. de Biol.,' March 19th and October 15th, 1897) have described probably the same anaërobic bacillus as present in the blood, in the joints, and in the serous effusion of a rheumatic pleurisy. Injected into rabbits it produced effusion in the joints, pleura, and pericardium. Sahli, Choostek, and other good observers have worked in the same direction, but no conclusive results have been attained.

Certain physicians always suspected that the apparent success of various methods of curing rheumatism might, after all, be fallacious, and that the result would have been the same if nothing had been done. Sir Thomas Watson used to cite, but without assenting to it, the dictum of Dr Warren, who, when asked what was good for rheumatic fever, replied, "Six weeks." Since then careful observations have been systematically recorded of cases left without active medication. Such an "expectant treatment," as it has been termed, seems to have been first tried by Lebert, who in 1860 published the results in nine patients. A few years later Sutton published in the 'Guy's Hospital Reports' for 1866 the details of twenty-five cases, treated by Sir William Gull.

It is wrong to consider the "expectant" treatment carried out by Gull as doing nothing. It was the same rational treatment used in cases of neumonia, of enteric or scarlet fever, and of fractured limbs. The patients were kept perfectly undisturbed, no strangers were allowed to approach heir beds, they were screened from draughts, and the light was shaded. Their joints were covered with cotton wool over lint dipped in laudanum, and were protected from pressure by cradles. Except under very special circumstances no laxative was allowed. They were given an effervescing julep, or any cooling drink preferred, and a grain of opium was administered every night, or oftener if the pain was severe. Under this treatment, if there were few brilliant recoveries, there was on the whole far less pain and distress, a shorter average course, less cardiac disturbance, and a more safe and rapid convalescence than under the antiphlogistic treatment of Bouillaud at La Charité, the lemon-juice of Rees, the alkaline treatment of Fuller (each of which the writer had the opportunity of comparing with Dr Gull's), or any other plan which was in use before the introduction of salicin.

These observations proved incidentally that rheumatism, unlike the specific fevers, has no fixed duration. The length of time during which active symptoms continued, including that passed before admission as well as subsequently, varied from nine to thirty-four days. On striking an average, the course of the disease, as measured by the period at which there is freedom both from pyrexia and from pain, is much shorter than that named by Dr Warren. Gull and Sutton made it nineteen days; Lebert gave sixteen days as the average time for marked improvement, twenty-two days as that for convalescence.

"In attempting to compare cases of acute rheumatism left to themselves with those submitted to treatment by drugs, there is a fundamental difficulty. Strictly speaking, those cases only are comparable in which the treatment was begun on the very day on which the patient was aken ill. But to include only such cases would be not only to limit the eld of observation almost entirely to private practice, but also to ensure at the cases accepted should be of far more than average severity, since ly patients seized with severe symptoms are likely to seek medical advice once. It is therefore impossible to reject cases which have come under eatment at varying periods of the disease. But now arises the question whether one should take into account the time which the disease has lasted before the patient is seen. It is clear that if treatment is effective the fact must be brought out most strikingly when this time before treatment is left out of consideration; on the other hand, if it was ineffectual, we should take the whole duration of the cases as a basis for comparison.

"It is easy to prepare both sets of figures, as was done by Gull and Sutton; and in dealing with average results, no serious error is likely to arise whichever set be adopted—provided the cases are numerous, so that each group may contain its proportion of patients brought under treatment at an early stage, and of those in whom the disease had lasted several days.

"But there remains another objection to the employment of averages, which invalidates the comparisons made by Gull and Sutton of the results obtained by the 'expectant' with those by other methods. The objection is that some cases of rheumatic fever run a protracted course, owing to frequent relapses, and instead of terminating in a few weeks, last for months. One cannot be surprised that no such cases appear in Gull's and Sutton's lists; for it would be impossible to keep the patients week after week under observation without attempting something for their relief; and afterwards, in tabulating results, the fact of treatment having been adopted would be sufficient to exclude them. But when one is dealing with a series of cases submitted to any particular treatment, it becomes impossible to reject the cases in question; and when an average is taken, a few such cases would swamp the rest in which treatment may have been successful.

"It appears that the only fair way of using Gull's and Sutton's cases is to tabulate the length of time that symptoms lasted after admission: the list is then as follows, a fatal case being excluded."—(C. H. F.)

TABLE I.—*Natural Course of Twenty-four Cases.*

Duration of Symptoms while in Hospital.	Number of Cases.	Duration of Symptoms before Admission.	Total days' illness.
3 days	1	21 days	24
4 days	1	5 days	9
5 days	1	Uncertain	—
6 days	2	7 days, 5 days (average 6)	12
7 days	3	3 days, 3 days, 14 days (average 6·3)	13
8 days	1	5 days	13
9 days	1	9 days	18
10 days	2	8 days, 5 days (average 6·5)	16·5
11 days	3	8 days, 5 days, 5 days (average 6)	17
12 days	2	6 days, 10 days (average 8)	20
13 days	1	6 days	19
14 days	1	14 days	28
16 days	2	12 days, 6 days (average 9)	25
18 days	1	12 days	30
21 days	1	4 days	25
27 days	1	7 days	34

Duration before admission of 23 cases in which it could be determined—180 days; giving an average of 7·8 days.

If we now compare the natural course of the disease with the results of three different methods of treating rheumatism, as recorded by their several advocates (see Table II), it seems clear that no striking success can be claimed for any of these three plans of treatment.

TABLE II.—*Results of Treatment by Lemon-juice, Alkalies, and Blisters.*

Duration of Symptoms after Commencement of Treatment.	Lemon-juice. Dr Owen Rees ('Guy's Hosp. Rep.,' xii).	Bicarbonate of Potass. Dr Garrod ('Med.-Chir. Trans.,' xxxviii).	Free Blistering. Dr Davies ('Lond. Hosp. Rep.,' i).
1 day	1	—	—
2 days	—	1	—
3 days	—	3	—
4 days	1	8	—
5 days	—	6	1
6 days	1	6	2
7 days	2	7	1
8 days	—	5	2
9 days	1	9	1
10 days	2	—	—
11 days	—	2	—
12 days	—	4	—
13 days	—	—	1
14 days	1	Doubtful	5
Total 73 cases	9 cases	51 cases	13 cases

Salicin.—Happily, it has now been conclusively shown that the duration of rheumatism may be shortened, and its pain quickly relieved, by the administration of salicylic acid or its alkaline salts or salicin. It was in January, 1876, that Dr Stricker, assistant in Traube's Clinic in Berlin, drew attention to the use of salicylic acid in rheumatism. It had been used there for some months, and the same medicine had been employed at Basle by Buss. Salicin was originally advocated by Dr T. J. Maclagan, in March, 1876, in the 'Lancet.' *

After a period of reasonable scepticism† and trial, the salicyl treatment became firmly established in this country and in Germany, and was introduced into France by Professor Sée. Almost all physicians in Europe and America now use it.

Salicylic acid, as originally used in Germany, is an unsuitable form of the remedy. It is insoluble, bulky, unpleasant, and irritating. It is now always neutralised by ammonia, potash, or soda, and the soda salt is the one generally used. It is probably less depressing to the heart than salicylate of potass, but it is often well to add sp. ammoniæ co. or alcohol so as to obviate this result. Salicin is an agreeable remedy, and is now scarcely, if at all, more expensive than the salicylates. It is also less irritating to the stomach and bowels, and milder in its general effects. But it sometimes fails when other salicyl compounds succeed, and therefore is best used as a substitute for sodium salicylate.

Dr Fagge took the pains to tabulate not only his own experience at Guy's Hospital, but also that of all his colleagues, in the treatment of acute rheumatism with salicylin or salicylates from the spring of 1876 to the end of 1880, and obtained results which compared very favourably with those given in either of the above tables. The symptoms were arrested within five days in no fewer than 180 of 355 patients, at which period only three of twenty-four patients had lost their symptoms. In many cases the patient within two days was almost free both from pyrexia and from pain. It is no uncommon thing for the patient to be conscious of experiencing great relief from the first two or three doses of the medicine; and house physicians used to remark the striking contrast, even on the first night after admission, between patients treated with salicylates and those to whom no medicine had been administered; the former lay quiet, even if they did not sleep; the latter cried out during the whole night.

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* Among the Hottentots and the Boers of South Africa, willow tea has, Mr F. Ensor informs me, long been a traditional remedy for rheumatism.—C. H. F.

† A criticism on the salicylic treatment of rheumatism, by the late Dr Greenhow, in 1880 ('Clin. Trans.,' xiii, p. 244), based on his experience of 61 cases, was carefully considered and discussed by Dr Fagge in the first edition of this book. It is remarkable that Dr Osler in his admirable treatise will only admit the efficacy of salicylates in relieving pain (3rd ed., p. 174).

Among other statistics of the effect of salicylates on acute rheumatism, the reader is referred to an elaborate analysis of 210 cases at St. George's Hospital by Dr Isambard Owen ('Lancet,' 1881), to the 158 cases reported in the 'Lancet' for 1879 by Mr R. H. Lucas, of Bury St Edmunds, and to the results of 536 cases reported in the 'Brit. Med. Journ.' for 1888, vol. i, p. 395. See also Dr Bristowe's paper read at Cardiff in 1885, and the discussion which followed ('Brit. Med. Journ.,' August 22nd). Sir Hermann Weber's early paper in the 'Clinical Transactions' for 1877 is of interest from its account of the introduction and tentative use of the new remedy (vol. x, p. 63).

The interest of these statistics has, however, for the most part passed away. For the results of salicylic treatment are notorious. They have made the description of rheumatism given on pp. 485-6 ancient history, and prevented the diagnosis of rheumatism at sight except the patient is seen before treatment has begun.

Effect on relapses.—In ninety-three of the above 355 cases relapses occurred. In one case a relapse lasted twenty-one days; but the average duration of thirty-one relapses was only between five and six days.

Now there are two ways of looking at the relapses of acute rheumatism. One is to regard them as continuations of the original illness, and to suppose that it has been interrupted and postponed, but not really cut short by the administration of the remedy. This view is supported by cases in which the symptoms return very soon after the discontinuance of treatment, sometimes within twenty-four hours. In one case the urine was tested with perchloride of iron for several days after the salicylate of soda had been left off, and the purple reaction indicative of the presence of a salicyl compound was obtained as late as the eighth day; so that relapse after a week may be explained as recrudescence of the primary attack. But, on the other hand, when the relapse begins three weeks or two months after the subsidence of the primary attack, and when salicin had long been discontinued, the case must be looked at in the same light as those which relapse without any treatment. There is no reason to suppose that relapses are more apt to occur when salicin or salicylate of soda has been given than when the disease was treated in other ways, or left to run its course. On the other hand, it is probable that the frequency of relapses is greatly diminished by continuing the administration of moderate doses of salicylate, or of salicin, until after the lapse of several weeks, and by prohibiting meat, and even fish or cheese, for as long as the patient will submit.

Doses.—The dose of salicylate of soda which is adequate to arrest acute rheumatism with rapidity is, as a rule, about twenty grains, given at inter-als of two or three hours; but sometimes a larger quantity is required. One patient of Dr Fagge's took twenty grains of salicylate of soda every two hours without marked result for two or three days, but the disease at once yielded when thirty grains were given. On the other hand, there are cases in which a dose of ten grains, repeated every six hours, proves effectual.

Salicin should be prescribed in larger quantities; Dr Maclagan recom-chloroformi, or in aqua carui. Or the acid may be dissolved in solution of citrate or of acetate of ammonia (the proportion being gr. xx of acid to ℥iij of liq. amm. acet.), and sweetened with extract of liquorice.

Salicin should be prescribed in larger quantities; Dr Maclagan recommends that from twenty to forty grains should at first be given every hour. It may be taken as a powder stirred up in cold water, or twenty grains may be dissolved in an ounce of warm water.

Influence on the heart.—Whether the salicylic treatment tends to prevent cardiac complications in rheumatism, it is very difficult to say. One cannot forget that of each new method of treatment—even that by local blistering*—it was asserted that the liability to cardiac inflammation was thereby lessened; and Gull and Sutton showed that when the heart is healthy at the time of the admission of the patients into hospital, it seldom becomes subsequently attacked.

Still, one may reasonably expect that any remedy which can arrest rheumatism, so that fresh joints no longer become affected, would hinder the development of inflammation in the heart. In sixty-nine of Dr Fagge's 355 cases, auscultation revealed some changes in the cardiac sounds while the patient was in the hospital, but there was hardly one in which there was reason to believe that pericarditis set in at a time when the action of the remedy was established.

Dr Church, comparing his cases under salicylic treatment with those of the late Dr Latham at the same hospital, long before its introduction, believes that the liability to endocarditis is much the same, but that pericarditis is less frequent, and pleurisy and pneumonia far less common and far less severe. That pericarditis is much less frequent than formerly, while endocarditis is not, is what the present writer has above stated as his experience; and, if so, it seems reasonable to refer the change to improved treatment.

In any case, it must, unfortunately, be admitted that the salicylic treatment has no power of arresting the cardiac complications of rheumatism when once present.

Drawbacks.—In certain cases the administration of salicylates is attended with inconveniences, and sometimes with alarming symptoms. An occasional result is nausea and vomiting, with epigastric pain.

A less rare effect is cardiac depression. In a few instances Dr Fagge noted that the pulse became weak, irregular, or intermittent: in one case it fell, after nine days, to fifty-two beats in the minute; in two the first sound of the heart became inaudible, and the heart's impulse could no longer be felt. Dr Greenhow ('Clin. Trans.,' 1880) observed after salicylic treatment" that "more or less weakening of the pulse, requiring the free administration of brandy, occurred in nearly every case. This was accompanied by great weakening of the impulse of the heart, and in ten cases by almost complete obliteration of the first sound." Dr Goodhart recorded a case in the same volume in which sudden death in the night took place, probably as the result of syncope, for the pulse had been rising in frequency. He was disposed to attribute this result to salicylic acid; but only sixty grains in all had been given, and none of the known effects of the drug were observed. At the autopsy, moreover, early pericarditis was found.

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* Dr Caton advocates the systematic application of small blisters over the anterior distribution of the intercostal nerves as a prophylactic against cardiac complications.

Cerebral symptoms sometimes follow the exhibition of salicylates—but very rarely of salicin. Deafness is a frequent and early effect of the remedy, accompanied with giddiness and buzzing or ringing in the ears, sometimes with headache or delirium.*

Another occasional effect, whether of salicin or of salicylic acid, is *epistaxis*. It was particularly noticed by Dr Greenhow, and has occurred in many cases at Guy's Hospital. It often recurs several times, but is not attended with evil consequences, except that it must tend to increase anæmia.

Dr L. E. Shaw recorded in the 'Guy's Hospital Reports' (vol. xliv, p. 125) three cases of severe *hæmorrhage* occurring during the administration of salicylate of soda for acute rheumatism. In the first patient, a boy aged fifteen, taking a scruple of the drug every three hours, delirium appeared on the fourth day (eleventh of the disease), with epistaxis and retinal hæmorrhage. He recovered, but the other two cases proved fatal. In one, a wardmaid aged twenty-one, after taking the same dose of salicylate every two hours, became delirious and passed blood in her urine. After death there was no cardiac lesion found, and the kidneys were normal, but the renal pelvis and the bladder were covered by ecchymoses. In the third patient, a woman aged twenty-six, taking the same 20-grain dose every three hours, for a severe febrile attack of doubtful nature, delirium and hæmaturia appeared, and after death, beside lesions due to latent enteric fever, a precisely similar condition of kidneys and bladder was found.

Taking all the cases of rheumatic fever which occurred in our wards i two years (1881 and 1886) as specimens, Dr Shaw found that in these 17 cases, all treated by salicylate of soda, except a few by salicin, and in near the same doses, there were more or less toxic effects in no fewer than 11 Of these, deafness was present in sixty-one cases, delirium in thirty-three headache, vomiting, and singing in the ears in about the same number, slow or irregular pulse in thirteen, epistaxis in eleven, and hæmaturia in th two cases above quoted.

There is reason to believe that some of these symptoms are due not salicylates, but to the phenol (C_6H_5OH) from which the acid (HC_7H_5O is derived. The first time in which the writer gave the new drug it w attended by such severe vomiting, purging, and depression that he gave up for several months; but such a result has not happened for many year

Some have supposed that after the salicylic treatment the patie regains health and strength more slowly than if the disease had been left to run its natural course. We may admit that the stay in hospital of cases treated with salicylic acid is little shorter than it used to be before the remedy was used. But long after active symptoms have ceased, one keeps the patient on low diet and confines him strictly to bed, for fear of a relapse.

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* It often happens that, when such effects are produced, the patient has already k his pains, and that his temperature has fallen to 99° or to 100°; the latter symptom d tinguishes the cerebral symptoms due to salicylic acid from those of hyperpyrexia. T face may be deeply flushed and bathed in perspiration, and in one case the delirium reported to have been attended with "typhoid symptoms, so that the prognosis for a ti was grave." As a rule the patient becomes rational in a few hours, or in a day or tw after the medicine is discontinued. A fatal result has very rarely followed, and in the exceptional cases we have usually found some other cause after death. The drug has oft been resumed after a few days' interval without further ill-effects; but sometimes deliriu has set in on successive occasions.—C. H. F.

Treatment of hyperpyrexia.—The discovery that very high temperature is the real cause of dangerous cerebral symptoms in rheumatism was soon followed by the employment of active antipyretic treatment. The administration of salicylic acid or of quinine fails in serious cases; and the newer drugs, as phenacetin and antipyrin (cf. pp. 153-4), are too transient in their effects and too depressing in their action on the heart to be either efficient or safe. The only satisfactory treatment is by the direct application of cold.

The first instance in which this treatment brought about recovery from hyperpyrexia in rheumatism seems to have been recorded by Dr Meding in the 'Arch. f. Heilkunde' for 1870; the temperature was 108·6°; the means employed were cold affusion and enemata of iced water. In the following year Dr Wilson Fox published two important cases ('Lancet,' 1871, vol. i, pp. 213 *et seqq.*).

The first occurred in a woman aged forty-nine, who was in the fourteenth day of her illness, and who had been five days in University College Hospital, when her temperature began to rise quickly. At 3 p.m. it was 105°; at 6, 106·4°; at 8.5, 107·1°; at 9.15, 108·4°; at 9.50, 109·1°. She was then completely unconscious, her pulse was imperceptible, her face cyanotic, and she appeared to be drawing the few last gasping respirations which precede death. There had been delay in preparing a bath, into which, at a temperature of 96°, Dr Fox had intended to put her when her temperature reached 107°. However, she was lifted into it at 9.50, and five minutes later the temperature in the rectum was found to be 110°. With admirable decision, Dr Fox sent for some ice; two large lumps were placed, one on her chest, and the other on her abdomen; a bag filled with ice was tied down the length of her spine; two assistants baled the warm water out of the bath, and two others poured iced water over her as fast as the pails could be filled. The temperature in the rectum gradually fell until at 10.25 it was 106·2°. The pulse now became perceptible, and slight signs of consciousness were manifested. At 10.35 the temperature in the rectum was 103·6°, and she was removed from the bath. At 10.55 the temperature in the rectum was 100·6°, and she was able to speak. The bath had to be repeated on the following morning, but she finally recovered.

Dr Fox's second case was that of a man aged thirty-six, in whom on the sixth day after his admission (the seventeenth of his disease) the temperature rose to 107°, having before been always below 104·5°. He showed signs of pericardial effusion, and also of inflammation at the bases of both lungs; he coughed and expectorated thin mucus stained with blood. This did not prevent Dr Fox from having him placed in a bath at 89° for twenty-five minutes, during which time it was cooled down to 66°. The rectal temperature fell from 107·3° to 103·1°; and after removal from the bath became normal. He ultimately got well, after eight baths in all.

Since Dr Fox recorded his cases, the treatment of hyperpyrexia by cold has been constantly followed by satisfactory results. A striking instance occurred in one of the pupils of Guy's Hospital, who in 1875 had a severe attack of acute rheumatism, during the course of which his temperature on twenty-six occasions, from the ninth to the twenty-fifth day of March, rose to a point between 105° and 107·2°, and was each time brought down by immersion in an iced bath. He recovered completely. Full particulars of this case may be found in a paper in the 'Liverpool Medical Reports' for 1876, by Dr F. T. Paul, who was house physician at the time, and who carried out the treatment ordered with unwearied patience and determination.

Unfortunately, even when the bath is successful in lowering the patient's temperature, it does not always save his life.

Dr Paul records the case of a man aged thirty-two, one of the porters in Guy's Hospital, who after a week's illness with rheumatic fever became extremely delirious and then comatose, and was found at 9 p.m. with a temperature of 108·8° in the axilla. As he lived out of the hospital, there was a delay of at least an hour before a bath could be procured. When he was put into it his temperature was 110·9°, and he was violently purged. The bath was at 90°, and he was kept in it for thirty-five minutes, during which time it was reduced to 66° by cold water. His temperature on removal was 106·6°; he was still perfectly insensible, with contracted pupils and with noisy and rapid breathing. Half an hour later the temperature in the rectum was 101·3°, and an hour after the bath it was 99·3°. Subsequently it rose slightly, but it never reached 103°. He died in the afternoon of the following day, the only change being that the contracted state of the pupils changed to wide dilatation.

Another instance, which may also be found in Dr Paul's paper, is that of a woman who died after having had twelve baths during a period of nine days. For the last two or three days mucous râles were audible widely over the chest; but at the autopsy there was only a little broncho-pneumonia at the bases of the lungs, and some mucus in the tubes. We have had five other cases at Guy's Hospital, which ended fatally, notwithstanding that the hyperpyrexia had been overcome by baths; in only one of them did the *post-mortem* examination reveal an adequate cause of death in severe pleurisy with pericarditis. Such patients seem generally to sink by failure of the circulation. Indeed, in both of Dr Wilson Fox's successful cases it was deemed necessary to give large quantities of brandy after the baths, and also to apply hot bottles to the feet and warmth to the back; his first patient took six ounces of brandy within an hour.

The best method of averting collapse after hyperpyrexia is to have recourse to a bath early, before the heart has been too much damaged by the heat. When the bath is delayed there is always some risk of death during immersion, a mishap which occurred at Guy's Hospital, once in 1874, and again in 1877. As soon as the temperature is rising to a dangerous height—say above 105°—the bath should be used. The bath should be repeated as often as the temperature rises to 105·5°. It is best to let the water have a temperature of 90° at the time when the patient is immersed: if it be much colder than this he is likely to shiver and complain; whereas when it is from 90° to 100° he often finds it pleasant, so that he will afterwards beg to have the bath repeated. He should be lowered into the water upon a sheet. As soon as this has been done the temperature of the bath should be reduced to 75° or 70°, by the addition of ice, which is more convenient than cold water because it occupies less space, so that no baling out is required. The patient should not be left in the water after the temperature in his rectum marks 102°, for it will continue to fall after his removal. When he has been lifted back upon the bed, he should be lightly covered with a blanket and allowed to sleep.

The milder methods of employing cold, which were described in the treatment of enteric fever, sponging, wet packing, &c., are insufficient to meet the unusual danger of excessive temperature in rheumatism. The only efficient substitute for the cold bath, if circumstances make its administration impossible, is rubbing the patient's body with ice.

Treatment of cardiac inflammation.—A most important question is whether the salicylic or any other treatment in rheumatism prevents the implication of the pericardium, the endocardium, and the muscle of the heart, which we chiefly dread for our patients, or checks it when it has taken place. To the latter half of the question we must reply with a disappointed negative. To the former the answer is less clear. While we must admit that there is no conclusive evidence to prove that fewer of our patients under salicylic treatment have the heart affected than when they were treated with rest and opium by Gull, it seems probable that the shortening of the active stage of the disease does diminish the chance of cardiac complications, for most cardiac murmurs occur while there are fever and synovitis, and few after they have subsided.

With respect to pericarditis, if the impression above stated is correct, that it is far less common than formerly, there seems nothing to which this can be ascribed except the new method of treatment. So that the writer is inclined to believe that salicylates do decidedly diminish the chance of pericarditis, while they have only a limited effect in lessening the number of cases of valvular disease.

Dr Caton, of Liverpool, has lately advised treating early rheumatic endocarditis by flying blisters to the præcordial region and very prolonged confinement to bed. The clinical facts by which he supports his method of treatment make it worthy of further trial.

Treatment during convalescence.—The treatment of rheumatism, apart from complications, is much modified by the good effect of salicyl. Relief is so soon obtained that in most cases we need only guard our patient from a relapse.

With this object, our duty is, first, to keep the patient in bed for many days after the fever has left him and his joints are free from pain; and secondly, to continue feeding him on strictly low diet, and only when his temperature is normal on farinaceous food. Many physicians believe beef-tea to be injurious, and undoubtedly no meat nor even fish should be allowed for a fortnight after convalescence is established. Nor should the patient be on any account allowed to leave his bed until the same period has elapsed. This rigorous system is often difficult to carry out, but one has rarely been tempted to relax it by the entreaties of the patient without regretting one's compliance. Moreover, in order to prevent a relapse, salicylates in less frequent doses should be continued for three weeks or even longer. When convalescence is well established, and before the specific drug is left off, it is usually well to administer tincture of steel to combat the anæmia which usually follows rheumatism, but it is probably injurious if given before a week or ten days of freedom from pain and fever have elapsed.

The only exception to the above rules which should be allowed is in the case of a patient who has suffered repeatedly from the disease, and whose heart is already damaged beyond hope of repair. The pyrexia in such cases is often moderate, and the pain not severe; but relapses are frequent, and the cardiac symptoms are more important than those of the rheumatism. For these patients strong soups, eggs, and wine or brandy are useful, while steel and digitalis may be given with benefit to the heart and with no apparent ill-effect on the rheumatism.

CHRONIC RHEUMATISM.—Although this phrase is in common use for a very frequent disorder, there is reason to doubt whether rheumatism, in the only exact sense of the term, is ever a chronic disease.

Formerly any ache or pain was "explained" as a result of a *destillati* or *catarrh* of cold, moist, peccant humours, and such a *rheum* might affe one part as much as another. In France it appears that medical usage i the seventeenth and eighteenth centuries applied the term *rhumatis* particularly to pains in the limbs rather than in the trunk or head, an in the bones and muscles rather than in the joints, while the nomenclatu of the classical writers was preserved in calling all particular pains—*i. e.* a cases of arthritis—*podagra* or gout. In England, since Sydenham intr duced the term rheumatism into medical literature, it has been chiefl restricted to inflammation of the joints, although we have seen with ho little precision the word was used.

At the present time, in all schools of medicine, rheumatism is recognised, in its most characteristic and typical form, as the acute febrile disease which has been described in the preceding pages. It is very desirable to restrict the use of the word to this, its only exact and definite sense. By "rheumatism" we should always mean the disease which appears as acute multiple synovitis with fever, and the epithet "rheumatic" should refer to this disease alone.

Chronic rheumatism, then, ought to mean a chronic arthritis of the same pathology as the acute outbreaks of rheumatic fever. Such a disease, we may affirm, does not exist.

Occasionally, as above stated, attacks of rheumatism relapse and recur so often that the intervals are shorter than the paroxysms, while the severity as well as the duration of the latter become less and less. Such successive subacute attacks are most often seen in young patients, and accompanied by cardiac lesions; they are often rebellious to treatment by salicylates, and are more benefited by opiates and by steel. But their origin is always in an acute attack; there is always more or less febrile movement, while the age of the patient, the aspect of the case, and, above all, the cardiac implication, show that the case is one of genuine rheumatism.

Occasionally when rheumatic fever has occurred frequently and severely, and particularly when the arthritis has lingered longer than usual in a particular joint, there may ensue *hydrops articuli*, and this will run a more or less chronic course. The articulations most liable to this sequel of rheumatic fever are the knee and less frequently the wrist. More rarely, under the same conditions, some distortion of the affected joint may result. This is most likely to happen after a long succession of subacute attacks as above described, and the fingers are the parts most likely to suffer.

Lastly, there is no reasonable doubt that some cases of the disease, to be described in a subsequent chapter as osteo-arthritis, originate in unusually severe or unusually protracted synovitis of genuine rheumatic character. According to observations quoted by Dr Archibald Garrod, some of these cases are found after death without the anatomical changes characteristic of osteo-arthritis. If so, notwithstanding some deformity and muscular atrophy, such cases should probably rank as rheumati rather than osteo-arthritic. But however they may end—and all thes chronic affections are rare and exceptional, not ordinary and freque results of rheumatic fever—each of them begins with a typical acute atta of genuine rheumatism, and never as a chronic, non-febrile, and ingra cent disorder.

Myalgia.—"Muscular rheumatism" has been considered a variety of "chronic rheumatism," but here there is no articular inflammation at all. The disease, *i. e.* the pain—for there is no other symptom—is in the muscles. At one time this was, as we have seen, the type of "rheumatism." Then the patient's feelings and the physician's hypotheses made up the whole of pathology. But now that the Galenical or humoral theory is exploded, the disorder in question, having no morbid anatomy, no ascertained chemistry, and no decided ætiology or clinical alliances, must find a new name; and that of *myalgia* is distinctive, convenient, and affirms nothing beyond the fact of the pain and its seat.

Myalgia has nothing in common with true rheumatism. We do not know whether its exact seat is in the muscular fibres themselves or in the perimysium and fibrous sheaths. Apparently it is not localised in tendons or in ligaments, for the pain is more often present in the thick fleshy masses of the neck and loins than in the tendinous muscles of the limbs. Movement always aggravates the pain, but if persevered in will frequently prove the best remedy.

The so-called "growing pains" of children are probably not muscular; they are often situated in the shins, and sometimes in the shoulder. Sometimes they are really articular and rheumatic; sometimes they are symptoms of tuberculous or syphilitic disease of the bones or periosteum; and often they seem to be symptoms of anæmia, and are quickly removed by the administration of iron.

Lumbago or pain in the loins occurs as a symptom of gravel and stone in the kidney; in its severest degree it is an early symptom of smallpox and other fevers; in a less acute form it is a common result of muscular fatigue, particularly from long standing, from long or hard riding, and from long sitting, as during a night's journey in a diligence or a railway carriage. This is the kind of backache with which some hard-worked people wake of a morning.

In women it is a frequent sign, not only of dysmenorrhœa and menorrhagia, but of uterine disorders unattended with hæmorrhage. In these cases, however, its characteristic seat is not so much the loins as the sacral region.

The lumbago which is only called forth by movement is often called "a stiff back," but the stiffness is not from mechanical immobility; it only depends on the patient's instinctive avoidance of what causes pain. It is sometimes produced by unusual exercise—a first day's shooting gives it next morning in the shoulder, and a first day's hunting in the loins. It is then the direct effect of unwonted muscular contraction, like the pain which follows prolonged movement of any kind. The erector spinæ, which has the most constant work to perform, suffers most.

Sometimes myalgia of the loins or of the shoulders follows definite exposure to wet, and this is the nearest approach in causation to true rheumatism. More often it can be clearly associated with thick and acid urine. But a large number of cases of lumbago are without ascertainable cause, come on suddenly, and as suddenly pass away. They are like the painful contraction of muscles known as cramp, and, as in a severe case lately under the writer's notice, may be accompanied with evident contraction amounting to slight tonic opisthotonos.

In the majority of cases lumbago is a disorder of later life, and (if distinguished from sacral pain) is much more common in men than in women. It is common in persons who suffer from gout, but perhaps not more so than in other men over forty.

Myalgia of the nuchal muscles, or a stiff neck, is usually ascribed to "cold" and to a draught rather than a chill from rain. Similar pains about the muscles of the scapula are more often associated with dyspepsia when not the result of unaccustomed exercise. The fleshy masses of the glutei and the calf are seldom the seat of continuous aching pain. In the former situation pain is usually unilateral, and depends on sciatica or hip disease; in the latter it is more often spasmodic, and due to the irregular contractions of cramp.

Mr Hutchinson, in the 'Archives of Surgery' for 1887, argues that lumbago is nothing but inflammation of the sacro-iliac joint. But surely the latter is a well-recognised disease, with its definite signs and course, much more local and more severe than lumbago; it is not bilateral, it is common in children, it does not recur, and it does not come or go with the suddenness of true lumbago. Is it not more accurate to put the facts thus—that sacro-iliac disease, like morbus coxæ, pelvic abscess, or aneurysm, nephritis and gravel—is one of the causes of pain in the loins which simulate lumbar myalgia, and must be carefully distinguished from it? Whether we use the term lumbago for the symptom or the at present "idiopathic" disease is not very important. As with "epilepsy," "eczema," and "neuralgia," the better plan seems to be to reserve those terms for the primary or "simple" cases, when the secondary symptomatic cases have been excluded.

The only natural clinical ally of lumbar myalgia is sciatica; how far there is any pathological relation between the two conditions will be discussed in the chapter on neuralgia.

The *treatment* of the various forms of myalgia is not satisfactory. Firm pressure, as with a hard pillow, gives relief from lumbago and sacral pain. A hot bath is an excellent remedy for general aching after the fatigue of a prolonged railway journey or a hard day's riding, or exposure to cold and damp; and many persons find a Turkish bath still more efficacious. Shampooing and hot ironing over flannel are extremely grateful in "myalgia" of the shoulders and in lumbago.

As local applications, mustard plasters, and friction with turpentine or hartshorn and oil (Lin. Camph. Co.), have not undeserved reputation. Equal parts of Lin. Belladonna and Chloroform liniments have often succeeded in the writer's hands when other remedies have failed; or a liniment made of one fluid ounce of Lin. Aconiti, two of Lin. Belladonnæ, and three of Lin. Chloroformæ. In one severe case of lumbago, the local use of methyl chloride on lint proved remarkably efficient in removing pain.

Internally, alkaline and diuretic medicines such as citrate, carbonate, and acetate of potash are generally indicated when the urine is thick with lithates. Chloride of ammonium in full doses is sometimes remarkably efficient; it probably acts like sodic chloride, nitre, and other neutral salts in favouring transudation. Guaiacum is a valuable medicine when the pains are relieved by warmth, and is always worth trying in cases of lumbago.

Whenever possible the myalgia should be traced to its source; and if there is no local cause, a knowledge of the patient's previous illnesses may occasionally lead one to use iodide of potassium, colchicum, salicylate of soda, or quinine with signal success. But this is treatment not of myalgia but of its cause.

GONORRHŒAL SYNOVITIS

> The gods are just, and of our pleasant vices
> Make whips to scourge us.
> *King Lear.*

Distinction from rheumatism—Sex and age—Relation to gonorrhœa—Distribution and local effects—Cases—Symptoms—sclerotitis and other complications—Prognosis—Diagnosis—Pathology—Treatment.

Synonyms.—Gonorrheal Rheumatism, Arthritis, or Syndesmitis Gonarthritis.—*Fr.* Rhumatisme Blennorrhagique.—*Germ.* Tripper-Rheumatismus, Trippergicht.

Definition.—A subacute or chronic inflammation of one or more joints, not suppurative nor liable to relapse, the result of infection with the gonococcus, and associated with sclerotitis, but not with cardiac or renal inflammation.

History.—Sir Astley Cooper, in his 'Lectures on Surgery,' published in 1824, mentions the case of a young American gentleman in whom gonorrhœa was followed by a painful disease of the joints and inflammation of the eyes.

This has since been commonly called "gonorrhœal rheumatism," but the present writer, in a paper in the 'Guy's Hospital Reports' for 1874, proposed to term it *gonorrhœal synovitis*. Whether this name or that of "gonorrhœal arthritis" be preferred,* we shall see that it differs in origin, course, prognosis, and treatment from true rheumatism as described in the preceding chapter; its name, therefore, should be no less distinct. The relation between the affection of the joints and that of the urethra is far too constant to be accidental. Moreover we shall see that the clinical course, concomitants, and sequelæ of the disease are quite characteristic, and different from those of any other articular inflammation, from gout, pyæmia, and osteo-arthritis, no less than from rheumatism.

Sex.—The present writer collected twenty-nine cases from the medical wards of Guy's Hospital in 1870-72; and during that time many others presented themselves in the out-patient room.

All these cases occurred in men, but he has since seen a few cases in women; and Mr Brodhurst, Mr Davies-Colley, and Dr Church have recorded others. Senator, in 'Ziemssen's Cyclopædia,' suggests that the toughness and thickness of the vaginal mucous membrane may afford an explanation of the rarity of gonorrhœal synovitis in the female sex. It would be interesting to know whether urethritis is *always* present when gonorrhœal synovitis occurs in women; in some it undoubtedly is.

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* The objection to "gonorrhœal arthritis" as a name is the unfortunate misuse of the word arthritis as a synonym for gout. "Gonarthritis" would, perhaps, be a possible makeshift.

Since the twenty-nine cases above mentioned were published, more than eighty others, sufficiently reported to be available, from our medical wards, were summarised for the present writer by Dr T. F. Rickets. Of these, seven occurred in women (two being open to some doubt), and seventy-six in men. Adding the twenty-nine earlier cases, we have a total of one hundred and five male and seven female patients.

Mr Burghard, now of King's College, then Surgical Registrar at Guy's Hospital, abstracted the reports of a considerable number of cases of articular disease combined with urethral or vaginal discharges, of which twenty-one are sufficiently complete to make it tolerably certain that they were examples of the disease, and not of accidental coincidence of gonorrhœa with inflammation of a single joint. Of these twenty-one surgical patients, seventeen were men and four women.

Age.—The above-mentioned 112 patients were, with few exceptions, in early adult life; 11 were between sixteen and twenty; 66, or more than half, were between twenty and thirty; 30 were between thirty and forty; 4 were between forty-one and forty-seven; and one was fifty-two.

Ætiology.—A urethral discharge was present in every case the writer has seen, but it was often not recognised until looked for to explain the synovitis, when a gleet was always found. The ætiological relation of the synovitis has been again and again proved by the patient having previously suffered from "rheumatism" after gonorrhœa, sometimes three and even four times. Among our 112 patients, eighteen gave a history of a similar painful affection of the joints having followed a previous attack of gonorrhœa; fourteen had twice, and one had three times suffered in the same way before.

Sir Benjamin Brodie recorded an instance in which, after two attacks of synovitis from gonorrhœa, two later ones were attributed to irritation of the urethral canal by the use of a bougie.

In fourteen out of 112 cases the patient had previously experienced what was called rheumatic fever; but in two of them this was pretty clearly the same complaint as that from which he was then suffering, and in all but one of these fourteen patients there was no sign of cardiac disease—an improbable exemption after rheumatism at an early age. With a disease so common as rheumatism in children and young adults, its occasional occurrence previously to gonorrhœal synovitis is probably a mere accident.

In a considerable number of cases there was a history of "rheumatism" or "gout" in the parents or brothers and sisters of the patient. In thirteen they were "rheumatic" or "subject to rheumatism," in ten the father was "gouty," or had "rheumatic gout," and in seven cases a near relation had suffered from "rheumatic fever" or "acute rheumatism." Whether this shows any predisposition to inflammation of the joints generally—an "arthritic diathesis"—is very dubious.*

The length of time which elapses between the commencement of the gonorrhœa and the development of the synovitis may vary from three or four days to six months, but most often falls between one week and three months. Sometimes the urethral discharge continues to be profuse after the appearance of the synovitis, but in most instances there remains only a slight gleet. Indeed, the patient often denies the presence of any urethral discharge.

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* According to Mr Brodhurst, exposure to cold and wet may be an exciting cause. He mentions the case of an officer serving in a tropical climate, who, while suffering from gonorrhœa, slept out of doors until after sunset, and awoke in such pain that he could with difficulty be removed to bed.

Mr Hutchinson believes that gonorrhœa only produces synovitis in those who have *already* suffered from rheumatism or gout, including osteo-arthritis, or at least who are the *children* of those who have suffered from these forms of an arthritic diathesis.

It is a curious question whether multiple arthritis ever occurs as the result not of urethral suppuration, but of purulent ophthalmia, leucorrhœa, otorrhœa, or other discharges from mucous membranes. Some cases of the kind have been published by Dr Ord, Mr Lucas, and other observers abroad, but they must certainly be rare, and need careful criticism before the proposed explanation is admitted. When, however, the ophthalmia or leucorrhœa is gonorrhœal, there is no doubt it may be followed by gonorrhœal synovitis, but probably not otherwise (see Mr Lucas's paper in the 'Medico-Chir. Trans.' for 1899, p. 137).

Locality.—Many writers say that the knee is the most frequent seat of gonorrhœal synovitis; but at Guy's Hospital it has long been taught that the feet are the parts most apt to suffer, and this was borne out by analysis of the first twenty-nine cases above mentioned; for in more than twenty of them the ankle, sole, heel, or instep was attacked, while the knee was affected in only fourteen, the wrist in six, the shoulder in three, the hip and the elbow in one each. This pain in the heels and soles of the feet has to be inquired after, for it is not attended by swelling or other signs of inflammation. Hence it is often overlooked in reports, while stress is laid on the more obvious changes in the knee and elbow. The "plantar fascia" is the traditional seat of these pains, which are almost always worse at night, and of an aching, grinding character; but it seems probable that the local lesion here, as elsewhere, is in the synovial membrane and ligaments of the tarsal joints.

However this may be, the much larger number of cases collected between 1873 and 1887, including the twenty-one from the surgical wards, give results more in accordance with general belief, so far as the pre-eminence of the knee-joint is concerned. For among 102 patients, one or both knees were affected eighty-two times, the ankle fifty-five times, the sole, heel, instep, or metatarsus only thirty-seven times, and the hip twenty-six times. Of the joints of the upper extremity, one or both shoulders were affected thirty-four times, the elbow twenty-two, the wrist twenty-six, the metacarpus (and occasionally the fingers) twenty-five, and the sterno-clavicular joint twice. In six cases the cervical vertebræ suffered, and in two the temporo-mandibular joint.

The great toe is never affected alone; but there is often pain referred to the metatarsus generally, and to the smaller toes, which usually escape in gout. The joints of the fingers are very seldom attacked, a point of distinction from subacute cases of osteo-arthritis in young subjects.

In a certain number of cases one joint only is attacked, but this is certainly rare; when it does occur, one knee or one ankle seems usually to suffer. But often, after the other joints are well, one remains as bad as ever, and this appears to be most frequently the elbow or the shoulder.

Cases.—(1) A healthy, well-built young policeman, aged twenty-one, who was in Philip Ward for nearly three months in 1885, had almost every joint in the body successively affected: first the ankles, heels, and instep, then one knee, then the elbow, shoulder, temporo-maxillary, atlanto-axial, and probably the cervical articulations. There was severe sclerotitis first of one and then of the other eye. From being stout and florid he became pale and emaciated, but with time and the free use of opium he gradually recovered, a went out free from pain, though stiff and weak. In February, 1891, this patient came t see the writer. He had very slowly recovered his flesh and colour, but about two yea after his discharge from hospital was as well as ever. He had married six months ago, an since then had felt return of pain in one hip. The right shoulder had never completel recovered its mobility.

(2) Another patient was a man of about twenty-five, who came into hospital in July, 1890 with a third attack of multiple arthritis, after a third gonorrhœal infection. Here, again, the extent and severity of the disease was remarkable. All the joints of the limbs, some of the neck, and one of the temporo-maxillary articulations were successively, and many of them simultaneously, affected. A moderate gleet was present, and also double otorrhœa, but this last discharge dated from long before puberty. It was successfully healed, but the urethral inflammation was only aggravated by attempts to cure it by local means, and at last he begged it might be left alone: the synovitis was not made worse when the gonorrhœa was most severe, nor benefited by its subsidence. The eyes were not affected, though they had been in a previous attack. Relief was obtained by full and increasing doses of opium, but the case was very obstinate, and afforded a good opportunity of demonstrating the effect, which was negative, of treatment by free and continued exhibition of iodide of potassium, guaiacum, and quinine. In his previous attack he had taken a great deal of iodide and had found it quite useless. The temperature was never above 102°, and after a time became normal. In October a new house physician tried new methods of treatment, but with no more success than before. The patient ate well and did not become anæmic, and the pains gradually subsided, but the joints were as stiff as ever. By slow degrees, helped by friction, shampooing, and passive movement, power of motion was restored, but he was only able to leave his bed about Christmas; and when at last he walked out of the ward in January, 1891, though in good health, several of the joints still remained more or less stiff.

Symptoms.—There is no marked onset and no rapid "metastasis" from joint to joint, as in true rheumatism; each suffers for many days, or even weeks. There is less local tenderness than might be expected from the pain, and but little redness or œdema. Effusion may always be detected in the larger and more superficial joints, particularly in the knee.

The pain of gonorrhœal synovitis is usually worse at night, and is always described as of a dull, constant, aching character.

The pyrexia is almost always moderate, and in some instances the temperature remains normal. Hyperpyrexia is unknown.

Gonorrhœal synovitis commonly runs a tedious and protracted course, lasting for several weeks, and even for months, notwithstanding treatment. In hospital practice the obstinacy of a supposed "subacute rheumatism" has often led to the discovery of the fact that the patient had a gleet. More acute cases sometimes occur, particularly when limited to one joint; but even then, though pain and other local symptoms are severe, there is but little pyrexia.

This disease differs from true rheumatism in the important fact that even in young subjects it shows little or no tendency to produce cardiac inflammation—pericardial or endocardial. Among the 112 cases on which this chapter is based, there was in two a slight systolic basic murmur heard for a time, probably pulmonary and anæmic; in three there was a systolic apex murmur (described as doubtful in one case), and in one only a diastolic, presumably aortic bruit, which, however, disappeared in a day or two In the very severe first case above mentioned there was a basic bruit during the height of the illness, but this had disappeared before he left hospital and on his reappearance after nearly six years there were neither signs no symptoms of cardiac disease. In no case does it appear that permane cardiac lesions have resulted from gonorrhœal synovitis, however young t patient and however protracted the disease.

Apparent exceptions are some of them cases of true rheumatism occ ring in a patient with gonorrhœa. Others are cases of suppurative arthritis from streptococcic infection, with subsequent pyæmic (streptococcic) peri- or endocarditis of the "malignant" ulcerative kind. But it would be unwarranted to doubt the authenticity of exceptional cases, recorded by competent observers, of true gonococcal endocarditis. All we can say is that they must be very rare.

However this may be, gonorrhœal synovitis is frequently accompanied by a peculiar form of *inflammation of the eye*. The American patient above mentioned, who came to Sir Astley Cooper on account of a gonorrhœa, told him that two previous attacks had each led to inflammation of the eyes, and a few days later to swelling of the joints. Precisely the same sequence occurred on the third occasion, under the observation of Sir Astley himself. The writer observed this affection in eleven out of nineteen cases under his own care, and it was noted twenty-one times in the eighty-three cases collected for him by Dr Rickets, *i. e.* in 32 out of 102 cases. It usually attacks first one eye and then the other. There is almost always injection of the small radiating vessels of the sclerotic which surround the cornea, sometimes with marked iritis, and often with some degree of catarrhal ophthalmia. It usually subsides in a few days, under treatment by cold bathing, covered from light, and applying atropine drops; but in more than one instance it has returned after having disappeared. This *sclerotitis*, as it may be distinctively called, is quite separate from the purulent ophthalmia which results from inoculation with gonorrhœal pus, and also from syphilitic iritis and choroiditis.

Event.—Recovery seems to have occurred in each of our 112 medical and 21 surgical cases, although some of the patients left the wards before they had entirely lost the pains and stiffness of their joints.

Though peculiarly tedious and obstinate, gonorrhœal synovitis when once cured does not relapse—another difference from true rheumatism. Numerous instances, however, prove that a fresh urethritis will almost certainly produce a fresh synovitis, and in this way the same patient may suffer twice, thrice, or even oftener from "gonorrhœal rheumatism."

Fibrous ankylosis is not an uncommon sequel, but there is no bony union of the articular surfaces.

Mr Brodhurst relates a case in which, during five years, ankylosis invaded the articulations of the vertebræ, the atlas, and the occiput, so that the head could not be moved. That patient had suffered from three several attacks of gonorrhœa, each followed by "rheumatism." About six months after the last attack, and when he was only just able to walk about, he married. Soon afterwards, although there was no urethral discharge, the articular inflammation recurred, and it finally led to the terrible results just described.

The most frequent seat of fibrous ankylosis is in the shoulder and the elbow; but when the adhesions have been broken down under chloroform, the joint generally regains its usefulness. In the more protracted cases it is not very uncommon for eburnation and the other changes of osteo-arthritis to supervene, but the deformity thus produced is, as a rule, only slight.

A remarkable case of *spondylitis deformans* (bony ankylosis of the spine) was brought before the Pathological Society by Dr Fagge in 1877 (vol. xxviii, p. 201). This Mr Davies-Colley believes to have been due to gonorrhœal synovitis of the vertebral joints, and quotes cases of his own in support of that opinion ('Path. Trans.,' 1896, vol. xlvii, p. 207).

Diagnosis.—The mere presence of a urethral discharge is obviously not enough for the recognition of the disease, though the presence of a stricture or a gleet is essential. A man suffering from gonorrhœa may be attacked by rheumatic fever, and the two diseases run their course apart. Or a man subject to gout may be infected with gonorrhœa. Nor can it be denied that some cases of osteo-arthritis, as we shall find in the next chapter, have their probable origin in severe gonorrhœal synovitis.

The distinction from *gout* depends on the age of the patient, the absence of tophi, the distribution of the synovitis, and the freedom from return, except from a fresh urethral excitation. Sclerotitis when present is a valuable help to diagnosis. When the foot is severely affected, the diagnosis is occasionally difficult.

It is more difficult to distinguish gonorrhœal synovitis from *rheumatism*, with which it is still commonly confounded. The ætiology, the different relation to sex and age, the continuous instead of transient affection of the several joints, the much longer course, the freedom from relapses or spontaneous recurrence, the affection of the eyes, and the immunity of the heart are amply sufficient grounds for the pathological distinction between the two diseases, and with care and attention are almost always sufficient for a right diagnosis in practice. The difference in prognosis from both rheumatism and gout, and the no less important difference in treatment, make the discrimination between these diseases of great importance.*

The only mistakes in diagnosis at all commonly seen are due to forgetfulness of the possibility of urethral infection in a case of polyarthritis; or, on the other hand, to the assumption that because a man has articular rheumatism and a urethral discharge, he must have "gonorrhœal rheumatism."

The absence of pyrexia and the normal state of the other organs, together with the fact that the joints do not suppurate, are distinctions from *pyæmia* no less than from rheumatism.

It is a question whether we ought to distinguish the cases above described from certain much more severe and localised inflammations of a single joint following gonorrhœa, which come under the notice of the surgeon rather than the physician. They have been described by Duplay and Brun in France ('Arch. générales de Médecine,' 1881), and by Mr Davies-Colley in England ('Guy's Hosp. Rep.,' 1882). In both articles they are accounted as belonging to gonorrhœal synovitis, but Mr Colley proposes to distinguish them as acute gonorrhœal arthritis or syndesmitis. The joint is very prone to ankylosis after this severe local inflammation.

However this question may be decided by further experience, there is no doubt that we must exclude from the disease now under consideration the rare cases of true pyæmia arising from gonorrhœa, with suppuration of the affected joints, of which an example was recorded in the 'Pathological Transactions' for 1885 by Mr Pollard.

Pathology.—Whether in its milder or in its more severe forms, gonorrhœal synovitis is certainly not a modified pyæmia, a process from which it differs at every point. Nor is it a "hybrid" or "modification" of gout or rheumatism. Possibly it may be connected with the abundant nervous distribution over the prostatic part of the urethra, and so be related on the one hand to the rigors which sometimes follow the passage of a catheter, and to the arthropathies of tabes on the other. But this is mere speculation. The diplococcus of gonorrhœa has been found in the effused serum in the knee-joint by Petrone and Kammerer, and since then by others; and the evidence points to the synovitis and ocular inflammation being infective and due to direct absorption of the specific micrococcus from the urethra.

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* See an excellent paper by Dr Thomas Bond published in the 'Lancet,' March 23rd, 1872, before the writer's paper in the 'Guy's Reports.'

Prognosis.—When the diagnosis of gonorrhœal synovitis has once been made, we have the proof of its accuracy in the prognosis it enables us to make. We can tell our patient that his disease will be obstinate and painful, that it will not affect his life or suitability for insurance, that it will at last get well and will never return unless he acquires gonorrhœa again, when it will certainly again follow and probably more severely than before.

Treatment.—It is important that every effort should be made to cure the urethral disorder, but the synovitis is often little benefited by success.

Some authors recommend iodide of potassium in full doses; but the writer has tried this drug in many cases for a long time, and is convinced of its inutility. Colchicum, quinine, guaiacum, and salicyl compounds have proved equally useless.

The patient must be kept in bed and upon light diet during the first week or two. Brodhurst found the Turkish bath very useful, and instances a patient who has lodged in a house attached to one of these baths, so that he could be carried down into the hot chamber every day; when profuse perspiration was obtained, the pain, which was very acute, left him for some time.

Opium should be given in sufficient doses to relieve the pain and procure sleep. It probably is useful in other ways also, and no other drug is of so much value. Good feeding, bark and nitric acid, quinine, and a moderate use of alcohol, particularly porter, are necessary as soon as the first severity has subsided. Steel is sometimes of use, and arsenic is perhaps still more valuable during the long and tedious convalescence.

In the chronic stage blisters may be applied with advantage, or liniment of iodine, or mercurial ointment. When one or more of the joints has become fixed, it is often advisable to give chloroform and to break down the adhesions by force.

OSTEO-ARTHRITIS

If I were feeble, rheumatick, or cold,
These were true signs that I were waxen old.
DRAYTON.

History and nomenclature—Pathology: relation to gout, to rheumatism, and to wear of the joints—Anatomy—Symptoms and course—more acute cases—Resulting deformity—Age and sex—Osteo-arthritis in children—Ætiology—Diagnosis—Prognosis—Treatment by drugs and external applications—by diet and climate.

Charcot's arthropathie ataxique—its relation to osteo-arthritis and tabes dorsalis.

Synonyms.—Arthritis deformans, Nodi digitorum (Heberden), Malum articulorum senile; Chronic rheumatic arthritis (Adams), Rheumatoid arthritis (Garrod), Rheumatic gout, Irish gout, Chronic rheumatism (in part), Nodosity of the joints, Crippling rheumatism.—*Fr.* Rhumatisme noueux, Goutte asthénique, Arthrite sèche, Usure des cartilages articulaires (Cruveilhier).—*Germ.* Arthritis nodosa sive deformans, Deformirende Gelenkentzündung, Chronische Rheumatismus.

Definition.—A chronic or recurrent subacute disease of the joints, leading to their external distortion and to characteristic atrophy, eburnation, and hypertrophy, unconnected with disease of the internal organs, and not leading to a fatal issue.

History.—This remarkable disease appears to have been first recognised by Sydenham. He says that rheumatism, when free from fever, is often called arthritis (*i. e.* gout), though really distinct from it. He goes on to describe the chronic course of the disease, with its remissions and exacerbations, and concludes his description as follows:—"Potest fieri ut æger omni membrorum motu ad mortem usque privetur, digitorum articulis quasi inversis, et protuberantiis, ut in arthritide, nodosis, in interna magis quam externa digitorum parte se prodentibus: stomacho nihilominus valeat, et cætera sanus vitam toleret." * The affection was again clearly described by Heberden in 1782 as "the chronical rheumatism," which, he writes, "is in reality a very different distemper from the genuine gout, and from the acute rheumatism, and ought to be carefully distinguished from them both. Being so very different in its symptoms, as well as in the event, it would be useful if it were distinguished by a peculiar name, which might prevent its being confounded with other disorders by being called a "spurious or wandering gout," or a "chronical rheumatism." Haygarth in 1805 wrote as follows:—"This disease has hitherto passed under the name of gout or rheumatism, or perhaps has been most commonly called rheumatic gout. But as several advantages would result from a separation of this disorder from others with which it has been confounded, I have ventured to call it the nodosity of the joints." Cruveilhier gave the first accurate anatomical account of osteo-arthritis under the title "arthrite avec usure des cartilages articulaires." Virchow and most German pathologists call it "arthritis deformans." By many English authors it is still misnamed "rheumatic gout," by Adams in his beautifully illustrated monograph, and by other eminent Irish surgeons, R. W. Smith, Colles, and Wilmot, "chronic rheumatic arthritis," and by Garrod "rheumatoid arthritis." It is also supposed to correspond to the "poor man's gout," (*arthritis pauperum*) of older writers; but true gout is far from uncommon among the poorer classes of this country. In popular language this disease is generally called "rheumatism," but often "rheumatic gout," although that self-contradictory term is also applied as a kind of euphemism to what ought to be called gout—*sans phrase*.

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* 'Obs. Med.,' sect. vi, cap. 5 (p. 256 of Syd. Soc. ed.).

Objection may be made to all the above names, and it is best to adopt the term *osteo-arthritis*, which is used in the 'Nomenclature of Diseases' of the College of Physicians, and in the Registrar-General's reports. The course is most often "chronic," but sometimes subacute, hence it is better not to use the adjective as a constant epithet. *Arthritis deformans* is a distinctive and expressive name; but *rheumatic arthritis* is ambiguous, and *rheumatic gout* misleading.

Pathology.—There are still some pathologists who maintain that all articular diseases, including rheumatic fever, are closely related to one another, and in common depend upon what is termed an *arthritic diathesis*. This view is held by many French writers, and in this country by Mr Hutchinson, who thinks that he can identify an "arthritic" iritis, and even an "arthritic" pneumonia.

It is true that the lesions of characteristic gout and those which belong to arthritis deformans are sometimes found in different joints of the same patient, or even in the same joint; and, apart from mere coincidence, eburnation and "lipping" probably follow repeated gouty arthritis and repeated subacute rheumatism. Between 1874 and 1879 Dr Fagge recorded four examples of this association in the autopsies at Guy's Hospital. Three of the patients were men, one aged thirty-six, who said he had rheumatic fever at the age of twelve, and at last died of mitral stenosis. In each case the great toe-joints contained biurate of soda, showing that gout had been present, while the lesions indicative of arthritis deformans were found in the knees or in the hip-joints. In one of three cases the left knee showed both kinds of change, the right that of arthritis deformans alone, and the two great toes that of gout alone. In a fifth and later instance the present writer observed the edges of the patella thickened, and other signs of osteo-arthritis present, together with patches of urate of soda in the cartilage.

In the great majority of cases true rheumatism leaves no deformity behind; and this is a most remarkable and distinctive point in its natural history; but that the synovitis of rheumatism, when unusually protracted, or when frequently repeated, should produce no structural change would be improbable.

The same results are produced more frequently by gonorrhœal synovitis, as we should expect, since here, as in gout, there is a far more persistent arthritis than in true rheumatism.

They may also follow traumatic synovitis, or even hard usage of joints without actual "inflammation" being set up. Thus when making the Catalogue of the Museum of Comparative Anatomy at Guy's Hospital, the writer described a considerable number of specimens of osteo-arthritis in the lower animals, particularly in the carpus or tarsus and phalanges of horses, which are probably due to frequently repeated small concussions.

Hence if we define osteo-arthritis by the peculiar anatomical condition of the joints to be presently described, we must admit that this condition may be the consequence of rheumatic, gonorrhœal, gouty, or traumatic arthritis, if prolonged or repeated. It is, on the other hand, never caused by the more acute and destructive arthritis of tubercle, syphilis, osteomyelitis, or pyæmia.

Nevertheless it remains true that in the great majority of cases this characteristic multiple articular lesion does not follow either rheumatism or gout, or any other form of articular inflammation,—that, in fact, such a sequence is exceptional and rare. On the contrary, it far more often appears independently. Sometimes it may be traced to specially hard usage of the affected joints; more frequently it depends upon the ordinary wear and tear of laborious work during many years; but often it appears in younger subjects, who have never worked hard, and have never suffered from any kind of arthritis.

Moreover the primary or idiopathic anatomical change in the joints is associated with a definite and constant pathological process, and with characteristic clinical symptoms. It has its own course, prognosis, and treatment, and therefore is, on every ground, practical and theoretical, entitled to be considered as an independent "disease."

Anatomy.—The process of arthritis deformans is often limited at first to a small area of one of the cartilages of a joint. Sometimes (as in several specimens exhibited to the Pathological Society by Mr Hutchinson in 1872) it begins round the margin, and spreads irregularly inwards; more often it starts in the middle of the articular surface.

The cartilage first becomes soft and velvet-like in appearance; afterwards it softens and gradually disappears, so that a depression is formed, reaching down to the bone. Histologically the change consists in a proliferation of the cartilage cells, so that each becomes replaced by from eight to twenty corpuscles; the matrix at the same time splits into fibres vertically to the articular surface. Presently the enlarged alveoli rupture into the cavity of the joint; the fibres then remain for a time as shaggy projections, until ultimately they too disappear. There is at no time true ulceration, *i. e.* no suppuration and no formation of granulations.

The denuded bone is said occasionally to exhibit an open cancellous tissue, but as a rule it is converted into a very hard, compact substance, *i. e.* undergoes *eburnation*. When, after the removal of the whole of the articular cartilages, the osseous surfaces everywhere come into direct apposition, they become scored and fluted with parallel grooves and ridges, corresponding with some particular line of movement. The texture of the subjacent part of the bone becomes filled with oil-drops, and so gradually absorbed. Thus the neck of a femur may be gradually shortened until what represents the head lies in a hollow between the two trochanters.

Intra-articular fibro-cartilages, as in the temporo-maxillary joints, resist the disease no better than the cartilages which cover bones. So also the ligamentum teres in the hip, and in the shoulder the long head of the biceps, both disappear.

Hypertrophic changes are associated with this atrophic process from an early period. From the edges of the articular cartilages there arise a series of outgrowths—"ecchondroses,"—which form a raised lip or border. These gradually increase in size, and lime salts are deposited in them, until they become converted into bony growths (*osteophytes*), which not infrequently grow into the capsular ligament, and unite to form a complete osseous shell around the joint. In like manner outgrowths from the edges of the bodies of the vertebræ often cohere together, so as to form bony splints, immoveably fixing a large part of the spinal column.

The synovial membrane of the affected joints is more or less thickened. The folds, which project into the articular cavity, are enlarged and vascular; they often form long villous processes, with numerous bodies like melon-seeds hanging from them; these bodies may ossify, become detached, and lie as "loose cartilages" in the joint. They cause fresh accession of pain, and are named *Mäuser* by the Germans, from their slipping away from the touch.

A modification of the bony outgrowth of arthritis deformans was long ago described by Heberden under the name of *digitorum nodi*, which, as he says, consist of little hard knobs, about the size of a small pea, situated near the joint of a finger; they have no connection with gout, continue for life, are hardly ever attended with pain, and are rather unsightly than inconvenient ('Comment.,' cap. 28). In Sir Dyce Duckworth's treatise on gout, and in that of Dr. Archibald Garrod on rheumatism, the relation of these *nodi digitorum* to gout is discussed. They are certainly not tophi, but exostoses, and in the writer's belief are neither rheumatic nor gouty, but osteo-arthritic.

The changes of osteo-arthritis are attended with a gradual loss of mobility, until at length the joints become completely fixed by the bony splints around. When, however, these are removed after death, the articular surfaces are found perfectly free and smooth; occasionally fibrous ankylosis is established, but neither bony union nor suppuration occurs.

At an advanced stage of arthritis deformans the deformity produced is characteristic. Most of the joints are fixed in a bent position, but the wrists are extended. The fingers lie at an angle with the rest of the hand, and are deflected to the ulnar side, so that the knuckle of the forefinger projects. Some of the phalangeal joints are usually over-extended, so as to be concave on the dorsal aspect, as was noted by Sydenham. The fingers and hand generally are deformed in a regular manner, and the two hands correspond—a striking contrast to the irregular, unsymmetrical distortion of gout.

The degree of enlargement of the affected joints is very variable. Sometimes each articulation is swollen into a smooth, uniform, bulbous tumour; sometimes the ends of the bones look nearly of normal size. The elbows and knees are usually the most enlarged, and are, next to the hands, the most obviously deformed.

Distribution.—The fingers and wrists are commonly first attacked (in about two thirds of the writer's cases), then the knees. The hip may be affected alone, but only in the later periods of life (*morbus coxæ senilis*). Next to the hands and knees, the elbows and shoulders are most often affected, the ankles less frequently, and the toes very seldom. The vertebræ are often found united by osteo-arthritis after death, and this is one cause of the stooping back and stiff neck of old people in the workhouse. Adams figures the disease in the temporo-maxillary articulation, and also in the sternal and acromial joints of the clavicle. These joints, the reader will remember, may be affected in gonorrhœal synovitis, whereas they almost always escape in rheumatism or in gout. Even the joints of the auditory ossicles and of the ossified laryngeal cartilages in old age may be the seat of this wide-spread disease.

Symptoms.—Usually the disease causes more or less local pain from the beginning, but occasionally its early stages are free from suffering. This fact is well illustrated by one of Mr Hutchinson's cases in the 'Pathological Transactions.' A man aged forty-one had his thigh amputated for disease of the knee, and it was not known that any other joints were affected; but the cartilage was found eroded in every articulation of the foot except the distal joints of three toes. As the same writer remarks, one may frequently detect a projecting lip round the articular cartilage of the lower end of the femur in those who are not aware that the knee has suffered. He admits, however, that practice is required to prevent one mistaking for a morbid "lip" a ridge which exists at the same spot in many healthy persons.

Dr Spender, of Bath ('Brit. Med. Journ.,' 1888, vol. i, p. 781), who has seen large numbers of patients in the early as well as in the chronic stages of osteo-arthritis, believes that it is sometimes marked at or even before its development in the joints by cold hands and feet, with a rapid and incompressible pulse (90 to 110 or 120) but with no rise of temperature. He has also noticed as an early symptom pigmentation of the skin, particularly on the forehead and face, and on the fingers, and afterwards maculæ over the legs and freckles on the face. A constant dampness of the hands, feet, and trunk from perspiration, and liability to fits of neuralgia in the affected limbs are other early symptoms which Dr Spender has observed. In chronic cases the writer can confirm the very constant presence of sweating and the cold limbs; but pigmentation he so rarely sees that it might pass for an accident.

It is probable that when a patient begins to complain of pain and stiffness in one or more of his joints, the disease has long been present. The pain is not generally constant; it "comes and goes," sometimes without obvious cause, sometimes in apparent relation to changes of the weather. In certain cases it has a shooting character, so as to resemble neuralgia. With many patients it is worse when the limbs are warm, in others when they are cold; in some by night, in others by day. It is very apt to be brought on by the use of the part; in the hip, for example, by walking even a short distance; in the shoulder, by carrying anything, and is worst in the hand. The stiffness, however, is usually more noticeable after rest, when the patient first attempts to get out of bed in the morning.

A sense of weakness and of distressing fatigue and impotence may be as marked a symptom as pain; and there is often far more wasting of the muscles than seems to be accounted for by mere disuse of the limbs; the thenar and the hypothenar eminences, for example, may be so hollowed that the case looks like one of progressive muscular atrophy, and the intercostal spaces are deepened.[*]

Another characteristic symptom, which comes on in the progress of the disease, is creaking or grating, which accompanies the movements of the affected joints; it can be plainly felt or even heard by the patient, and is easily recognised by grasping the joint with one's two hands while it is slowly bent and straightened.

Occasionally osteo-arthritis sets in from the first with well-marked symptoms; it produces swelling, heat, and perhaps redness of the affected joints, and is attended with more or less pyrexia; so that Sir Alfred Garrod described an *acute variety* of the disease, which closely resembles ordinary acute rheumatism, differing in the greater length of the paroxysm, in the absence of profuse sweating, and in its having no tendency to attack the heart.[†] Mr Hutchinson has sometimes seen it give rise to paroxysms almost like those of gout. In one of the writer's cases this occurred, and in one of Dr Ord's the temperature one evening rose to 102·8°. These cases, however, are quite exceptional, and the disease is in most persons insidious and gradual from the first.

There is no doubt that osteo-arthritis is often attended with effusion of fluid into the affected joints, and that the designation of *arthritis sicca* is inappropriate. Many cases of so-called *hydrops articuli* really belong to this disease.

In rare cases hæmorrhage takes place into the affected joints. Thus in 1875 Dr Goodhart, in examining the body of a man aged fifty, who had been admitted for arthritis deformans, but had died of hernia, found that each ankle contained several drachms of liquid blood, and that there was also blood in both knees, with swollen synovial membrane and vascular œdematous fringes; all these joints, as well as the hips, showed the characteristic changes of arthritis deformans.[‡]

Beside the accumulation of serum in the affected joints, we often see the accessory bursæ and synovial sheaths swollen in like manner, and they add to the deformity of the joint.

The atrophy of the muscles is more than can be explained by want of use. In one case recorded by Dr Spender it affected the sound as well as the osteo-arthritic arm. It probably depends on peripheral neuritis.

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[*] "The chronical species of rheumatism equally partakes of the palsy, for there is always a trembling, weakness and numbness left for some time in the limb affected, and the use has at last in many been wholly taken away" (Heberden, 'Comment.,' cap. 79).

[†] Trousseau states that in four out of nine autopsies of "nodular rheumatism" made at the Saltpêtrière by Cornil, pericarditis was present. It is, however, incredible that this was connected with a disease which rarely, if ever, produces cardiac lesions; and on looking into the cases it is at least probable that chronic cirrhosis of the kidneys was the real cause of the pericarditis which ended some of these long-standing cases of (? gouty) arthritis.

[‡] Among the less common symptoms of arthritis deformans is the presence of fibrous nodules at a distance from joints,—as, for instance, among the muscles of the arms or forearms. I once saw an old lady, aged seventy-one, who for about three months had complained of a curious affection of the tongue and cheeks, which perhaps belonged to this disease, inasmuch as she also had hydrarthrosis of each shoulder-joint, and a less marked affection of her knees. The tongue was uniformly enlarged, and had a peculiar firm, fleshy consistency, without being at all indurated; its surface was rather smoother than natural. At each corner of the mouth there was a button-like mass, which extended outwards for some distance into the substance of the cheek, and on which the mucous membrane adhered more closely than elsewhere to the subjacent tissues.—C. H. F.

Age and sex.—Osteo-arthritis is decidedly more common in women than in men. Of Haygarth's 34 cases all but one were in women. Of 75 well-marked cases which have come under the writer's personal observation, 48 occurred in women and 27 in men. Of Adams' 21 patients, 16 were men and only 5 women, but many of the former were cases affecting the hip alone. Among 500 private cases of Sir Alfred Garrod there were 411 in women and only 89 in men ('Med.-Chir. Proc.,' Nov., 1887).

This disease is very rare in children, and when it occurs has peculiar features (*v. infra*). It is not commonly met with under thirty. Of the 75 cases quoted above, 3 occurred before the patient was twenty-one years old—one in a girl of sixteen, and 7 between twenty and forty; 3 patients were above seventy years old, 7 between sixty and seventy, and all the others were between forty and sixty. In many cases, as Haygarth remarked, it first develops itself in women at the climacteric period, and it has been supposed to depend on uterine disturbances. It is, however, by no means confined to any one period of life. Garrod says that he has seen it in children of ten or twelve years, and he has also met with instances in which it began in people above seventy years of age.

The characters of arthritis deformans differ somewhat according to the sex and age of the patient, so that two types of the disease may be recognised.

One begins in the hands and feet, and subsequently spreads to the larger joints; this almost always occurs in women; it also belongs to the earlier periods of life, possibly because they use their fingers most in needlework. Men are more liable to have the disease in one hip or one shoulder, before any other parts are affected, and this variety is especially frequent at an advanced period of life. Moreover, as Mr Hutchinson remarks, the change in the affected joints is not quite the same at different ages; under middle age the outgrowths of bone, which are so striking a feature in old people, are rare, and if present they are usually small.

Osteo-arthritis in children.—As above stated, the youngest patient the writer had until lately met with was sixteen, and the youngest recorded by Sir Alfred Garrod was ten. But in 1895 a child only six years old was admitted into Mary Ward with marked arthritis deformans. Rickets, congenital syphilis, and tuberculous disease could all be excluded; the heart was normal; the disease had come on gradually, and there had been no ophthalmia, vaginitis, or other suppuration. It was therefore recognised as an unusually early case of osteo-arthritis; but the remarkable point was that the spleen and some of the lymph-glands were enlarged. While the child was in the ward, a late clinical clerk of the writer's, Dr Still, was going round with him, and said that he had observed more than one similar case at the Hospital for Children in Great Ormond Street. He soon afterwards made these cases the subject of his graduation thesis at Cambridge, and has contributed an excellent article on the subject to Allbutt's 'System of Medicine' (vol. iii, p. 102). He distinguishes three kinds of chronic arthritis in children:—true chronic "rheumatism" in the strict sense of the word; slightly modified osteo-arthritis; and the new and remarkable combination of multiple chronic synovitis, with enlargement of the joints, associated with hypertrophy of the spleen and lympharia, as in Hodgkin's disease. It seems to be equally common in girls and boys. Ten out of Dr Still's twelve cases were under six years old. No cause has been recognised. The joints are swollen but not hard, and there is no great effusion. The lesion is more or less symmetrical, and may affect any joint, but usually begins in the knees, wrists, and neck. There is never suppuration nor bony ankylosis. The muscles become wasted and contracted, but do not show the reaction of degeneration. The lymph-glands are swollen constantly, and the spleen usually. The heart is unaffected. There is marked anæmia, diaphoresis, and sometimes moderate exophthalmos. Pyrexia seems to be always present, either a persistent but moderate rise of temperature, or occasional fits like those of anæmia lymphatica and anæmia idiopathica.

The joints, in two cases examined anatomically, showed no eburnation nor even fibrillation of cartilage, and no bony outgrowths. No treatment is known to be of use. Probably the cases described by Chauffard and Raymond belong to this category.

Ætiology.—Osteo-arthritis is the disease commonly called "chronic rheumatism," which cripples the joints of old men in the country who have been exposed to wet and cold during a lifetime of hard work, and often scanty food. These patients have never suffered from true rheumatism; the heart is unaffected (unless from chronic atheroma), and they live to an advanced age; but they suffer much from these crazy joints, and not infrequently are bedridden from the same cause during the last years of their lives.

This "chronical rheumatism" is much affected by the weather, usually worse on damp and better on dry and warm days, sometimes worst with an east wind. The patient feels the changes of the barometer, and suffers least in the settled summer season.

> "The hollow winds begin to blow,
> The clouds look black, the glass is low,
> Hark how the chairs and tables crack!
> Old Betty's joints are on the rack."
> *Signs of Rain*, a Poem by EDWARD JENNER.

The ætiology of arthritis deformans seems to differ altogether from that of gout or true rheumatism. Sir Alfred Garrod was unable, after looking over a large number of cases, to find much evidence of its being transmitted by inheritance; one member of a large family not infrequently suffers severely from it, while the rest remain free. Again, it is not produced by indulgence in rich food or in alcoholic stimulants. On the contrary, it is apt to occur in weakly, ill-fed women, who are exhausted by repeated child-bearing, by menorrhagia or prolonged lactation, by grief or mental anxiety.

Dr Ord, in the 'Transactions of the Clinical Society' for 1877, has brought out the relation of osteo-arthritis to dysmenorrhœa and "ovario-uterine provocation," which the late Dr Fuller had insisted upon. In one of his patients it was regularly developed paroxysmally just before, throughout, and for a short time after each menstrual period, and underwent no less regular remissions in the intervals. He noticed three cases in which the arthritis was limited to, or began and remained excessive in, one side of the body, while the ovary on the same side was painful and tender. Haygarth remarked that his nodosities "usually begin about the period when the menses naturally cease."

Osteo-arthritis sometimes comes on after influenza, but what disease from enteric fever to carcinoma does not!

Garrod believes that tuberculous subjects are especially liable to be affected by osteo-arthritis; as also are "individuals of weak frame, whose circulation is languid, and whose extremities are habitually cold." Among thirty-four cases examined by the writer with this point in view, there were three in which phthisis was also present, and one's general impression is that patients suffering from osteo-arthritis are often troubled with a cough; but the coincidence may be only fortuitous.

The nervous system has often been regarded as the origin of this remarkable disease. Embarrassment in business, mental distress, and "being frightened by shells in a siege," have been called predisposing causes of osteo-arthritis. But we must remember that as every disease may have a cellular (local, "solidist") theory or a (constitutional) "humoral" theory, so in modern language it may be ascribed either to a bacterium or to trophic nerves and centres. Since the last edition of this chapter the expected microbe has been described, first by Schüller, and afterwards by Ballantyne and Wohlmann, confirmed by Blaxall. It is a short dumb-bell bacillus, sometimes looking like a diplococcus; but a French pathologist has found a more slender bacterium in the synovia of osteo-arthritic joints.

To some extent we must recognise any kind of slight but frequently repeated irritation as a cause of arthritis deformans. To a still larger extent we must admit that it is a degenerative or senile change. And, lastly, we shall find that in many cases there is a distinct traumatic origin, such as has often led to the lesions of this disease being confounded with those of fractures and dislocations.

Apart from actual injury, overwork, or the wear and tear of many years, is a cause or an occasion of osteo-arthritis. Senator insists that the joints most used are most apt to be attacked; as, for instance, the fingers and wrists of watchmakers, and of women who have worked hard with the needle or at knitting. Dr Spender has come to the same conclusion from observation of the patients in the Bath Hospital. The result of different kinds of labour in producing pressure on certain joints with "usure des cartilages," eburnation of the articular surface, and prominence of its edges, has been ingeniously analysed by Mr W. A. Lane in several interesting papers published in the 'Guy's Hospital Reports,' the 'Pathological Transactions' (1886), and the 'Journal of Anatomy' (vol. xxi).

The effects of pressure and irritation in producing the anatomical deformity of osteo-arthritis are not confined to the human race. A similar affection occurs in the feet of horses, probably as the result of travelling on hard roads, and is marked by the characteristic osteophytes, eburnation, and "false" ankylosis which we observe in human patients.* The same lesions are found in other animals, including the Cetacea.

Geographical distribution.—Trousseau, after stating that arthritis deformans is "a disease of rare occurrence" (in Paris), remarks that in

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* This may perhaps be the affection described by Aristotle as ἡ τῶν ἵππων ποδάγρα.

"certain damp countries it is so common as to be almost endemic." Signs of osteo-arthritis were detected by Delle Chiaje in bones taken from Pompeii, and by Lebert in skeletons from the catacombs of Paris, so that the disease is clearly not of modern origin, although it has only been distinguished of late years. For an account by Dr Norman Moore of a specimen from a Roman tomb, see 'Path. Trans.,' 1883.

There is no question that arthritis deformans is more common in Ireland than in England or Scotland, or that it is more frequent among agricultural labourers than in towns. Adams found specimens more numerous in Holland than anywhere else except Dublin.

Diagnosis.—The distinction of osteo-arthritis from other affections of the joints rests upon recognition of the characters above given. Its gradual onset, the absence of pyrexia and febrile urine, the freedom of the heart from attack, and the age of the patient sufficiently distinguish it from true rheumatism. From so-called chronic rheumatism it cannot be distinguished, if we use that term as Sydenham and Heberden did (p. 518), and as many patients and surgeons use it now; but from all other chronic affections of the joints it is distinguished by its characteristic deformities. The distribution of the lesions and the whole clinical *facies* of the disease separate it from gout; while the age if not the sex of the patient, and its gradual and insidious approach, render it unlikely to be confounded with gonorrhœal synovitis.

In its early stages the rapid pulse, cold sweating hands, and pigmentation of the skin observed by Dr Spender are of help in recognising the true nature of slight articular pains. In later cases we are helped by the symmetrical deformity, the implication of the fingers, of one hip, or of the mandibular joint, and by the freedom of the toes.

Prognosis.—The tendency of osteo-arthritis is to grow inveterate. The patient often becomes completely crippled, unable to dress or to undress, to carry food to the mouth, or to hold a pen in his hand.

Nevertheless the prognosis is not always unfavourable. Dr Fagge knew the case of an old gentleman who, when he must have been nearly seventy, was unable during one winter to move the right shoulder, so that he had to be shaved by his servant; but during the following summer he became free from the complaint, and it never returned. Dr Ord speaks of more than one of his patients having regained a fair state of health when menstruation became normal; but he does not say that the enlarged joints returned to their natural size.

The disease seems to have no tendency to shorten life, its duration from first to last being perhaps ten, twenty, or even thirty years; and death, when it arrives, is attributable to some intercurrent affection. Haygarth's first patient lived to be ninety-three.

In ten years at Guy's Hospital (1875 to 1884) nine patients died with osteo-arthritis, and none of them from it. The youngest was fifty and the oldest seventy-four; five were men and five women. Two died of strangulated hernia, three of cancer (one of whom showed signs of gout and of old tubercular phthisis), one of cerebral hæmorrhage, and three of chronic interstitial nephritis.

Treatment.—The medicines most serviceable in arthritis deformans are arsenic, guaiacum, and cod-liver oil. Trousseau, following Lasègue, recommended the tincture of iodine in doses of ♏x up to xxx. Iodide of potassium is often prescribed, and is most likely to be useful when warmth augments the pain; but even then it often fails. Garrod speaks highly of the syr. ferri iodidi. Ringer says that actæa (cimicifuga) racemosa has yielded him satisfactory results, and that it is most useful when the pain is worse at night, and when the disease is traceable to uterine derangement.

The writer has found arsenic the most efficient drug in this disease, given in full doses, and continued for a long time; though no doubt cases occur in which it is useless. Patients have told him that as soon as their eyes begin to itch, they know the pains and stiffness will be relieved. If arsenic does not suit the patient or the disease, steel or bark is the best substitute. Guaiacum is indicated when the pains are better at night and worse in the cold, but it is less often useful than in gout and in myalgia. Cod-liver (which indeed was first introduced by Laycock for "chronic rheumatism") is most valuable in checking the progress of osteo-arthritis. A generous diet, with porter or wine, is a great help to treatment, and there is no reason to prohibit meat; but fatty food is the most desirable. When the pain is severe, there is no objection to the administration of opium in doses sufficient to subdue it. Belladonna and chloroform liniment is often useful, but if there is much synovial effusion, a blister is the best remedy.

Hot douches of water or of steam, hot sand poured over the joints affected, hot baths, and counter-irritation are all valuable. The application of hot air has, on the whole, disappointed expectations. Warmth and flannel and powdered sulphur do good, cold and damp are harmful. Passive movement is necessary, and after the hot bath or douche the joints should be well shampooed. Moreover the patient should be encouraged to persevere in exercising the affected joints, to knit, to play the piano, or to make any other exertion of the fingers. Splints are decidedly harmful, for in this case rest is not indicated by pain.

Drinking alkaline waters probably does harm; but the chalybeat springs of Tunbridge Wells or Schwalbach may sometimes be useful. T warm baths of Bath and Buxton, with hot douches and shampooing, a means of relief in some cases; but in others warm baths, whether at hom or abroad, seem decidedly to aggravate the condition.

Lastly, removal from Ireland, and if possible from England, to a warm dry, and equable climate is invaluable. Perhaps most cases might thus b cured if the treatment could be early adopted.

CHARCOT'S JOINT DISEASE.*—It is to the acumen of Professor Charco that we owe the recognition in 1868 of a remarkable chronic affection c the joints which is liable to occur in the earlier stage of tabes (locomoto ataxia). It is sometimes abrupt in its origin, but chronic in its course an remarkably wanting in local signs of inflammation. It was first describe by Charcot in 1853 as a variety of osteo-arthritis, under the title "rhum tisme noueux d'origine nerveuse."

The joint affected, after a period of swelling probably produced b intra-capsular effusion, gradually becomes completely disorganised. Th synovial membrane and cartilage disappear, and the bone undergoe extreme atrophy. But the whole shaft becomes porous and brittl

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* *Synonyms.*—Arthropathie ataxique—Maladie de Charcot—Arthritis tabidorum atr *phica*—Chronic atrophic arthritis—Atrophic arthritis of tabes.

Tuber

the ends, as in ordinary osteo-arthritis. In the knee, which is en affected, the tuberculosis of the tibia and the condyles of the e absorbed, or, when the hip is attacked, its entire head and ith this there is little or no hypertrophy. At last the ligaments laxed, and the ends of the bones so altered, that the joint swings rections like a flail. The shoulder and elbow are also liable to ut not, it appears, the joints of the fingers or of the vertebræ.

zzard has called attention to the frequency of the gastric attacks when this articular lesion is present. He found them present mes in twenty-six cases. Perforating ulcer of the foot has also with in cases of tabes along with Charcot's disease of the joints.

her this affection is a variety of osteo-arthritis deformans, and ts true pathology, are still disputed points. One can, however, doubt that it is a tropho-neurosis in origin, and that it approaches ritis rather in its results than in its cause.

ussion of its nature by Sir James Paget, Mr Hulke, Mr Hutchin- late Mr Morrant Baker, Sir D. Duckworth, Mr Lucas, and other sts, in which many cases were recounted and specimens shown, und in the eighteenth volume of the 'Clinical Society's Transac- d an earlier paper by Dr Buzzard ('Path. Trans.,' vol. xxxi, pp. should also be consulted. A good case was published by Mr Keet- in. Trans.,' 1882), two by Dr Charles Atkin in the Manchester Chronicle,' April, 1885, and six by Dr Sydney Roberts in the hia 'Medical News,' February 14th, 1885. In a well-marked case the knee in a man suffering from tabes in John Ward (1890-91), ained of rather severe pain. This, however, is not usually the fact, nothing is more curious than to see the limb, with its joints nd distorted, freely handled and moved in all directions.

rmany this condition has attracted little attention. The account Eichhorst's text-book merely reproduces the statements of Charcot upils. Tabid arthropathy is not common in this country, although a late sequel of tabes, which might only be found in sick asylums house infirmaries; it belongs to the earlier stages of the disease.

pears to be not at all uncommon in the upper extremity, whereas the arms is very rare compared with that of the legs.

hief distinctions between Charcot's arthropathy and osteo-arthritis bsence of pain, the rapidity of the destructive process, and the atrophy over hypertrophy.

fections of the joints which do not end in suppuration or pulpy ion tend towards the same result, eburnation and deformity. We that chronic gout occasionally, true rheumatism when very fre- epeated, recurrent gonorrhœal synovitis, and chronic traumatic together with the mere wear and tear of long-continued labour, and all produce an anatomical condition of joints, with thickened trophied cartilage, which is indistinguishable from primary osteo- Possibly tabes is only another cause of a similar result. If so, self an important pathological discovery; but on the whole the s above stated, and above all the nervous ætiology of Charcot's dis- to be more important than its resemblances to osteo-arthritis.

RICKETS

AND OTHER GENERAL DISEASES AFFECTING THE BONES

> Whence multitudes of reverend men and critics
> Have got a kind of intellectual Rickets,
> And by the immoderate excess of study
> Have found the sickly head to outgrow the body.
>
> SAMUEL BUTLER.

RICKETS—*History and nomenclature—Definition—Anatomy of the rachitic limbs—of the thorax—pelvis—skull—craniotabes—Histology—The spleen in rickets—Symptoms and concomitant disorders—course, event and complications—Ætiology: age, sex, climate, food—relation to tubercle and syphilis—Pathology—Diagnosis and prognosis—Treatment, preventive and curative.*
MOLLITIES OSSIUM—*History—Ætiology—Symptoms and event—Histological and chemical changes—Pathology—Diagnosis—Treatment.*
Osteitis deformans—Acromegaly—Hypertrophic osteo-arthropathy.

Synonyms.—Rachitis, doubling of the joints.—*Fr.* Rachitisme.—*Germ.* Rachitis, Englische Krankheit. The vernacular term in French is *chastre* (castrum), and in German *doppelte Glieder*.

History.—In the middle of the seventeenth century the famous anatomist and physician, Francis Glisson, drew attention to a disease affecting the bones of children, which he supposed to have recently sprung up, where it was first observed, in the counties of Devon and Somerset. It was then known as "*the rickets.*" *

The term *Rachitis* (or Rhachitis) was proposed by Glisson on account of its similarity in sound, although in his work on this disease (the second edition is dated 1650) he offers his readers a Greek derivation, on the ground that the dorsal spine (ῥάχις) is one of the first parts to be attacked. The word ῥαχίτις, *morbus spinalis*, having been suggested by a friend, he accepts it as expressive of the affection of the spine, as distinctive, and as near to the vernacular term.

He says that "the rickets" is the word common for the disease in the

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* This word has been derived from a verb in use in Dorsetshire—"rucket" (=to breathe laboriously); or from "rick" (=elevation or hump, as "hayrick"); or, by Trousseau, from a Norman word—"riquets," applied to deformed persons, itself said to be derived from Alberiquet, dim. of Alberic, a dwarf in Gothic mythology (see Koch's paper, 'Arch. f. Gynäkologie,' 1885, and Virchow's criticism in the same year. 'Arch. f. Path.,' vol. cii). But the true derivation, according to Skeat, is from *wrikken*, to "wrest," to twist "awry:" whence the phrases a "ricked" ankle and a "rickety" chair.

In framing a Latin word, Glisson bore in mind the following excellent rules: "(1) Ut nomen morbi notabilem aliquam ejus conditionem comprehenderet; (2) Ut id satis esset distinctum ab aliorum morborum et symptomatum nominibus; (3) Ut esset satis familiare, pronunciatur facile, memoriæ quoque accomodatum, non nimis longum, neque operosius decompositum."

' England, in London, and in the southern and midland counties, ;t in the north it is hardly known.

, thesis by Whistler (said to have been published at Leyden in 1645) ıplaint was spoken of as *morbus puerilis Anglorum*, and probably : foreign writers to call it *morbus Anglicus*. It was hence supposed : spread from England to the Continent; but there is little doubt had existed among children from time immemorial on both sides of annel, and at the present day it is as common in Holland and ıy as in England.

y few and doubtful allusions to the disease are to be found in older but there is an antique statue of Æsop, figured by Stiebel in his aph published in 1863, which, he says, exhibits the characteristic ities of rickets. A cast of this statue from the Villa Albani at is in the collection of antique casts in the South Kensington n (No. 229). It represents rather the effects of extreme angular ıre of the spine than of true rickets. Two of the children in 's frontispiece have cyphosis, and are not obviously rachitic.

Grant's 'Observations on the Bills of Mortality' (1662) it is noted the rickets" first appeared in the returns for London in the year when fourteen deaths were ascribed to it. In 1658 the number was r the disease was better recognised), and in 1659 it was 441.

inition.—Rickets is not merely a disease of the bones. It is like atism and Gout, a general, or, to use a bad term, a "constitu-' disease. But just as gout and rheumatism find their most obvious ion in the joints, so does rickets in the bones.

nay be defined as a disease attending the period of development in lier stages of childhood, affecting the whole body, but particularly ised by its effect on the growth of the bones, leading to enlargement ends of the long bones and deformity of the chest, pelvis, spine, ınd limbs. In its extreme form it affects the whole skeleton of the but it often begins in some particular region, and it may remain : to the chest, or to the head, or to some of the limbs, at least so far more obvious manifestations are concerned. But it is far from confined to the skeleton. Its symptoms, as we shall presently see, to the organs of nutrition and secretion. It is a cachexia, proof dietetic origin, and certainly not contagious or specific. It runs nic course, and usually ends in recovery. For some reason the lar form of malnutrition which shows itself in rickets can only be ed at a certain age.

hologically we may perhaps compare it best with scurvy.

atomy: the limbs.—The earliest sign of rickets is swelling of the ses of the long bones. This is well marked at the wrist, where the and ulna form a flattened swelling contrasting with the small hand. ilar deformity may be seen in the ankle. The knee-joint forms w between the enlarged ends of the femur and tibia, and hence the ion "doubling of the joints," which has sometimes been applied to . As the affection advances, the limbs become curved. In the foree bones almost always become convex towards the extensor surface; upper arm the distortion is less uniform. In the thigh the femur knees are thus thrown apart, and the patient becomes bow-legged. The shape assumed by the tibia and fibula varies. They often carry on the curve formed by the femur, so that the ankles meet one another, while the knees are separated; but sometimes they are bent with the convexity inwards, so that the feet are widely separated. Each leg, as a rule, presents a sharp angle, projecting forwards at the junction of the middle and the lower thirds, and producing with the narrow shaft what is called the sabre-shaped tibia.

These deformities are the natural result of the bones yielding to the traction of the muscles and the weight of the body. The tissues are so soft that little force is required to bend them, as can easily be demonstrated after death. The curves in the forearms and upper arms are probably due to efforts made by the child to raise itself by laying hold of fixed objects with its hands: there is often a marked angle at the insertion of the deltoid into the humerus. The distortions of the thighs and legs seem to be caused by the weight of the body in the erect posture; but the angular bend just above the ankles is probably due to pressure transmitted to the tibiæ from the child's feet in crawling about.*

Not only do the soft bones readily bend, but a very slight accident suffices to break them partly through. Such "green-stick" fractures, as they are called, may be caused by abrupt movements; sometimes more than one are seen in the same child. Their effects complicate the more regular distortions resulting from the disease; and interfering but little with movement, and causing but little pain, they are apt to escape notice until a large amount of callus has been thrown out.

The thorax.—More important as regards the patient's health than these distortions of the limbs are the changes produced by rickets in the shape of the chest. Here, again, the first indication of the disease is an enlargement of the growing ends of the bones; namely, of the ribs just where they join their cartilages. The consequence is the formation of a series of little nodules, which can be easily felt, and may often be seen through the integuments, arranged in a vertical line, slanting outwards as it passes downwards on each side of the sternum. This "beading" of the ribs, or "rickety rosary," is sometimes the only discoverable sign of rickets, and must therefore be carefully looked for. In most cases, however, there is alteration in the form of the thorax. If one watches a healthy chlid who is suffering from extreme dyspnœa, due to obstruction of the larynx or trachea, one may observe that at each inspiration the middle parts of the ribs are dragged forcibly inwards. This is especially the case with those ribs which lie towards the base of the chest on each side, and cover the lungs; for the effect is not to be seen where they overlie the solid organs, the heart and liver. The cause of this is that the ribs are unable to resist the atmospheric pressure when they are no longer supported by the counter-pressure of air entering the lungs freely from the trachea. Now, in rickets, it would seem that the mere elasticity of the lungs is sufficient to turn the scale and to prevent the lateral portions of the softened ribs from moving outwards when the child draws its breath; or it may be that this result i brought about by slight passing obstruction of the bronchial tubes. I either case the effect is not transitory, as it would be with healthy ribs but there ensues a persistent flattening, or even a depression, of the chest walls. This generally runs, as a vertical, broad and shallow groove, downwards and outwards from just below the fold of the axilla on each side; as it approaches the margins of the costal cartilages, it forms an angle and slopes away so as to lie almost parallel with the diaphragm. Or one may describe two sulci—the one nearly perpendicular, the other more horizontal —meeting at an obtuse angle near the base of the xiphoid cartilage. The vertical groove is generally said to be formed by the ribs themselves, outside their beaded ends. But Dr Gee ('St. Barth. Hosp. Rep.,' vol. iv) has pointed out that the beads sometimes occupy the bottom of the groove, and that in exceptional cases they may lie to its outer side, so that, in fact, it corresponds with the cartilages only and not at all with the bones. One result of the depression of the ribs is that the higher abdominal viscera are pushed down; the liver projects beyond the costal margins more than in a healthy child; and, as the intestines are commonly full of gas, the belly becomes protuberant, and contrasts strongly with the narrow chest. Another effect, according to Sir William Jenner, is the production of a white friction-patch on the surface of the heart, just above the apex of the left ventricle, where the fifth rib presses on it (see his well-known Lectures in the 'Medical Times' for 1860). Lastly, there is increase in the antero-posterior diameter of the thorax; the sternum is pushed forwards, and the vertebræ form a rounded curve (*cyphosis*).

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* It has been objected that in stillborn fœtuses, believed to be rachitic, similar chang have been observed in the limbs. But there is no evidence that the distortions are identic with those of rickets, or that the intra-uterine disease is rickets at all.

These changes together form what is called the *pigeon-breast*, from its resemblance to the outline of a carinate bird's breast. They are associated with the presence of emphysema in the anterior edges of the lungs, beneath the projecting sternum; while in correspondence with the flattened ribs one may often notice a collapsed condition of the inferior edges of the lungs, and even of parts of their lateral surfaces. Another feature of the disease is that the clavicles are more bent than in the normal state, and carry the shoulders further backwards, with the effect of increasing the apparent prominence and narrowness of the upper part of the chest.

The vertebræ.—The spinal column in a rachitic child becomes gibbous, curved with the convexity backward. In contrast with this *cyphosis* of the dorsal spine, the cervical and the lower lumbar vertebræ have their natural forward curves exaggerated (*lordosis*). The part especially affected is, as Mr Lane has shown in the 'Guy's Hospital Reports' (vol. xlii, p. 319), the junction of the thoracic and lumbar vertebræ, the eleventh and twelfth dorsal, and the first and second lumbar.

The pelvis.—Here various deformities occur, but these are not obvious during the acute stage of the disease, and are only important because in women they may permanently narrow the cavity and obstruct parturition. The sacrum becomes more flexed on the iliac bones, and its promontory moves downwards and forwards. In most cases the brim assumes an hourglass or oval shape, the pubes being approximated to the sacrum, but the opening may be triangular, and the symphysis pubis rostrate.

The dentition in a rachitic child is late and irregular, and the jaws remain narrow. An infant a year old will sometimes have cut none of its teeth; and when two or more of the incisors have appeared, no others may follow for several months. The teeth, moreover, are imperfectly formed; their enamel is defective; in a year or two they turn black and break off, or fall out. Dr Gee has noticed that the second dentition is also delayed.

The cranium.—In marked contrast with other parts of the skeleton, the skull is disproportionately large, so much so that until recently it was believed to be larger than in healthy children of the same age. Ritter von Rittershain has, however, proved by accurate measurements that the enlargement is generally only comparative.

The rachitic skull is square in shape, both looked at from above and from in front, and flattened on the top from the fontanelles not closing at the proper time. The eminences which represent the centres of ossification of the frontal and parietal bones remain very prominent.

Clinically, this is the most important of all the symptoms of the disease, except the beading of the ribs. The principal fontanelle not uncommonly remains open up to the age of three years or even longer; moreover there is often separation of the bones where they meet to form sutures; their margins, being the growing parts, are generally more or less thickened, so that sometimes one can feel a distinct ridge along the vertex, and even down the front of the forehead.

There is often an irregular thinning of the occipital or other of the skull bones—a condition first described by Elsässer, in 1843, as *craniotabes*. The way to detect it is to grasp the head with the two hands, and to make very gentle but firm pressure with the tips of the forefingers over all parts of the cranium in succession. One may then find that certain small spots, generally near the lambdoidal suture, yield and become indented, just as though the bone were replaced by cardboard.

No morbid change in the brain has hitherto been detected. Dr Gee thinks that its growth is dwarfed, like the rest of the body, and that fluid is effused into the ventricles to fill up the empty space within the skull.* Hydrocephalus is of frequent occurrence as a complication of rickets, but in many cases in which the head appears large there is no excess of fluid. The remarkable affection known as hypertrophy of the cerebrum is sometimes met with.

The growth of the whole body is retarded in rickets; a child two years of age may be taken for not more than six months old; a boy of twelve may be no taller than he ought to have been at three. Among forty-two cases in which von Rittershain (1863) made careful measurements at ages between four months and three years, there was only one which the length of the body was not from one and a quarter to two and a half inches below the mean length in healthy children at the same age. Rickety infants, however, are not infrequently fat, and sometimes excessively so.

Histology.—The textural changes in rickets are characteristic. with a strong knife one cuts through a rib and its cartilage, across the plane of the union between them—or if one divides the end of a long bone so as to show on the cut surface the junction between its shaft and one its epiphyses—certain morbid appearances are at once obvious, even to the naked eye. The "zone of proliferation" of the cartilage ought to be a well-defined, straight, narrow, bluish-white line, perhaps one sixteenth of an inch in width; and the yellow "ossifying zone" beneath it ought to be still narrower. Instead of this, in a rachitic bone the zone of proliferation is considerably thickened, reddened, and of a soft spongy texture. Moreover the meeting line between the two zones is irregular and sinuous, with promontories and islands of bone and medullary spaces invading the cartilage. Rindfleisch aptly sums up these changes by saying that the processes which prepare the way for the conversion of cartilage into bone are accelerated, without the ossification keeping pace with them. So, again, beneath the periosteum there is in rickets a soft, red, vascular layer, perhaps one twelfth of an inch thick. It dips into the superficial vascular canals, and often has embedded in it numerous minute osseous processes, which tear away with it from the shaft, leaving the latter rough. The whole of the interior of the bone also, including the medullary cavity, is unnaturally red and vascular.

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* Trousseau imagined that the softness of the cranium allows of a rapid development of the brain, and so accounts for the precocious mental power of rachitic children—a fact, if it be one, more reasonably attributed to their habitual association with adults.—C. H. F.

In microscopic sections the exact nature of the affection can be traced more minutely. The broad bluish-white zone contains long columns of proliferated cartilage cells, thirty or forty deep. But, unlike what occurs in the normal process of ossification, these cells can easily be seen to be directly transformed into stellate bone cells, each of which remains surrounded by a delicate ring, corresponding with the former cartilage capsule. Rindfleisch says that the homogeneous chalky appearance of this "cartilage-bone" enables one to recognise it with the naked eye, even when it is embedded in normal osseous tissue.

The *chemical* composition of the bones in rickets has been several times investigated, and the proportion of inorganic to organic matter has been found much below what is normal.*

Visceral changes.—Following Sir William Jenner, some English observers attach considerable importance to a change in the liver and spleen and lymphatic glands, which he described as an "albuminoid infiltration."† Dr Dickinson has investigated the microscopical characters of this affection, which he finds to be an overgrowth of the fibrous tissue in the portal canals of the liver and in the trabeculæ of the spleen respectively, with some excess of cellular elements also. The organs, he says, feel hard, dense, and elastic; the liver shows yellowish acini, each surrounded by a thin pinkish or grey line; the spleen, which may be so large as to extend below the umbilicus, is of a deep red or purple colour, besprinkled with smooth white spots, or mottled into a pale buff. It is possible to ask whether this fibrous induration of the liver is not identical with syphilitic cirrhosis (*supra*, p. 356). The lymph-glands are moderately increased in size, tough, white, and opaque. It must be remembered that the liver in healthy children is larger in proportion than in adults, and comes down below the ribs, and that the contraction of the rachitic thorax pushes it further down still. Dr Gee states that when rickety children die with an enlarged spleen, its appearance differs in no respect from that of the spleen of ague, or of cachexia due to unknown causes. He thinks that the affection is really a result not of the rickets, but of the general state of ill-health which caused the rickets ('St Barth. Hosp. Rep.,' vol. iv. pp. 69, 265). Dr Dickinson connects it more particularly with emaciation and anæmia, others with congenital lues. It is certainly a rare complication.

General symptoms.—Before any changes in the bones can be discovered, rachitis may begin with sickness, diarrhœa, and fulness of the abdomen, accompanied by languor, drowsiness, loss of appetite, and febrile disturbance. More often these symptoms accompany the osseous changes.

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* The analysis of Friedleben, published in 1860, made the percentage of earthy salts from 33 to 52, which is considerably higher than that given before, although much less than the percentage of 63 to 65 obtained from the bones of healthy children.—C. H. F.

† See 'Medical Times and Gazette' for 1860, vol. i, p. 259.

It is important to remember when called to a child with croup or broncho-pneumonia that the case is often one of advanced rickets, although the mother may have thought the child to be healthy.

Some minor symptoms often lend considerable aid in diagnosis. One is a peculiar *restlessness* at night, which causes the child, even in cold weather, to kick off the bedclothes, as often as it is covered over. Another is the outbreak of profuse *perspiration* on the head and neck and the upper part of the chest, especially during sleep. Elsässer laid much stress on this in connection with his craniotabes. A third sign of rickets is extreme *sensitiveness* of the body and limbs, so that the child dislikes being moved or handled. He will sit quietly on a stool if left alone, but at once begins to cry if taken up. The tenderness appears to be partly in the bones and periosteum, but Sir William Jenner and Dr Gee have noticed that gentle pressure upon the loins or abdomen is sometimes no less painful.

The muscles are soft and flabby, and more or less wasted. In severe cases the child is unable to walk or even to stand, even though he may have been on his feet for some time before he became rickety, so that Dr Gee speaks of "pseudo-paraplegia" under such circumstances. Jenner relates the case of a girl, six years old, who could neither change her position in bed without assistance nor lift her arm an inch from the surface on which it lay; even at a later period, when she had greatly improved, she was obliged to be tied into a chair with a pillow at its back to support her head; and if the head fell forward the nurse had to raise it for her. She afterwards recovered so as to walk without assistance.

The abdomen is swollen and tympanitic, the bowels are often loose, and the motions pale and slimy. Bronchitis and broncho-pneumonia are frequent and dangerous from the weakness of the muscles of respiration. Infantile convulsions are particularly frequent when rickets is present.

The rachitic process, like that of scurvy, is unaccompanied by fever. Any rise of temperature is due to some inflammatory complication.

Course.—Rickets generally runs a somewhat chronic course, but subsides, under favourable circumstances, at the end of a year or two. Some writers, however, have described an acute form of the disease. Senator records the case of a child, four months old, who became feverish, and in whom the epiphyses of several of the long bones of the limbs were swollen and very tender, but without redness: the affection subsided entirely in about six weeks.* Moller published three cases of "acute rickets" so early as 1859.

A remarkable form of what may be called "acute rickets associated with purpura" or rather scurvy complicating rickets—has been described by several authors,—Dr Cheadle ('Lancet,' 1878), Dr Gee (who called it "osteal or periosteal cachexia," 'St. Bart.'s Hosp. Rep.,' vol. xvii), Mr Thomas Smith ('Path. Trans.,' vol. xxvii), Dr Goodhart ('Dis. of Children,' p. 556), Dr Samuel West ('Clin. Trans.,' xxi, p. 209), and Dr Barlow, whose excellent account of eleven cases with two autopsies will be found in the 'Med.-Chir. Trans.,' vol. lxvi, p. 159. See also Dr Eustace Smith's remarks on the point in his 'Disease in Children,' p. 255.

Event.—After recovery from rickets the bones lose their soft spongy character, and become denser and harder than natural. The articular ends are no longer enlarged, probably because they have been overtaken in their growth by other parts, so that the normal proportions are restored. Many of the deformities which are so conspicuous in young children seem slowly to disappear, at least when they do not exceed certain ill-defined limits. It used to be traditionally taught at the Hospital for Children in the Waterloo Road that although tibiæ which were laterally curved might become straight in the course of time, a similar improvement never occurred when they were sharply bent with the convexity forwards near the ankles, in the manner which is attributable to crawling on the floor (p. 532). The pigeon-breast is very generally permanent; and in too many cases the limbs, as well as the trunk, remain distorted for the rest of life. Even when there are no striking alterations in the shape of the bones, one can often recognise the fact that a person was rickety in childhood by his short stature, thick-set body, and large protuberant head.* Such persons are often erroneously supposed to have suffered from hydrocephalus.

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* Fürst is said to have recorded instances of this kind in 1832; but on referring to them it appears that his were cases of rapidly fatal multiple abscesses of joints occurring in infants.—C. H. F.

Complications.—That rickets is not a mere local disease of the skeleton is proved by its being frequently associated with disorders of distant parts, either as complications or sequelæ. These are laryngismus stridulus, tetany, trismus, the slighter form of convulsion called carpo-pedal contraction, and more severe infantile eclampsia; bronchitis and broncho-pneumonia with collapse of the lungs; scurvy producing "acute rickets;" and lastly, an obstinate form of intestinal catarrh with severe diarrhœa.

Congenital syphilis is a frequent complication of rickets,—according to Barlow and Lee's cases in nearly half the number; according to the late Dr Baxter in more than half. The bosses giving the natiform shape to the skull, and the thinning in patches called craniotabes, are then extreme. The diagnosis depends, however, on the recognition of other signs of hereditary syphilis (cf. *supra*, pp. 356, 358).

Age.—Rickets is a disease of early childhood. The common belief is that it is most apt to be developed from the sixth month to the eighteenth, or the end of the second year; corresponding, in fact, with the first dentition. If we inquire closely into cases which are said to have begun later than this, we usually find grounds for suspecting that a slight form of the affection had existed for some time previously, although it may recently have undergone rapid increase. Dr Gee, who collected 635 cases, is disposed to agree with von Rittershain in thinking that rickets does not often begin after the end of the first year. The latest case that he had himself observed was one which seemed to have begun at twenty months. The child, who showed considerable beading of the ribs, had cut the first tooth at six months, and at twelve months it had been weaned, and had walked; six weeks before it came under Dr Gee's notice it had begun to get weak in the legs and loins, and during the last three weeks it had sweated much. Even in that instance the absence of the disease at an earlier period was merely a matter of inference.

Some have supposed that rickets may develop itself in young adults, but this idea appears to have been based upon mistaken diagnosis of certain cases of spinal curvature and of articular disease. Indeed, it seems scarcely possible for the morbid process described above to arise when ossification is completed. No case of "late or retarded rickets" can be accepted without full details of its histology.

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* This is the type described by Victor Hugo in Quasimodo, the hunchback of Notre Dame, who must certainly have had rickets in childhood.

These have been given in the account of a case of rickets in a boy of eleven, shown by Dr Drewitt ('Path. Trans.,' xxxii, 385), and examined after death by Drs Abercrombie and Barlow. Mr Clutton and Mr Davies-Colley have published similar cases at about the same age, and Dr Cheadle one at nine with characteristic histological changes in the bones. But none of these patients were over puberty, and Jenner's two cases were three and nine years of age.

It is certain that rickets may occasionally be seen before the sixth month. Dr Gee speaks of unquestionable beading of the ribs in infants only three or four weeks old. It may be that the starting-point of the disease was in intra-uterine life; but whether it can be recognised at birth is very doubtful. A few supposed instances of such an occurrence in the stillborn fœtus were recorded by Jules Guérin in his 'Mémoires sur les Caractères du Rachitisme' as early as 1839, but Urtel (1873) and Eberth (1878) independently showed that the histology of the affection described as fatal rickets is altogether different; for the process by which the epiphysial cartilages normally undergo conversion into bone was arrested at a much earlier period, before the cartilage cells had begun to proliferate and to arrange themselves in vertical columns. The most conspicuous character of such cases is the extremely stunted form of the limbs.

Achondroplasia.—Cases of so-called "fœtal rickets" have been published by Dr Felix Schwartz of Vienna ('Med. Jahrb.,' 1887), Dr Thomas Barlow and Mr Shattock ('Path. Trans.,' 1881, pp. 364, 369; *ibid.*, 1884; and 'Clin. Trans.,' vol. xxi, p. 291). The condition is probably allied to cretinism when not due to congenital lues. It has been studied with much care during the last ten years, and has been definitely separated both from syphilis and from true rickets, under the title *Achondroplasia.* Dr A. E. Garrod showed the following case at the meeting of the Clinical Society (February 25th, 1898).

A girl six years old had lordosis of the spine, a deep depression at the root of the nose, and shortness of the limbs, particularly in the proximal segment. The membrane-bones of the skull were well ossified, but not those of the base. The long bones were dense and irregular on the surface, without the proper growth at the epiphysial junction. Hence the shortness of the humerus and femur.

Most cases have been described from stillborn children. Those who survive birth are not idiotic, nor are they true cretins, and thyroid medication has so far been found fruitless.

Sex.—With regard to the comparative liability of boys and girls to the disease, authorities differ. Guérin, in Paris, recorded 148 cases in male and 198 in female children; von Rittershain, at Prag, 290 male to 231 female cases; Brunnicke, at Copenhagen, 108 male and only 55 female; and Dr Ritchie, at Manchester, 128 male and 91 female. The general result would be about 53 to 47 in the 100; a difference so small that we may conclude that rickets is equally common in the two sexes.

Dr Gee found rickets in no less than 30·3 per cent. of all children under two years old brought to him at the hospital in Great Ormond Street in 1867. It is far less common in the country than in crowded cities, where children are apt to get too little light and air.

Climate.—The frequency of rickets varies in different countries. It is greater where the climate is damp and cold than in the Mediterranean regions or the tropics. Cases have been recorded from the great towns of the United States, and more recently from Australia.

The geographical distribution in England is approximately given in an interesting report drawn up by Dr Isambard Owen on the returns obtained by the Collective Investigation Committee ('Brit. Med. Journ.,' Jan. 19th, 1889, p. 114). Rickets is common in London, in the great industrial districts of the Black Country, of Lancashire and the West Riding, and of the Tyne and Tees; also in the mining districts of South Wales, in Glasgow and its neighbourhood, in Edinburgh, Dundee, and other large towns in Scotland, and in the only large Irish towns, Dublin and Belfast. It is rare in rural and sparsely populated districts,—in the Scottish Highlands, in Cumberland, North Lancashire, and the agricultural districts of Yorkshire and Scotland, in Derbyshire, North Wales, and generally in the southern, eastern, and south-western English counties. In Ireland, towns as large as Cork, Limerick, Londonderry, and Galway are almost entirely free from rickets.

Ætiology.—Rickets is not hereditary; but it has been attributed to other conditions in the parents, such as syphilis, phthisis, anæmia, and even old age. Sir William Jenner doubted whether impairment of a father's health has any influence in producing rickets in his children. Von Rittershain thought that he traced the disease to the presence of tubercle in the father more often than in the mother. But among the poor a husband's illness may deprive the wife of nourishment, throw more work upon her, and in many ways render her likely to bear weakly infants. So, again, even when the parents of a rickety child have, one or both of them, had rickets in early life, it is very doubtful whether the disease is ever transmitted. A point on which Jenner laid stress, is that the first child of a family, or even the first two or three, may be found free from rickets, where later ones are affected by it; and, again, that if once a woman has borne a rickety infant, those that follow are almost sure to have the same disease. This is due not only to the progressive enfeeblement of the mother's health by repeated child-bearing, but also, among the poor, to the overcrowding and deficiency of food and clothing which are implied by a large family; and perhaps among the middle classes to the way in which children are sometimes kept indoors, when there is but one nursemaid for several of them.

The relation of rickets to *tuberculosis* is very dubious. Jenner, although he contrasts the two "diatheses," adds that rickets does not by any means exclude tubercle. Dr Eustace Smith says that rickets never occurs in children in whom "the tubercular disposition" is well marked; and Trousseau held rachitis and tuberculosis to be opposing diatheses. In contradiction to the statements of von Rittershain just quoted as to the inheritance of rickets from tuberculous fathers, Jenner refers to a table made for him by Dr Edwards, which appeared to show that phthisical parents are *less* likely than others to have rickety children.

The relation of rickets, and particularly of craniotabes, to *congenital syphilis* was discussed at the International Medical Congress of 1881 ('Trans.,' vol. iv, p. 35) by MM. Parrot, Guérin, and Bouchut, of Paris, by Dr Rehn, of Frankfort, and by other pathologists. See also on this point an admirable paper with tables, by Drs Lees and Barlow, in the 'Path.

Trans.,' vol. xxxii, p. 323. There can be little doubt that M. Parrot's view of the ætiology of craniotabes was wrong, and that congenital syphilis when present is only an accidental complication of rickets.

Diet.—Clinical physicians had long suspected that the true cause of rickets, however it may be aggravated by want of fresh air or of sunlight, lay in some definite deficiency in the food on which these children are brought up. Too prolonged suckling impoverishes the mother's milk and starves the child, but does not produce rickets. Too early weaning is only injurious when no adequate amount and kind of milk and other food is given. But ample supplies are no guarantee that a child is being properly fed; it needs a due proportion of proteids, carbohydrates, fats, and salts. These are all provided in milk; but when a child is weaned the natural tendency is to give more of the cheaper starchy and saccharine food than of nitrogenous and oily compounds. Wheaten bread—cheap, good, and abundant—includes sufficient of the former, but needs butter to make it a complete food. The starchy preparations advertised and given are not in themselves injurious, but need mixing with milk. If given exclusively, or in too large a proportion, not only is the child so far starved, but fermentation changes (both lactic and butyric) produce flatulence, acidity, and chronic gastro-intestinal irritation.

It was formerly supposed that the deficient element of the child's food was the earthy salts which are used in the process of ossification. Guérin in 1839 tried to produce artificial rickets by feeding puppies on meat instead of their mother's milk. Chossat and Alphonse Milne-Edwards made the bones of dogs soft by depriving them of phosphate of calcium: but this was not rickets; it was a chemical, not a histological change, as was shown by Friedleben.

The constituent wanting in the diet of a rachitic child is fat. This is proved first by the remarkable experience of the Zoological Gardens in Dublin and in London; and secondly, by the daily experience of treating rickets with milk, butter, and cod-liver oil. See Dr Norman Moore's thesis (1876), with analysis of 200 cases of rickets, and Dr Cheadle's paper (1888) and book (1889) on 'Artificial Feeding of Infants,' and article in 'Allbutt's System' (1897); also the account of Dr Eustace Smith, Dr Goodhart, and Dr Ashby and Mr Wright in their respective monographs.

Rickets is not uncommon in the lower animals. The lion cubs in Dublin were all born with cleft palate, which prevented their sucking the dam's teats until she was properly supplied with bones (including the marrow) as well as flesh. Mr J. B. Sutton has reported some striking cases of rachitis from the Regent's Park Gardens, and finds that in lion cubs the disease is cured by oleum morrhuæ and ground bone-earth.

Pathology.—It still remains difficult to explain how the symptoms of rickets are produced by its cause. It was once supposed that the immediate cause of the changes in the bones in rickets was due to lactic acid dissolving out the lime salts. The acid was said to have been detected in the bones themselves as well as in the urine; and it was stated that more than the normal amount of phosphate of lime was excreted by the kidneys.* Recent analyses disprove both these statements, and our present knowledge of the histology of the disease renders such a theory untenable. Perhaps we cannot at present say more than that the symptoms of rickets are produced by starvation, but starvation in a single constituent of necessary food (oily compounds); and that excess of another constituent (carbohydrates) leads to indigestion, flatulence, and gastroenteritis. The natural ally of rickets is not either tuberculosis or syphilis, but scurvy.*

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* In 1871 Dr Wegner, of Berlin, in the course of some experiments upon young animals with minute doses of phosphorus, found that if, while administering the poison, he withheld lime salts from the food, there arose an affection of the bones resembling

Diagnosis.—The recognition of rickets is very easy when it is fully developed. As a source of fallacy in regard to the craniotabes of Elsässer may be mentioned here a case of cerebellar tumour, in which a somewhat similar thinning of the occipital bone was observed.

The only real difficulty is as regards early cases in young children; and here the mistake usually made is not to look for the signs of rickets. This should never be omitted in cases of infantile diarrhœa, of infantile convulsions, of laryngismus stridulus, of tetany, or of bronchitis in infants.

Prognosis.—It is doubtful whether death is ever caused by rickets alone, apart from complications. Dr Eustace Smith, however, says that he has seen it directly fatal, with extreme dyspnœa and lividity.

As a rule, the death of a child with rickets is due to secondary causes, to diarrhœa, or bronchitis, to convulsions, or laryngismus stridulus, and sometimes to one of the exanthems. The weakness of the muscles, the smallness of the chest, the distension of the abdomen, and the frequent presence of patches of collapsed and airless lung, make a rickety patient a very bad subject for measles or whooping-cough.

If untreated, a case of rickets is more grave in proportion as the child is younger. It is therefore important to be on the look-out for signs of the disease whenever an infant during the first dentition begins to fail in health, or suffers from any of the above-mentioned disorders. For, as follows from its ætiology, rickets is eminently a preventable disease. It is also one that can be cured.

Treatment.—The most important treatment is dietetic. When an infant is suckled, the breast should during the first six weeks be given every two hours, except from 11 p.m. to 5 a.m., during which interval the mother or the wet-nurse should be allowed to sleep, and the intervals be afterwards gradually increased. The child should never lie asleep with the nipple in its mouth, while the mother herself is asleep. If a baby does not thrive, one cause for it may be that its mother's milk is insufficient in quantity or too poor. According to Dr Eustace Smith, it is a sign that this is the

rickets as it is seen in the human subject. He supposed that the phosphorus was a stimulant to the osseous tissue. Heitzmann afterwards stated that lactic acid is capable of acting in the same way. The hypothesis, therefore, as given by Senator, was that the disease is the combined result of the irritant influence of that acid upon the growing bones, and of a deficiency of phosphate of lime, consequent either on there being too little of it in the food, or on its being carried away through the bowels by diarrhœa. But this theory rested upon too slender a foundation of facts, almost as slender as that which supports the theory of lactic acid as the cause of rheumatic fever. The two theories seem, indeed, to be mutually destructive.

* Beside the discussion on rickets in the International Congress of 1881, referred to above, an instructive debate will be found in the 'Pathological Transactions' for the same year (vol. xxxii, pp. 312—404). It was introduced by Dr Fagge, and continued by Dr Norman Moore, Mr Haward, Dr Dickinson, Mr Parker, Sir William Jenner, Mr Hutchinson, Mr Lucas, the late Dr Baxter, Mr S. Watson, and Dr Goodhart.

case when the infant falls asleep while sucking; or one may notice that it sucks at its thumbs until they become raw. If something in addition to the breast-milk is required, one may employ ass's milk; or cow's milk sweetened and diluted with water, according to the age. In many cases the "humanised milk" so prepared is invaluable. No farinaceous food should be given to young infants. The secretion of saliva appears not to be established before the third month; and it is believed that up to that time starchy foods pass through the intestines unaltered, and are discharged with the fæces. Liebig's or Mellin's malted food may afterwards safely be used mixed with milk. Some infants, however, will not take it; and there is no doubt that what suits one perfectly may not do for another.[*]

After five or six months the mother's milk should be supplemented by farinaceous food. At eight months a little veal or chicken-broth, or beef-tea, may be given with advantage; and at ten or twelve months the child should be completely weaned.

When a feeding-bottle is used, the most extreme care is required to keep it and its tube clean, so that it may not turn the milk which is put into it sour. It should be scalded out every time it is employed; and the tube should be always kept in water.

No one who had not witnessed it would believe how utterly mistaken is in most cases the feeding of young children among the frequenters of a London hospital. At a very early age they are allowed to have bacon, fried fish, pickles, potatoes, and beer. If brought up by hand they are given advertised "foods," which are often little better than pure starch; and are sometimes made up with water instead of with milk. If suckled, a child is frequently not weaned until it is eighteen months or two years old; but long before this they sit with their parents at meals, and share what they eat.

A rickety child after weaning needs particular precautions to be taken for its food. Not only must it be nutritious and digestible; it must also be easy of mastication, for the teeth are few in number and often decayed. All the meat should be finely minced or pounded in a mortar; the potatoes should be mashed, and all the lumps carefully picked out. The milk should be diluted with barley water or lime water if there is vomiting or diarrhœa, and when fruit is given it should be stewed or baked, and the skin rejected. Next to milk and butter, fat bacon and bread and dripping are the most valuable articles of food for a rickety child.

A rachitic patient should be kept lying down, and should not attempt to walk, so long as the bones are soft. Splints projecting below the feet may be used for the purpose of rendering such attempts impossible; but mechanical appliances seem to be of little service in straightening the spine or the limbs. Jenner found that when the ribs were inclined to yield, a well-adjusted bandage round the abdomen was useful by restraining the descent of the diaphragm. Shampooing the bent limbs is a useful practice.

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[*] Swiss milk and the other condensed preparations of milk are often given to young children. They undoubtedly fatten rapidly; but the more important tissues seem not to be well sustained, and infants so brought up are apt to succumb if attacked by diarrhœa or bronchitis. Probably these effects are attributable to the quantity of sugar contained in it: but one feels a prejudice against the attempt to preserve artificially a substance so liable to decomposition as milk; and the same doubt would apply to Nestlé's compound.—C. H. F.

At night the child should keep on a mattress, and if its head is tender and inclined to perspire, it should have a horsehair pillow made with a hole in the centre so as to remove pressure from the occiput. Dr West often found this give quiet sleep for the first time for weeks.

The child should be sponged with warm soap and water once or twice a day, or, according to the season, with tepid or "chilled" water, in which sea-salt may be dissolved. Dr West recommends tan baths, made by adding a decoction of oak bark to the water.*

Drugs.—Among medicines the most valuable is cod-liver oil. It should be given even when the bowels are relaxed, unless it causes an increase of diarrhœa, which is not often the case. Small doses of ʒj or ʒiss twice a day are more efficient than larger ones, which are apt to cause nausea. Steel wine and quinine are also useful, and the *liquor ferri perchloridi* is often prescribed with advantage. Dr Eustace Smith has seen marked benefit result from Alison's prescription of tannic acid in doses of half a grain to a grain twice or thrice daily. If an occasional aperient is necessary, a little castor oil or a powder of rhubarb and soda, or the syrup of senna, may be given.

Mollities Ossium.—*Synonyms.*—Malacosteon—Osteomalacia—Le rachitisme des adultes.

In the middle of the eighteenth century instances of extreme deformity produced by softening of the bones were recorded almost in the same year by three observers, Duverney, Morand, and Pringle; and the names of two of the patients—the Marquise d'Armagnac and Madame Supiot—have become historical. Similar cases have since been met with from time to time, but very rarely, although in certain districts near the Rhine Senator states (in 'Ziemssen's Handbuch') that they have been somewhat less infrequent.

Sex and age.—Mollities ossium is far more common in women than in men; among 145 cases quoted by Mr Durham, in the 'Guy's Hosp. Reports' for 1864, thirteen only occurred in males. A case in a young man was reported by Dr Burgess in the 'London Medical Chronicle' for October, 1888, and there is the skeleton of a sweep in Guy's Museum (1000-30) which is an example of the disease.

The *age* at which mollities is least uncommon is between twenty-five and thirty-five; a few of the patients have been under twenty years old, and a few over fifty. In a well-marked case which the writer saw with Dr E. O. Day, the disease began when the patient, a girl, was about sixteen. The only other case he has seen was in a woman a little over thirty. Dr Rehn has recorded the disease in an infant ('Internat. Med. Congr.,' 1881, vol. iv, p. 59); so also has Dr. Bury, of Manchester ('Brit. Med. Journ.,' 1884, vol. i, p. 213).†

Ætiology.—No real cause of this singular disease can be assigned. Habitual exposure to cold and wet, or living in a damp house, have been supposed to give rise to it, but this is absurd; nor is there any constant defect in the diet or surroundings of the patients. Occasionally they have been affected with rickets in childhood; but this was, no doubt, a mere coincidence. In ninety-one of Durham's 132 cases the disease began during pregnancy, or shortly after childbirth; but it may occur before puberty or after menstruation has ceased.

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* The receipt is to take three handfuls of the bruised bark, and boil it in a linen bag in three quarts of water for half an hour.

† See, moreover, the report on Dr Goodhart's case ('Path. Trans.,' vol. xxxiv, p. 201), and Mr Davies-Colley's (*ibid.*, vol. xxxv, pp. 285, 292); also Mr Thos. Jones's 'Diseases of the Bones' (1888).

A case of extreme distortion of the limbs, thorax, and pelvis, recorded by Dr W. J. Webb, of Chicago, in the 'New York Medical Journal' for March 21st, 1885, occurred in a man some of whose brothers and sisters had symptoms more or less like his own. But this case was quite an exception.

Symptoms and course.—Among the earliest symptoms of the disease are pains in the trunk or limbs, which seem to vary in character in different cases, but are said to wander from part to part, so that they are often called "rheumatic." The next thing may be that one of the bones breaks without cause, or during some slight effort, as in getting out of bed. Or a progressive change in the figure may be noticed, the body becoming short, the back rounded and distorted laterally, and the head stooping so that the chin approaches the sternum. There is extreme lassitude, with disinclination for any kind of muscular exertion; and when she walks the patient waddles in her gait, and has to help herself with sticks or crutches.

The bones of the pelvis suffer as much as the rest of the skeleton. The weight of the body thrusts the sacrum downwards and forwards, while the pressure of the thighs pushes the acetabula inwards and forces the pubes together, so that they form a narrow pointed beak. The result is so much distortion that if the unhappy patient is pregnant at the time delivery becomes impossible, and either abortion must be induced, or, if the fœtus is too advanced for this, cephalotripsy or Cæsarian section becomes necessary. In a case under Dr Oldham in 1866 the pregnant uterus had become retroverted, and after the induction of premature labour the patient made a good recovery.

At last the victim of this strange disease is obliged to take to her bed. The long bones break almost of themselves, and the fractures remain unrepaired, the broken ends being merely surrounded by a soft callus, and forming so many false joints. Her limbs also become bent in the strangest way, perhaps one leg outwards and the other inwards, according to the pressure to which they have been subjected while she is lying or sitting, propped up with pillows, and "all of a heap." Towards the last the softening of the bones may be so extreme that one can bend them backwards and forwards without injury. The superficial bones may be indented by the finger, and feel like egg-shells, as if they were merely covered by a thin osseous layer.

The only symptoms, apart from the osseous tissues, are found in the urine. This sometimes, but not in every case, contains a large amount of earthy phosphates. Bence Jones recorded in 1848 ('Phil. Trans.,' vol. i, p. 55) the occurrence in the urine of a patient with osteomalacia of a peculiar proteid, which was afterwards identified by Kühne with his hemialbumose. It has been repeatedly observed in other cases, though it is often absent. See a remarkable case by Dr Bradshaw with a bibliography ('Med.-Chir. Trans.,' 1898, p. 260).

Event.—For a time the general functions of the body may be little interfered with; the appetite and the digestion may be good, and menstruation may go on naturally. Pyrexia is occasionally present, but not to any marked degree.

Ultimately, however, the disease ends fatally, most often from inability to breathe; for the ribs are dragged inwards at each inspiratory effort, so that scarcely any air enters the lungs. Another cause of death is the obstruction to parturition caused by the *rostrate* pelvis. Mollities ossium seems to advance step by step during successive pregnancies, the patient in the intervals regaining strength and being sometimes able to get about. The duration of the disease is usually from four to six years; but sometimes it has lasted eight, ten, or thirteen. Once, in a case recorded by C. Schmidt, it is said to have ended fatally in three months. Only in rare exceptions has it ended in recovery.

Anatomy.—The bones of malacosteon are not only fragile but soft. After death they are readily cut with a knife, and they feel as soft and inelastic as cheese. On section, the compact osseous tissue has often entirely disappeared; if present, it is reduced to a thin lamina beneath the periosteum, within which there may be nothing but a soft pulpy material filling the enlarged medullary canal. Or the cancellous tissue, instead of undergoing uniform absorption, may be hollowed out here and there into rounded or oval cavities. In the case which occurred at Guy's Hospital in 1864, this change was so striking in the bodies of the vertebræ that at first it almost seemed as though masses of a soft myeloid growth had eaten away the bone. The skull was considerably increased in thickness, and showed no distinction between diploe and tables, while its consistence was that of wet pasteboard.

On chemical analysis the proportion of inorganic constituents is reduced to about 30 instead of 66 or 67 per cent.* It is stated that the carbonate of lime is diminished in quantity even more than the phosphate, and that in the latter salt the calcium is deficient in relation to the acid. The result of this loss of mineral matter is to make the bones very light.

As a rule the morbid process seems to be most advanced in the interior of the bone, and least so towards the circumference. The microscope shows that there may be wide differences even within a single Haversian system, the lamellæ nearest the central vessel being completely decalcified, while the outer ones still retain their inorganic constituents. The orifices of the Haversian canals on the surface of the bone are widened, and sometimes a viscid fluid has been seen to exude from them when the periosteum is stripped off.

In early cases the microscopic section of a bone looks like that of one decalcified by acids; but as the disease advances, the Haversian systems become obscured, the canaliculi disappear, blood-discs and pigment become frequent, the osteoclasts, at first increased in number, disappear, and oil-drops accumulate.

The substance which fills the hollow of the bones in mollities seems to be somewhat altered medulla. It varies in appearance, being sometimes of a deep red colour, and spotted with ecchymoses, sometimes opaque and yellow from fat-granules, sometimes translucent from mucous or colloid degeneration. In the red material there are numerous osteoclasts. The periosteum is very vascular and much thickened.

Pathology.—This is as yet unknown. There is no reason to call the process of softening inflammatory. The decalcification of the bones has been supposed to be due to the action of some acid, and lactic acid is said to have been detected in the osseous tissue, and also in urine passed during life.

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* It must be remembered that the percentage given above represents not the actual constitution of osseous tissue transformed by mollities ossium, but an average derived from some parts where the change is extreme, and others which perhaps deviate little from normal.

In the case of Dr Oldham's above cited, the writer found abundant earthy phosphates in the urine for several weeks, and the excess of salts in one case reached four times the normal quantity (cf. 'Med.-Chir. Trans.' xxvii [1844]. Four vertebræ from this case are in the Guy's Museum, 1004-88).

The muscles not only waste from disuse, but Friedreich found histological appearances like those which occur in progressive muscular atrophy. Trousseau and Lasègue observed in some cases that gently stroking the surface of the limbs excited painful contractions of the muscles beneath. It may therefore be that mollities ossium is, after all, something more than a mere disease of the bones.

Diagnosis.—Mollities ossium is easy to recognise at an advanced stage of the disease, but it must be borne in mind that mere brittleness of the bones is not mollities. In aged people, and in those who have been long bedridden, the ribs and some other parts of the skeleton are apt to undergo atrophy, so that one can readily snap them with the fingers (*atrophia ossium senilis*). In the inmates of lunatic asylums this change seems to be particularly frequent. But even in young persons a somewhat similar state of *fragilitas ossium* is sometimes met with. Dr Fagge once saw a young man who was dying of bronchitis, and in whom a large number of the ribs were found to be broken, as the results of muscular efforts in coughing. He had at different periods of his life had fractures of several bones from very slight injuries. Another affection which might be mistaken for mollities ossium is sarcoma, developing itself in a large number of the bones at once, and causing their spontaneous fracture. The distinction of mollities from true rickets is a fundamental one: not only the histology, but the incidence in age and sex, the progress, event, and whole natural history of the two diseases are different.

Treatment.—No means of checking the progress of this remarkable disease has been ascertained. Phosphate and carbonate of lime have been given with a view of supplying the deficiency of earthy salts in the bones; and although usually without benefit, Trousseau narrated two striking cases (one published by Beylard in his thesis in 1852) in which complete recovery of health followed their administration. It is most important for women who show signs of *mollities* to avoid pregnancy, since the evil is aggravated each time that it recurs.

OSTEITIS DEFORMANS.[*]—In 1877, and again in 1882, Sir James Paget described in the 'Medico-Chirurgical Transactions' (vols. lx and lxv) a remarkable affection of the bones to which he gave the above name. His first paper contained five, his second seven additional cases.

The process seems, as the term denotes, to be of inflammatory or irritative nature, with induration and hypertrophy, very chronic, and without pain, fever, or suppuration. It is less rare in men than in women, but befalls both sexes, and usually those who have reached or exceeded the middle eriod of life. It is insidious in its beginnings, gradual in its progress, nd apparently unlimited by any natural check.

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[*] This title Paget states in his second paper had previously been applied to a local *osteomalacia* of the tibia and fibula by Professor Czerny, of Freiburg-i.-B. The correct *form is* osteitis (ὀστεῖτις, from ὀστέον), not ostitis.

The bones affected are those of the skull and the limbs. The long bones enlarge, soften, bend, and become spongy, but afterwards appear to grow harder and denser than before, acquiring a weight and a closeness of texture which used to be described as *osteoporosis*. The overgrowth is from the periosteum like that of splint-bones.

The patient loses his previous stature, from curvature of the back and bending of the knees; and even the ribs may sometimes become fixed by their own overgrowth. The skull is often monstrously enlarged; but the facial bones escape, so that the disease never presents the aspect of what is called *Leontiasis ossea*, or hypertrophic deformity of the bones of both skull and face. The head is pyriform with the apex below, and is carried forwards while the shoulders drop inwards. The thighs are bent out and the legs forward, as in rickets.

There are pains, vaguely styled rheumatic, in the affected limbs; but no internal lesion appears to accompany the disease of the skeleton.

The pathogenesis of this remarkable affection is quite unknown. It is certainly not due to syphilis or tubercle, nor is it allied to gout or osteo-arthritis. The fact that three of the cases first observed died of malignant disease may have been a coincidence (*supra*, p. 90); but in a case of osteitis deformans affecting the skull and limbs, but not the hands or feet, with ankylosis of several dorsal vertebræ in a woman of forty-five, death came about from a pelvic sarcoma (Fielder: 'Path. Trans.,' 1896, vol. xlvii, p. 190).

A case was recorded and figured by Mr Bryant in the 'Guy's Hospital Reports' for 1877 (p. 337), and another by Mr C. J. Symonds in the volume for 1881. The former was a further stage of one of Paget's five cases. Dr Goodhart's first case in the 29th volume of the 'Path. Trans.,' p. 175, was probably one of *osteitis deformans*, and Dr Cayley's in the same volume (1878), p. 172, was certainly such. A remarkable case which lasted twenty years was reported by Dr Watson, of Baltimore, in the 'Johns Hopkins Hospital Bulletin' for June, 1898 (quoted by Osler).

No remedy is known to be of any service, but the disease may go on for many years without shortening life.

The writer saw many years ago, in the University Museum at Prague, an enormously and uniformly thickened skull, which was taken from the body of a patient who died, still young, after narrowing of the bony foramina had gradually compressed the several cranial nerves, and thus successively deprived him of all his senses. Other remarkable cases of more or less limited osseous overgrowth have been recorded, one in the 17th volume of the 'Path. Trans.;' and another more nearly resembling the one above cited, except that the growth was excentric instead of concentric, was described by Sir Prescott Hewett; it is preserved in the museum of the College of Surgeons, and is figured by Mr Holmes in the second volume of his 'System of Surgery,' 3rd ed., p. 330.

ACROMEGALY.—The remarkable affection so called, in which the head and limbs hypertrophy, affects not only the bones but the soft parts. It was first recognised and named by M. Pierre Marie in 1886. It has sometimes been confounded with Myxœdema. A memoir by Erb appeared in the '*Deutsche Medic. Zeitung*' for October, 1887, and two cases were described and figured in the 21st volume of the 'Clinical Transactions' by Mr Godlee (1888), and another by Dr Hadden and Mr Ballance (*ibid.*, 1885, p. 325, and 1888, p. 201).

An excellent monograph on Acromegaly, based on 210 collected cases, by Dr Sternberg, of Vienna, was translated for the New Sydenham Society in 1899. The same Society had previously published the original essay of Marie and a subsequent one by his pupil, Sousa Leite, with a collection of cases up to 1890.

Sternberg has shown that some of the skulls and skeletons of giants preserved in museums are really examples of Acromegaly, among them that of the Irish giant O'Brien in the Hunterian Museum, and that of the Sieur Mirbeck in the Musée Dupuytren. The physiologist, Majendie, described two "femmes-monstres" in the Hôtel Dieu, one of whom from his account of the head, the hands, the tongue, and the voice was certainly a case of Acromegaly. The case of "pathological" (as distinct from "normal") "gigantism" described in 1872 by v. Lauger with huge underhung lower jaw, swollen tongue, and dilated pituitary fossa, is evidently of the same kind. Prof. Cunningham, of Dublin, published in 1879 a case of tumour of the hypophysis cerebri, with diabetes, in a patient with a "gorilla-like" skeleton, still preserved, and agreeing with the cases of Marie, Erb, and Sternberg.

The name is intended to convey the fact that the extremities of the limbs and the head are chiefly affected. The hypertrophy is not confined to the bones, as in Osteitis deformans, but extends to muscles, connective tissue, and skin; the genital organs also hypertrophy, as in elephantiasis, and the hair becomes thick, long, and coarse. The voice is often deep and gruff. Next to the hands and feet the head is most often affected, particularly the nose, the tongue, and the lower jaw, with the same overgrowth and deformity. Glycosuria is not infrequent, and Hertel finds optic atrophy present in the majority of cases.

The disease affects both sexes equally; and all ages, from puberty or soon after to past middle life.

Sternberg has succeeded in demonstrating the hypertrophy of the skull during life by means of the Röntgen rays (figs. 10 and 11, *loc. cit.*, p. 54).

Case.—A patient of Dr Wilks was young and not unpleasing in appearance, until her features underwent so extraordinary a change that the boys shouted at her when she showed herself in the streets. Her face became elongated; and her nose, lips, and mouth enormous. Her hands could not be fitted with ordinary gloves. There was not the slightest infiltration or hardening of the skin or subcutaneous tissue. She gradually became blind, and died after six years "comatose." She was then living in the country, and there was no autopsy. The urine did not contain albumen. This (Wilks informs the writer) was certainly an example of Marie's *Acromegalie*, although at the time it was regarded as a case of Gull's cretinoid condition in adult women (myxœdema).

The writer has only once had a case of Acromegaly under his care, a lady about fifty, in whom all the symptoms were well-marked, though not to an extreme degree. It had come on very slowly soon after the menopause, and was accompanied by characteristic macroglossia and deepening of the voice. The feet were little affected, the lower jaw and the hand more severely. Thyroid extract had been administered without benefit, bu a course of pituitary extract appeared to be beneficial; at the same tim however, arsenic was prescribed, and to this the benefit may have been d

The most remarkable fact in the pathology of this curious disease that the pituitarium (*hypophysis cerebri*) is, almost if not quite with exception, either occupied by a new growth (sarcoma or glioma) or hyper phied, or wasted. The thyroid body also has been found hypertrophied or cystic, but this may be accidental.

HYPERTROPHIC OSTEO-ARTHROPATHY.—In 1890 Marie described a curious condition resembling in some points osteitis deformans, in others osteoarthritis, and in others acromegaly, from which last he proposed to separate it under the term *Ostéo-arthropathie hypertrophiante d'origine pneumique* ('Revue de Médecine,' 1890, p. 1). Three cases were recorded by Mr Thorburn, of Manchester ('Brit. Med. Journ.,' 1893, vol. i, p. 1155), and another by Mr Springthorpe (*ibid.*, 1895, i, 1257), while a few others have been observed in France and Germany, and four in Osler's wards at Baltimore by Dr Thayer, who was able to collect more than 50 recorded cases. The patients are more often men than women.

The hypertrophy concerns the hands and feet like acromegaly, but differs in chiefly affecting the fingers and toes and their nails, and also in being associated with synovitis of the wrists and ankles, and sometimes of the knee. The spine is often curved, as in acromegaly, but the face and skull are unaffected.

In most of the cases yet recorded there has been present either phthisis or chronic bronchitis with emphysema, and Mr Thorburn regards the whole disease as probably tuberculous in character. See his figures of the patients during life in the paper above quoted, and the account of the autopsy with figures of the bones and joints by his colleague, Mr Westmacott, in the 'Pathological Transactions' for 1896, vol. xlvii, p. 177. The pituitarium was shrivelled and hard.

DISEASES OF THE NERVOUS SYSTEM

"Nam, quoniam variant animi, variamus et artes;
Mille mali species, mille salutis erunt."
OVIDO, *Remedia Amoris*, v. 525.

General symptomatology—Clinical diagnosis—Determination of seat and nature of lesions—General pathology—Prognosis and treatment

GREAT and important as have been the advances in our knowledge of infective fevers and other contagious diseases during the last fifty years, the same period has witnessed still greater progress in the difficult and obscure group of nervous diseases.

This progress has been due first to improvement in the anatomy and histology of the spinal cord, and of the brain; secondly, to experiments on animals; and thirdly, to invention of new methods of observation, of which the ophthalmoscope devised by Helmholtz is the most important.

In clinical work, the labours of Erb and many others in Germany, of Duchenne, Charcot and his pupils in France, and of Hughlings Jackson, Ferrier, and Gowers in this country, have resulted in greatly increased knowledge of the seat and course of the various forms of paralysis. Our knowledge of the pathology of nervous diseases and our power of diagnosis have vastly improved; but unfortunately our means of curing them are still very limited. We have learnt better how to use old drugs, but the promises once given by electricity have failed; of the greatest value as a means of diagnosis, its therapeutical value is restricted. Nevertheless increased knowledge of the seat and nature of morbid changes, of their origin and natural progress, has in all other departments of medicine led to increased power of either curing or preventing them, and in this belief we shall not grudge the space needful for describing the complicated disorders of the nervous system, though full pathology and complete diagnosis are often followed by an unfavourable forecast and almost negative treatment.

The peculiar interest of the disorders of the Nervous System is that they are revealed not by physical signs—*i. e.* establishment of the physical state of the affected organs, as is the case with the diseases of the chest and abdomen; nor by chemical observations, as is the case with diseases of the urine—but by physiological symptoms, which often enable us to infer with the greatest accuracy the precise region affected in the central organs of the nervous system. The most important exception is the information quired by the ophthalmoscope. The "physical signs" revealed by this eautiful instrument—hæmorrhage, inflammation, atrophy, degenerations, nd new growths—are found not only to apply to the retina itself, but also o denote disorders in the brain, of which it is originally an outgrowth.

The symptomatology of Diseases of the Nervous System may be arranged as follows:

I. Motor symptoms:
  Paralysis of movement. Paresis.
  Spasms—tonic or clonic.
  Convulsions—Tremors.
  Trismus—Tetanus—Contractures.
  Response to galvanic and faradic stimulus, including
  The reaction of degeneration of motor nerves.
II. Sensory symptoms:
  Paralysis of touch, anæsthesia, of pain, analgesia, loss of muscular sense, of sense of temperature.
  Amaurosis, amblyopia, diplopia, &c.
  Tinnitus aurium.
  Hyperæsthesia, paræsthesia, pruritus, formication, &c.
III. Reflex symptoms:
  Diminished cutaneous reflexes, plantar, cremasteric, conjunctival, pupillary, &c.
  Absence of knee-jerk and other deep reflexes.
  Exaggerated reflexes, superficial and deep (spastic symptoms).
  Disorders of micturition, defæcation, and other complicated reflex mechanisms—of respiration and cardiac action.
IV. Trophic symptoms:
  Acute bedsores—bullous eruptions—glossy skin—atrophied joints and muscles.
V. Mental symptoms.
VI. Symptoms of other organs:
  Otorrhœa — vomiting, constipation, &c. Retinal changes. Wasting.

The practical *diagnosis* of Nervous Affections, after the symptoms have n ascertained, is perhaps most usefully conducted in the following er:

1. Is the patient a malingerer? This is more frequent and more difficult detection in nervous than in other cases—as diseases of the eyes, nach, and skin. It is rare, but its possibility must not be forgotten.

2. Is the disorder hysterical?—more than mere shamming and not to be ted in the same way, but without regular symptoms or course, and able of a good prognosis under suitable treatment.

3. Is it toxic?—due to the rapid or gradual action on the nervous em of arsenic or lead, of alcohol or opium or belladonna, of the chemiproducts of Uræmia, Diabetes, or Cholæmia, of the toxines of Diphtheria, uenza, Tetanus, or Hydrophobia?

4. Is it "essential"?—that is to say, not secondary to injury or to strucd lesions (so far as we know them at present), nor due to "intoxication" n any drug or poison, mineral or organic; yet "real," neither simulated produced by disorders of the will or mental faculties, neither imaginary trivial, but having certain definite symptoms and course and prognosis. h are Epilepsy, Chorea, and Neuralgia.

5. Lastly, do the symptoms depend on organic, structural, or anatomical changes in the nervous system? If so, we then inquire as to the *seat* of the changes in one or more of the following parts:

(*a*) The muscles or the peripheral nerves.

(*b*) The cord and bulb, at what height and in what part of its transverse section.

(*c*) The brain: the pons and crura, the base, the cerebellum, the internal capsule and centrum ovale, and lastly, the cortex of the hemispheres, and even the pituitary and pineal bodies.

(*d*) The dura and pia mater, the skull and vertebræ.

When the seat has been approximately fixed in the nerves or cord or brain, we may inquire next as to the *nature* of the lesion: hæmorrhage, suppuration, softening, sclerosis, syphilitic and tuberculous processes, new growths, parasites; and when this has been more or less probably ascertained, we may turn back to reinvestigate the precise seat of the process with increased chance of success.

The general *pathology* of nervous diseases may be indicated as follows:

1. Congenital deficiency of structure, either morphological, as in the case of microcephalic idiots; or histological, as when certain tracts in the cord are imperfectly medullated.

2. The effect of habit, as in palpitation or constipation or gutta rosea or histrionic tic.

3. Direct mechanical injury.

4. Parasites—animal or vegetable.

5. Poisons—mineral, vegetable, or animal; including toxines and ptomaines.

6. Malnutrition, and particularly the malnutrition which results from general or local anæmia.

7. The wear and tear of usage, acting chiefly by the decay of the arteries leading to anæmia, rupture, or thrombosis.

The *prognosis* of nervous diseases is, with few exceptions, as uncertain as their diagnosis is in most cases difficult. As a rule, it is better in youth than in adult life, and worst in old age. It is also better in women than in men, probably because their nervous system is more easily and gravely disturbed by slight causes.

The *treatment* of diseases of the mind and of the nerves, of paralysis and of pains, is on the whole less effectual than that of other disorders. The indications are less definite, the therapeutical agents weaker, and surgical interference more hazardous and less often successful.

Long perseverance and anxious patience are needful, but already treatment is far less injurious and more successful than it was fifty years ago, and it will certainly advance with the recent progress in nervous pathology and diagnosis.

AFFECTIONS OF THE NERVE-TRUNKS

> "But pain is perfect misery, the worst
> Of evils, and, excessive, overturns
> All patience." MILTON.

NEURALGIA.—*Definition—Pain—Pathology and general clinical characters—Trifacial neuralgia—Neuralgia of arm and trunk—Referred visceral pains—Treatment of neuralgia—Local neuritis—sciatica.*

PERIPHERAL PARALYSIS.—*Causes—Motor palsies of vertebral nerves in particular—The reaction of degeneration—Sensory paralysis—Pain—Causalgia—Glossy skin and other trophic results—Diagnosis, prognosis, treatment—Paralysis of cranial nerves—Motor, of the portio dura, hypoglossal, and oculo-motor—Sensory, of the trifacial and olfactory—Treatment.*

MULTIPLE SYMMETRICAL NEURITIS.—*A form of peripheral paralysis—Its history and symptoms—Histology—Ætiological varieties—alcoholic, plumbic, diphtheritic, &c.—Beri-beri—Prognosis and treatment of peripheral neuritis.*

THE affections of the nerve-trunks naturally fall into two clinical groups. In one *pain* is the principal symptom, without loss of power or of touch. No histological change can be discovered in the affected nerves, and we therefore name these cases, from the perverted function, *Neuralgia*. In the other group the chief symptoms are loss of muscular power or of sensation in the area of the nerve; this is known as *Peripheral Paralysis*, to distinguish it from paralysis due to disease of the brain or cord.

NEURALGIA.—Pain is a symptom common to most diseases, and in one sense all pains are neuralgia. We limit the term technically to pain of peculiarly acute kind, running in the course of a sensory nerve, and not due to any obvious primary lesion. It follows that our object in every case of neuralgia is to refer it to some cause and thus get rid of the name.

Pain itself appears to be an ultimate fact of consciousness, incapable of definition or of explanation. It has often been said that pain is the reaction of the sensorium to a certain degree of excitation beyond what would cause common sensation; but hyperæsthesia may exist without pain, and pain is often accompanied by anæsthesia. In fact, it is now established that there are afferent nerves and centres for painful impressions, distinct from those for common sensation and for temperature, and their separate course in the grey substance of the posterior cornua has been detected.

Pain may be excited by injury to one of the sensitive nerves in its course or at its periphery, or by lesion of its origin. In each case the pain is referred as a conscious sensation to the peripheral distribution of the nerve. The most common cause of pain is increased pressure on the nerves, but other mechanical injuries, and chemical or electrical disturbance, may produce the same effect. Pressure on the papillæ and their touch-corpuscles or other tactile end-organs produces the sense of touch; when these are destroyed, as on the surface of an ulcer, no true tactile sensation is produced: but pain may still be excited. If, however, the pressure or injury passes a certain limit, pain disappears again and numbness or paralysis ensues.

Under ordinary circumstances, the impressions which give rise to pain are made upon the *terminal* filaments of a nerve; and the sufferer recognises as the seat of the pain the part to which the filaments in question are distributed. In neuralgia, on the other hand, the pain is not excited by direct irritation of the distal extremities of the nerve to which it is referred.

If we accept this distinction, we must deny the pains of toothache when directly referred to the aching tooth to be true neuralgia, and we must admit as true neuralgia the pain produced by an aneurysm or other tumour pressing on the branches of nerves.

Practically, however, we must be content to call neuralgia that kind of pain which is, so far as we know, primary or idiopathic. When pain is not produced by pressure due to inflammatory engorgement of vessels (the most frequent cause), when it is not caused by injury or other direct cause, then we may call it neuralgia, at least provisionally. This is the way in which we shall find that epilepsy and some other functional disorders of the nervous system are defined. Its justification lies in the empirical fact that the pains which on this diagnosis are called "neuralgia" have certain clinical characters in common which separate them from secondary pains.

In one group of cases we can give some explanation of the neuralgia—namely, when the neuralgia is due to peripheral irritation, but not irritation of the painful nerve, so that the patient is mistaken in his interpretation of the local sign. This is sometimes called "reflex neuralgia." Thus the trifacial neuralgia which is often excited by disease of a tooth is most readily explained by supposing that the irritation received from the affected dental nerve spreads from one nerve nucleus to another within the cerebro-spinal centres. This "referred visceral pain," as Dr Head calls it in his interesting articles in 'Brain' (1893), and in 'Allbutt's System' (vol. vi, p. 724), is distinguished from idiopathic neuralgia of the less severe kind by superficial tenderness more usually accompanying the pain.

The clinical characters of neuralgia are as follows:

(1) The pain is not only referred to the peripheral ends of the nerve, but is felt to shoot or dart along its course.

(2) The pain comes and goes; it rises rapidly to severe intensity and as rapidly subsides again. Moreover it returns again and again in fits, sometimes with long intervals of ease, sometimes with only short intermissions; or again, while a certain degree of discomfort or tenderness is almost constant, severe paroxysms of pain occur from time to time.

(3) The pain is stabbing or piercing in character; not throbbing like that of inflammation, nor grinding like that of toothache, nor [illegible]
by movement like that of lumbago. It has not the di
tual, baffled character of the pain which accompan
efforts of obstructed peristalsis, micturition, or ch
ing, subduing pain of orchitis; but it is clear,

in quality, coming in bouts which force cries from the sufferer, and leave behind them a massive, wearying after-swell of exhaustion.

(4) Certain "tender points" are often developed, pressure upon which causes increase of the patient's sufferings. Valleix, who first drew attention to these *points douloureux*, showed that they are constant in position for each of the principal cutaneous nerves, and correspond with the spots at which they emerge from bony canals or pierce the deeper fasciæ. Trousseau afterwards laid stress upon the presence of an additional tender point at the spinous process of the vertebra, beneath which the affected nerve escapes from the spinal canal—the apophysial point. He regarded the painful points as fixing the maximum pain in an area of superficial hyperæsthesia, and that they were usually found in cases of reflex neuralgia from visceral disease: or as Dr Head puts it, they occur not in true neuralgia but in "referred visceral pain."

Eulenburg, who includes migraine under neuralgia, found tender points in only about half the cases which he examined. Anstie believed that they only appear when the neuralgia is severe and persistent. It seems probable that they are, after all, merely spots at which nervous filaments happen to be so placed that they can be readily compressed.

(5) Another character of neuralgia, which is nearly constant, and is often useful for diagnosis, is its limitation to one half of the body. Even when it attacks a large number of nerve-trunks on the same side it seldom spreads across to those of the opposite side; and it is perhaps never quite symmetrical, with the same degree of severity and the same extent of distribution, both on the right and the left side.

(6) A frequent effect of neuralgia, particularly secondary or reflex neuralgia, is that after its subsidence it leaves the affected parts tender to the touch.

(7) Lastly may be mentioned, as a negative diagnostic sign, the remarkable absence of signs of inflammation.

Even with the help of these characters, it is often a question whether a given pain should be rightly termed neuralgia or not. The pain in the shoulder which often attends abscess of the liver, the pain in the arm which is caused by aortic aneurysm, the pain in the testicle due to a renal calculus, are each neuralgic, because they are not due to lesions of the shoulder, the arm, or the testis; they are reflex or referred neuralgia. Gastrodynia and angina pectoris are neuralgic in character, and so is the pain which accompanies the passage of a renal calculus; but they are neither idiopathic nor reflex.

This remarkable "sympathy" between the viscera of the great cavities and the nerves (vaso-motor as well as sensory) of corresponding parts of the surface is probably referable to the fact that the visceral and parietal nerves were once united, before the splitting of the somatopleure and splanchnopleure, and are still connected, at their proximal point of union, with the centres of the several segments of the spinal cord. Perhaps the nerves of the scalp may bear the same relation to the brain beneath, which was shown by Hilton and by van der Kolk to exist between the [illegible] nerves generally and the organs which lie beneath. Just as
of the lung may be accompanied by pains referred to
ted to the skin over the chest, so we shall
brain and its membranes may be at-
of the frontal, the temporal, and

the occipital nerves. The pains themselves are "neuralgic," but the presence of other symptoms shows that the disease, as a whole, is something more than neuralgia.*

Neuralgia of the fifth nerve. Trifacial neuralgia. Prosopalgia. Tic douloureux. This anatomical group includes the most severe of all neuralgiæ—a terrible malady commonly known as *tic douloureux*.† By Trousseau it is called "epileptiform neuralgia," because it occurs in paroxysms with sudden onset. It was described by Fothergill, in 1773, as "a painful affection of the face, distinct from the rheumatism and the ague in the face." True tic douloureux is happily very rare, and can generally be separated from reflex or referred neuralgia, and from less severe and more curable idiopathic neuralgia.

The absolute suddenness with which the pain of tic douloureux comes on is one of its most remarkable characters. The patient is perhaps sitting quietly reading, when he jumps up from his seat, and walks up and down the room, stamping, and pressing his hand against his cheek; or he rocks himself backwards and forwards in the chair, crying out or uttering deep groans. In ten or twenty seconds, or a minute at the longest, the paroxysm is over; it ceases as abruptly as it began. The pain sometimes affects all the branches of the fifth nerve; sometimes only those of the second or of the third division. In certain cases the muscles of the affected side of the face are thrown into violent spasms during the fit of pain, so that the patient makes horrible grimaces and contortions. These are the cases which properly deserve the title of *tic douloureux*. Flushing of the face, redness of the eyes, and lacrymation, usually following pallor, are frequently present, and show that vaso-motor and secretory fibres accompany the fifth nerve. The paroxysms may return every few minutes; Trousseau mentions one patient who had sometimes twenty in an hour; and that in the worst cases they do not intermit, even during the night. They are often brought on by movements of the jaws as in speaking, or in eating or drinking; and sometimes pressure upon one of the teeth will instantly excite an attack. It is not worse at night, and is irregular in the time of its attacks. Sometimes remissions occur, the patient remaining free from the disease for several days together, or even for months. But sooner or later it returns, and is as severe as ever.

The "tender points" or maximal points are usually well marked; they are situated at some or all of the numerous spots at which branches of the fifth nerve emerge from bony channels, or perforate fasciæ. Pressure upon any of them is instantaneously followed by agonising pain; and a breath of cold air upon the face may have a similar effect.

When the disease is of long standing the hair disappears from the affected side of the face, being worn off by friction. Zona not infrequently follows or is followed by neuralgia, particularly when it affects the s[illegible] orbital nerve. The hair of the scalp in supra-orbital neuralgia and of the beard in infra-orbital is sometimes turned white on the affected side.

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* On this subject the writings of the late Dr Ross, of Manchester ('Diseases of the Nervous System,' vol. i), of Dr Mackenzie, of Burnley ('Brain,' 1892, and 1893, 'Med. Chronicle,'), and of Dr Head ('Brain,' 1893–4–6), should be mentioned. Also Dr L[illegible] important section on the nervous system in Schaefer's 'Text-book of Physiology,' and the experimental work published in the 'Phil. Trans.,' by Prof. Sherrington.

† "I hope your tick-doleroo, or however you spell it, is vanished. I have frightful impressions of that tick, and so altogether hate it as an unpaid score or the tick of a death-watch."—*Chas. Lamb to Bernard Barton.*

As Fothergill originally stated, the disease scarcely ever occurs under forty, and often begins at a much later age. Anstie says that the worst case he ever saw was in a woman who was eighty years old when she was first attacked, but this was an exception. The disease usually begins between forty and sixty. It is not more common in one sex than in the other.

Sufferers from severe facial neuralgia are often the subjects of suicidal melancholia, and their mental condition is almost always one of depression. This is no doubt due to the severe pain which they endure, but, also, they are sorely tempted to seek a temporary relief in drink, which brings its penalty in increased despondency afterwards. The ætiology of this severe form of facial neuralgia is quite unknown. It is not hereditary, occurs in both sexes, and is not connected with insanity or hysteria. No constant or important histological changes have yet been found in the trunk of the nerve or in the Gasserian ganglion.

It does not appear that this form of neuralgia is ever caused by peripheral irritation of any branch of the fifth or of other nerves. According to Tomes, true tic douloureux never depends upon diseased teeth; he quoted, as a warning, a case of Trousseau's, in which the pain came on when the patient touched with the tip of his finger his few remaining teeth, but in which they were extracted without the slightest benefit resulting. Patients, who have in vain had a large number of teeth removed, come to us with their disease unmitigated.*

Neuralgia facialis minor.—Less intense trifacial neuralgia often occurs in young persons, and may depend on disease of the teeth. The pain shoots and darts along the branches of the fifth nerve, but it is generally accompanied with a dull aching or gnawing sensation. It is often paroxysmal, and (like other forms of neuralgia) it is apt to come on when the patient is fatigued or exhausted from want of food. It is seldom severe enough to prevent him from attending to the duties or pleasures of life, although it may cause him great discomfort. In many cases it is relieved at once by a glass of wine, or a dose of quinine. The most frequent cause of this form of neuralgia is chronic inflammation of a tooth-pulp. Other conditions mentioned by the elder Tomes and by James Salter are the difficult eruption of a wisdom-tooth (or its impaction in its socket), the presence of secondary dentine in a pulp cavity, exostosis, hypertrophy of the crusta petrosa, alveolar periostitis, decomposition of a dead pulp in a confined space, and even the exposure of sensitive dentine, or the crowding of the teeth together from insufficiency of room. In many instances the affected tooth is tender, so that sudden pressure on it, or the contact of substances much hotter or colder than itself starts an attack of the pain. Salter related one instance in which, although the offending teeth were painful when touched, doubt for a time prevailed as to the real cause of the neuralgia from which the patient suffered, because this returned again and again, there being only a temporary interval of ease after each tooth in succession had been extracted, until the last two were removed at one operation, when the pain entirely ceased. All the teeth had nodules of exostosis on their fangs. It might be expected that any affection of a tooth in the lower jaw would give rise to neuralgia in the course of the branches of the third division of the fifth nerve, and that a diseased tooth in the upper jaw would set up pain in the distribution of the second division. But this is not always the case. Indeed, Salter mentions, as very frequent seats of "dental neuralgia," the supra-orbital nerve, the globe of the eye, the temple, and particularly a spot a little to one side of the vertex. The practical rule would seem to be that the only way of avoiding the risk of overlooking affections of the teeth as causes of the milder forms of trifacial neuralgia is to have the jaws thoroughly examined by the dentist in every case. Salter, who suffered severely from neuralgia himself, recorded some curious instances in which the nutrition of parts affected with reflex neuralgia from caries of the teeth underwent perversion; in one, the iris of the affected eye, from being of a deep hazel colour, became of a dull grey.

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* In the case of Dr Pemberton, a London physician of great repute in his day (1765—1822), the os frontis was found after death to be unusually thick, and a bony mass lay within the falx cerebri. It was supposed that these changes had set up an irritation which caused the neuralgia, but there is no evidence that any branch of the fifth nerve was involved in them, and outgrowths of bone of a precisely similar kind are often found in those who have had no pain in the head or face, nor any cerebral symptoms.

In a celebrated case of Romberg, in which the carotid artery was dilated to twice its usual size where it passed through the cavernous sinus, it seems very doubtful whether the so-called aneurysmal condition of the vessel had anything to do with the tic douloureux from which the patient had suffered on the corresponding side of the face; for that disease had existed for eighteen years at the time of his death, and one cannot suppose that the artery had been dilated during the whole of that time. I have repeatedly seen a similar affection of the carotid at the same spot in the bodies of those who had made no complaint of neuralgic pain during life. Again, it is difficult to believe that the fifth nerve could have been pressed upon without the sixth and the third nerves having been first paralysed.

In Jeffries' oft-quoted case, a triangular piece of china from a broken cup remained lodged in the cheek of a girl, and gave rise for fourteen years to violent pains, which ceased a few weeks after its excision; but that patient was too young for true tic douloureux.

It must be borne in mind that pains undistinguishable from those of neuralgia are often the earliest symptom of diseases of the maxillary and other facial bones. There is in the museum of Guy's Hospital a specimen of Bright's, in which a morbid growth made its way from the sphenoidal sinuses into the middle fossa of the base of the skull, and doubtless pressed upon the fifth nerve; the patient, a woman of forty, whose countenance showed strong indications of suffering, complained of severe pain on the same side of the face, which became more violent in paroxysms.

The less severe forms of facial neuralgia as well as other local varieties are more often seen in women than in men; they are most common between puberty and marriage or at the climacteric period. They often accompany anæmia of the slighter kinds, hysteria, and malaria. The so-called neuralgia of gout, diabetes, and alcohol is probably due to peripheral neuritis.

Cervico-occipital neuralgia needs but brief consideration. Among the ascending branches of the cervical plexus, the great occipital, or posterior division of the second spinal nerve, is the one to which neuralgic pain is most often referred. One must, however, be careful not to overlook disease of the cervical vertebræ in these cases.

Brachial neuralgia generally affects more than one branch of the brachial plexus. Numerous "tender points" are developed, most of them at spots where the nerves pierce the fascia, and one at the inferior angle of the scapula.

It is probable that some cases are not, like *tic douloureux*, purely functional, but are due to a chronic interstitial neuritis of the affected trunk. Such cases are distinguished by their chronic course, the less acute but more constant pain, local tenderness of the limb on deep pressure, and the occurrence of trophic symptoms, such as a glossy state of the skin (*infra*, p. 576), the peculiar sensation known as causalgia, and more rapid wasting of the muscles than is accounted for by their disuse.

In fact, while the fifth cranial nerve seems to be the favourite seat of pure neuralgia, and the lower limb of painful neuritis, both forms of disease are met with in the upper extremity; and here the neuralgia is most often secondary and "referred."

Brachial neuralgia is liable to be aggravated by muscular movements of the arm. Anstie mentions how, when convalescence was almost established, the act of playing on the piano for half an hour brought back the pains at once. Salter ('Guy's Hosp. Rep.,' 1867) has shown that neuralgia affecting the nerves of the upper limb is sometimes due to diseases of the teeth. He says that pains in the shoulder and acromion, over the insertion of the deltoid, or at the bend of the elbow, not infrequently depend on this cause; and he cites the case of a lady who, whenever any of the teeth in the left side of the lower jaw became tender from caries, was immediately attacked with severe neuralgia at a circumscribed spot in the forearm.

The most severe case the writer has met with was in the person of a well-known man of science who, when considerably over seventy years of age, was attacked by neuralgia of the shoulder and arm following zona. The pain was incessant, except when deadened by narcotics, for several months, and was so severe as to overshadow the ordinary symptoms of a fatal intercurrent disorder, which at last relieved his sufferings.

Among the rarer causes of pains in the arms that might be mistaken for neuralgia may be mentioned disease of the articular processes of the cervical vertebræ. In 1876 a woman was admitted into Guy's Hospital under Dr Wilks, who had for four months been suffering from pains in the right shoulder, round the side, and down the arm to the tips of the fingers. No cause for her complaints could be discovered, but after a few weeks she was attacked with pneumonia, and died. Dr Goodhart made a *post-mortem* examination, and found that there was disease of the joint between the seventh cervical and the first dorsal vertebræ, and in this the lowest root of the brachial plexus was involved. In a case of brachial neuralgia quoted by Watson, a sailor suffered in the distribution of the median nerve after a bullet wound in the arm had healed; the arm was at last amputated, with immediate cessation of the pain, and in the median nerve a fragment of lead was found embedded.

Intercostal neuralgia.—Under this head may be mentioned the stitch in the side which often comes on in healthy children after running. It may occur on either side, and therefore is certainly not due to hypothetical "congestion of the liver." It seems by its acute character and its speedy disappearance to be a true neuralgia, and it ought to be carefully distinguished from intercostal myalgia (often called rheumatism), which is increased by movement of the ribs. To both the vague term pleurodynia is applied, as well as to many cases of dry pleurisy. But there is a kind of "stitch" in the left side commonly observed in cases of chlorosis, and of mitral disease, which seems to be in some way due to pressure on the intercostal nerves.

The most important intercostal neuralgia is that which precedes, accompanies, or follows an attack of zona. It is acute, unilateral, follows the course of a nerve, and corresponds exactly to the supra-orbital neuralgia which follows zona of the forehead. It also conforms to the rule that the severity and persistence of neuralgia increase with age; for it is either slight or entirely absent in the case of children, sometimes severe but always of limited duration in adults, while in old patients alone do we meet with cases which are as terrible both in degree and in duration as the worst cases of tic douloureux.

Neuralgia of the *lumbar nerves* is less frequent than that of the face or limbs, and agrees closely with cervical neuralgia. The cause may be disease of the vertebræ, or of the sacro-iliac joint, and, like cervical neuralgia again, it is often associated with myalgia.

The affection described by Astley Cooper as irritable testis is probably neuralgia of the spermatic plexus. In one case recorded by Brodie the testis became swollen and tender. This was from reflex neuralgia dependent on the passage of a calculus down the ureter.

Neuralgia of the *ovaries* is far from a rare affection, though difficult always to distinguish from pain in these organs due to local lesions. Its affecting one side only, its severity and occurrence in short paroxysms, are the most characteristic features. *Coccygodynia* also has most of the signs of a true neuralgia, but may be secondary to caries of the bone or malignant disease of the rectum or pelvis. Other forms of abdominal neuralgia, no doubt, exist; but it is almost impossible to distinguish them from the secondary nervous pains due to moveable kidney, renal calculus, gall-stones, or intestinal irritation.

Neuralgia of joints was many years ago described by Brodie. In former days, when surgical diagnosis was less perfect than now, limbs were amputated at the patient's request on account of constant pain in the knee or elbow; and when the joint was afterwards found to be perfectly healthy the case was described as neuralgic or hysterical. Some of these cases were probably due to reflex neuralgia; for instance, *gonalgia* dependent on disease of the hip-joint, or on femoral aneurysm. The pathology of such cases is set forth in Mr Hilton's masterly 'Lectures on Rest and Pain,' particularly in the fourth and the ninth.

The *girdle-pains* of transverse myelitis are to be compared to those caused by impaction of biliary or renal calculi, and are therefore secondary, not idiopathic, as are the pains of spinal meningitis, which depend on pressure on the posterior nerve-roots. But the "lightning-pains" of tabes are in all their characters true neuralgia, and they are not due to any demonstrable lesion of the nerves.

Crural and sciatic neuralgia.—There is no doubt that the anterior crural nerve, and more rarely the great sciatic or some of its branches, may be the seat of true neuralgia, marked by the characters enumerated above, and in addition by its occurrence in persons liable to facial or brachial neuralgia, by the absence of tenderness over the trunk of the affected nerve, and by the absence of trophic symptoms. Plantar neuralgia and metatarsal (so-called Morton's) neuralgia are probably most often due to flat-foot, and are best treated by local measures.

Among the neuralgias of the lower extremity should be included the cases of numbness and pain in the distribution of one lateral cutaneous nerve (usually the left) which have been described as *Meralgia paræsthetica*

Prognosis of neuralgia.—The duration of neuralgia and its amenability to treatment vary greatly. The most protracted and obstinate loca form is Tic Douloureux; most writers speak of it as incurable, and Trousseau mentions a case in which it went on for thirty years. The less severe cases of facial neuralgia seldom last more than a few months. The older the patient is, the more likely is the disease to be prolonged as well as severe. Neuralgia, however, does not appear to shorten life.

Recovery is generally gradual, and it is apt to be interrupted by relapses. Even after the pain is gone, the affected part sometimes feels stiff for a long time, and its muscles are quickly fatigued by exertion.

Treatment.—This often taxes one's patience and skill to the uttermost. Fresh air, regular exercise, plenty of sleep, an abundant supply of food, are essential. Anstie's dictum that neuralgia is the cry of a nerve for food conveys an important truth. Whenever he could he made his patients take cod-liver oil; if the stomach revolted at that, he insisted on their eating butter, Devonshire cream, or sometimes olive oil, or even cocoanut oil. He strongly objected to allowing sufferers from neuralgia to have wine or brandy, except in very moderate quantities, and with meals, and all experienced physicians concur in his repeated protests against prescribing stimulants for the temporary relief of pain.

Quinine in large doses is often of striking benefit, especially in trifacial neuralgia, and in cases which show periodic exacerbation, even when the patient has never lived in a malarious country.

Ammonium chloride in full doses is believed, in Germany, to be an efficient remedy in certain cases. The writer has seen it more useful in sciatica and lumbago than in pure neuralgia.

Iron has long been a trusted remedy in England. It should be given in as large doses as possible. Chalybeate waters and the milder preparations are useless. But saccharated carbonate of iron by the drachm and tincture of steel by 20 or 30 minim doses are often effectual.

Of all drugs, however, *arsenic* holds the first place, or the next after quinine, whether as the liquor sodæ arseniatis, which Dr Fagge preferred, or as Fowler's solution; it should always be given after food, well diluted, and the dose should be regularly increased until some physiological effect is produced.

While iron, arsenic, and quinine with fattening food are our best weapons in treating the condition which leads to neuralgia, we need also a drug to give immediate if only temporary relief. Fothergill gave conium with some success; but it is not a powerful enough anodyne for any but the milder cases.

When neuralgia particularly affects the lower jaw, and whenever it is connected with dental irritation, *butyl-chlor-aldehyde hydrate* ("croton-chloral") is always worthy of trial. It often unmistakably succeeds, and often as unmistakably fails.

Of late years *Antipyrin* (phenazone) has been much used for neuralgia, particularly facial neuralgia, and in many cases its effect is decided and complete. *Phenacetin* is another most valuable remedy, equal, or sometimes superior, to antipyrin. Gelsemium in full doses has also proved efficacious.

Local treatment, though only palliative, is worth trying in the less severe and inveterate cases. Neither hot water nor ice is of much service, but rubefacients when used over the painful points or spots of maximum pain sometimes give relief even in facial neuralgia. The local application of belladonna, or of chloral hydrate with camphor, or chloroform liniment, or menthol, often removes a slight attack. One of Sir Thomas Watson's well-told cases is that of a patient who was cured by an ointment of aconitine; and in the symptomatic neuralgia of the second division of the fifth nerve from the teeth, tincture of aconite applied locally has a most welcome benumbing effect on the pain.*

Galvanism is often useful in the treatment of neuralgia. One of the poles of a battery may be placed close to the spine of the affected side, or near the roots of the painful nerves, the other being applied upon the various "painful points" in succession. An "ascending" current is said to be most efficient. A good example of the effect of this form of electricity is afforded by a case related by Anstie in the fourth volume of the Clinical Society's 'Transactions.' A woman had for nearly five months suffered from cervico-brachial neuralgia, which lasted for several hours each day, and had defied all kinds of medicinal treatment. The first application of a battery of from ten to fifteen cells arrested the pain; the attack which followed was much less severe than usual; and after thirteen days the complaint ceased to recur. Another case in which great relief was afforded was read to the Clinical Society at the same time by Dr Buzzard. Faradisation of the nerve is rather injurious than beneficial, and the application of a powerful current to the skin by means of the electric brush is only a severe mode of counter-irritation.

Blisters should be placed, as Anstie recommended, not upon the painful part itself, but close to the spine at the level of the affected nerves, or a small blister may be put on the points of maximum pain with great relief for the time. Even in the desperate tic douloureux of old age, a blister applied to the nape of the neck sometimes removes the pain for a long time.

Opium.—In severe cases, when all treatment by local measures has failed, we must fall back on opium. One is loth to administer it when there is any hope of the ultimate subsidence of the complaint; but in cases of incurable tic douloureux Trousseau increased the dose boldly until sometimes two fluid drachms, or even half an ounce of laudanum was taken daily, and in a few instances the result was more than a palliation of the patient's misery. The frequency and severity of the attacks of pain were lessened, so that after a time the drug could be discontinued; and although a relapse occurred, the interval of ease was a great gain. Sleep had been obtained and food had been taken, which had before been almost impossible.

In many cases the administration of opium by the mouth disorders the digestive organs. Hence the subcutaneous injection of morphia is preferable; and this method of treating neuralgia has been extensively employed. The quantity should be very carefully regulated: not more than a sixth of a grain of the Pharmacopœial solution should be used on the first occasion. If relief is not afforded by such doses, larger ones may be tried; a quarter of a grain, half a grain, or even a grain. One seldom has to use the syringe oftener than twice in the twenty-four hours; but Anstie laid stress on the importance of performing the operation as early as possible after the commencement of an exacerbation of the pain. It seems to be a matter of indifference whether the morphia is injected at the seat of pain or at any other part of the body, except for the effect on the patient's imagination.

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* *Phosphorus* has been recommended, dissolved in a teaspoonful of cod-liver oil, one-twelfth of a grain every four hours. Dr Ashburton Thompson reported fifty cases of neuralgia treated by phosphorus, in several of which the effects of the remedy seem to have been remarkable; but he admitted that if decided results are not attained within three days it is useless to persevere.

Subcutaneous injection of morphia is the most rapid method of using opiates; but except for this advantage it is less adapted to neuralgia than is laudanum. There is fear of making the patient an opium-eater; but the habit of subcutaneous injection of morphia is to that of taking laudanum like intemperance in ardent spirits to intemperance in wine.

The patient gradually falls into a state of depression and irritability, which can be relieved by nothing but a fresh dose. Even after the original disease is relieved patients sometimes find great difficulty in discontinuing injections of morphia to which they have been accustomed; and if they are to succeed, must endure much discomfort and even misery for several days. For this reason, as well as for others, the syringe should not be entrusted to the hands of the sufferer himself, nor even to those of a relative or servant, unless perhaps in incurable cases.

Operation.—Formerly it was the practice to divide the infra-orbital nerve in severe cases of neuralgia; and, when the relief was found to be only temporary, to excise a piece. This operation was performed on Dr Pemberton by Sir Astley Cooper early in the century without any benefit, and the present writer saw it done by Nélaton in 1863. It might have been thought from experiments on animals that the cut ends of the nerve could be turned back and sutured so as to make reunion impossible. But probably the formation of bulbous ends at the stump would be an additional source of failure; and there is the possibility of a neuritis above the point of section or even of a central origin of the neuralgia in some cases.*

Surgeons have long been convinced that neither section nor excision of the nerve is of permanent service. Of late years, however, Fedor Krause in Germany, Dr Hartley in America, and Mr Horsley in England, have successfully carried out in about fifty cases the formidable operation of opening the skull, lifting out of the way the temporo-sphenoidal lobe, and removing the Gasserian ganglion with the sensory root of the fifth nerve. Only one fatal result appears to have been reported; and the neuralgia appears not to have returned.

SCIATICA.—The common affection known as sciatica (*passio ischiadica*) differs so markedly from true neuralgia in its clinical course, its pathology, and its treatment, that, although most writers have regarded it as such, the present writer would put it on a different basis as a true Neuritis. The part of the nerve which most commonly suffers is in the upper half of the thigh, but often the whole length of the trunk, the small sciatic over the buttocks, and both the popliteal nerves with their digital branches are involved. Occasionally it may be limited to the back of the knee, or to the terminal twigs in the calf. The symptoms of sciatica differ widely from those of tic douloureux. Instead of paroxysms of acute pain, darting like lightning through the nerve, and separated by intervals of comparative freedom, there is a constant, heavy, gnawing sensation; its intensity is not uniform, but the variations are comparatively slight and uncertain; and if it runs along the affected nerve at all, it travels slowly. It often appears to be worse at night.* Few diseases cause more peevishness, restlessness, and misery, spoiling the appetite, perverting sleep, and interfering with every kind of work, as well as with most amusements and all athletic sports.

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* See Mr Bowlby's Astley Cooper prize essay on "Diseases and Injuries of Nerves, and their Surgical Treatment" (1886).

The pain is increased by pressure on the sacral plexus within the pelvis, or at its exit from the sciatic notch, and thus defæcation may be attended with much suffering. The patient is afraid to sneeze or cough, and suffers from any sudden movement, especially from stooping.

When sciatica has lasted long tender points are generally to be found; one is situated where the nerve emerges from the pelvis. The whole length of the nerve, however, becomes most sensitive to pressure, and especially that part of the external popliteal branch which lies under cover of the biceps tendon.

Wasting of the muscles is another symptom which supervenes in chronic cases of sciatica. The buttock becomes flattened and flabby, the muscles of the thigh and calf are reduced in size, and feel soft and flaccid. From the first the patient is compelled by the pain to limp in walking, so that he puts only his toes to the ground, and is glad to make use of a stick. In severe cases he lies on the sofa all day long, with every joint of the limb bent, dreading to make the slightest movement. Painful cramps in the flexor muscles of the toes may come on, especially when the patient is just falling off to sleep.

A certain degree of anæsthesia or impairment of tactile sensibility appears to be almost constant in the more severe cases of sciatica, and sensations of numbness and tingling are often present. The affected limb is sometimes colder than the opposite one.

It will be seen from this account that sciatica differs in the character of the pain and in other clinical features from those given on pages 554—5, which particularly apply to tic douloureux. If we mean by sciatica to denote any painful affection in the course of the sciatic nerve, we must admit that its pathology is very varied. But if we confine the term to primary neuritis, it is most important that we should not confound the true disease with the effects of pressure upon the sacral plexus and sciatic nerve.

In the 'Guy's Hospital Reports' (vol. x, 1864) a case was published that was regarded as an example of sciatica until the patient unexpectedly died; when a large aneurysm was found upon an abnormal artery, which passed down through the sciatic notch and along the back of the thigh. That case is probably unique, such a course of the vessel (which is normal in birds) being one of the rarest of abnormalities in the human subject; but a precisely similar pain might be caused by an aneurysm upon one of the regular arteries, or by a sarcomatous growth pressing on the nerve.

It has been supposed that sciatica may be due to pressure of impacted scybala upon the nerve of the left side; and the result of emptying the sigmoid flexure is said to prove the fact. But in cases which occasionally are met with of pain from pressure during labour or from retroverted uterus or ovarian tumours which have fallen into the pelvis, the pain is accompanied with more decided numbness and paralysis than attend ordinary sciatica.

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* This was ascribed by the late Dr Henry Lawson (himself a sufferer from sciatica) to the fact that the patient then has less to distract his thoughts than in the day.

The relation of sciatica to rheumatism is altogether imaginary, if any definite meaning be attached to the word "rheumatism;" but there is a kind of sciatica which accompanies lumbago, and probably owns the same cause. In fact, myalgia is the natural ally of sciatica, and occurs in the same kind of patients and often in the same individual.

There is no doubt that occasionally sciatica is due to gout—probably to local gouty neuritis. We had a marked case of this kind in Philip Ward in February, 1887; a man of thirty-seven, who had suffered repeatedly from hereditary gouty arthritis in the foot, was the subject of severe sciatica of the left side. He had been treated in various other ways without any benefit, and was so quickly and effectually relieved after taking colchicum that one could scarcely doubt the justice of the diagnosis.

Again, some cases of sciatica occurring in syphilitic patients and becoming worse at night are speedily relieved by iodide of potassium. Here, as in cases due to gout, the pain probably depends upon neuritis of the sciatic nerve.

The writer has noticed a frequent connection between sciatica and uric acid crystals in the urine, and also with interstitial nephritis. All these associations are with chronic or subacute inflammation, not with true neuralgia.

Inheritance plays no important part in the ætiology of sciatica, but there is no doubt that it is often traceable to cold. Lawson believed that nine tenths of all cases of sciatica are referable to this cause, and Erb affirms almost as much. Getting wet through and sitting on damp ground are so often followed by sciatica that few can doubt their influence, and sciatica is one of the evils caused by an East wind.

The complaint sometimes follows a strain or severe and unwonted exertion; it is said to be common in those who work at a sewing machine.

Sciatica is more common in *men* than in women (according to Erb, in the proportion of four to one), and adults from thirty to sixty years old are most subject to it. Lawson saw one well-marked case in a boy aged fourteen, who was addicted to masturbation.

The question arises, if the ordinary chronic continuous sciatica be no true neuralgia but a neuritis, why do its symptoms, ætiology, and natural history differ as much from those of multiple peripheral neuritis, to be treated later in this chapter, as they do from those of tic douloureux as described in its earlier pages.

The answer seems to be that the seat of the lesion in sciatica is the vascular connective tissue of the nerve-trunk, its perineurium, and that this by its inflammatory swelling presses on the sensory fibres enough to give pain but not enough to abolish their power of conduction. Whereas in cases of peripheral neuritis we shall see that the nerve-fibres themselves are degenerated with the usual physiological results of such degeneration, more or less complete paralysis of motion and sensation, abolition of reflex unctions and secondary wasting of the muscles.

The *diagnosis* of sciatica is seldom difficult to the practitioner who first searches for the numerous other diseases that may cause pain in the thigh and leg. Where the calf is the part mainly affected one must think of hrombosis of the femoral vein, make digital examination of the vessel at he groin, and look for œdema of the ankle. Disease of the hip-joint can e put out of consideration by the fact that neither pressure on the trohanter, nor forcing the head of the femur against the acetabulum, nor extreme flexion, gives rise to pain; and disease of the sacro-iliac synchondrosis by the fact that no tenderness is elicited by tapping the articulation, or by pressure of the two ilia together. Advanced cases, in which the patient limps in walking and has wasting of the muscles, are very likely to be mistaken for spinal disease; this occurred some years ago in the case of a medical man whose fellow-practitioners in his own neighbourhood all felt sure that he had disease of his vertebræ. Even if one is satisfied as to the seat of the pain, one must still search carefully for local causes of irritation. In one instructive case a surgeon, feeling carefully along the course of the nerve, was fortunate enough to detect the presence of a piece of broken needle, the removal of which led at once to the cure of the patient.

Treatment of sciatica.—In treatment, as in symptoms, sciatica has little likeness to true neuralgia. It is not benefited by quinine, and not often by either steel or arsenic. Far more beneficial is calomel or blue pill every or every other night, followed by a saline laxative draught every morning. After this guaiacum is a very useful drug, given in as frequent doses as the patient can be induced to take. Locally the application of a long blister from the fold of the buttock down the back of the thigh is perhaps the most effectual measure. Liniments are of little use, less than in lumbago, but the writer has sometimes seen relief from the aconite, belladonna, and chloroform liniment.

The practice of cutting down on the sciatic trunk and forcibly stretching it, and the milder plan of stretching the nerve by flexing the thigh on the pelvis while the knee is extended, have both been extensively tried; first extolled, and since neglected. A galvanic current, so slowly interrupted as to throw a segment of the nerve for a minute or two into anelectrotonus, is certainly sometimes a valuable means of treating sciatica.

For immediate relief the deep injection of morphia over the nerve is the most effectual treatment, much more effectual than in facial neuralgia. Not infrequently we see suffering which had been almost intolerable thus removed, as by magic, within a few minutes. And this is sometimes the first step towards the complete cure of the disease. It was so, for instance, in the severe sciatica of which Dr Lawson was himself the victim, and in which other plans of treatment had been tried without the least good result. A similar local injection of cocaine is also sometimes useful. The needle should, if possible, be passed into the nerve itself.

Anstie believed that the evils connected with the establishment of a morphia habit never arise when the dose is kept below a certain point. He insisted strongly on the importance of "economy" in the use of morphia, and he related the case of a lady, who for three years had enjoyed a complete immunity from severe sciatica under the daily use of a dose which was at first one twelfth of a grain, and was never raised above one fourth.

Paralysis of Spinal Nerves.—In studying the causes and symptoms of paralysis from lesions of the nerve-trunks generally, we will take the nerve of the arm for the example, because the movements performed by its severa muscles are more distinct than those of the individual muscles of the leg and hence their impairment is more easy of determination.

Injuries of the *brachial plexus* may be followed by loss of movemen and feeling in the whole upper limb. The surgeon sees cases in which thi occurs as the result of severe accidents in which the shoulder-joint is dislocated, or the humerus or scapula fractured. But similar symptoms not infrequently follow pressure upon the nerves, or an injury so slight that its occurrence, especially in a child, may be overlooked. Thus Paget ('Med. Times and Gaz.,' 1864) mentions the case of a boy whose left arm, while he was still an infant, was violently pulled by a little brother; the limb was powerless for long afterwards, and it remained permanently weak and much smaller than the other one. The employment of force to reduce a dislocated shoulder has sometimes been attended with like consequences; so also the pressure of a crutch, tying the arms of a prisoner with a cord, or carrying, as the water-carriers of Rennes do, large vessels with handles, through which the arm is passed. "Crutch paralysis" is not uncommonly seen in hospital practice in London; and the patient is often unaware of its cause. A similar effect may also be produced by lying on one side with the weight of the body resting upon the arm; or, more often, by sleeping with the arm across the back of a chair. In most instances of this kind the sensibility of the patient has been blunted by drink, and hence they used to be known in the hospital as "Sunday morning" paralysis. When the cause is doubtful the axilla and neck must be carefully examined, lest an aneurysm, exostosis, or other tumour should be overlooked. Some writers believe that the brachial plexus is sometimes affected by external cold, as we shall presently see to be the case with the facial nerve; but this is very doubtful. Salter related ('Guy's Hosp. Rep.,' 1868) two cases, in each of which caries of a wisdom-tooth, beside causing severe pain in the arm, led also to partial "reflex" paralysis of the muscles, so that the patients could not grasp objects, nor raise the hand to the head, nor hold a fork at dinner. In both cases all the symptoms vanished as soon as the tooth had been extracted, or a few hours later.

It is exceptional for the whole of the brachial plexus to be paralysed at once by any of the above causes; and the loss of power may often be limited to the muscles supplied by a single nerve. In such cases the interpretation of the symptoms is sometimes so obvious that no anatomist could possibly misunderstand them. But sometimes they require careful study.

Special motor paralyses.—A remarkable isolated paralysis is that of the serratus magnus muscle. The *long thoracic nerve* which supplies it from the fifth and sixth cervical roots, leaves the plexus at so high a point that it is never involved when the cause is the pressure of a crutch, or sleeping upon one arm. But it may be affected by a blow; by carrying a heavy weight upon the shoulder; or by strain of the shoulder muscles, as in the case of mowers, puddlers, cobblers, or ropemakers.

The principal symptom is an alteration in the position of the scapula. Even when the shoulder is at rest, with the arm hanging down, the scapula, as a whole, is seen to be drawn slightly upwards and inwards, and has its inferior angle tilted, so as to be nearer to the vertebral column than the rest of its posterior border. Moreover this posterior border stands off a little from the part of the back on which it should rest, forming a "wing-like" (pterygoid) projection. These peculiarities are greatly exaggerated when the patient is told to perform certain movements. Thus if he is directed to lift his arm straight over his head, he finds himself unable to raise it above the level of his shoulder, because the necessary rotation of the scapula by the serratus cannot be effected; but when once another person has moved the shoulder-blade to the proper position for him and fixed it there, he acquires the power of lifting the limb to whatever height may be desired. During the attempt to raise the arm, the scapula, if left to itself, moves further than ever towards the spine; and where both serrati happen to be paralysed together the two bones may actually come into contact at their inferior angles. If the raised arm is brought forwards the "wing-like projection" is increased, and the posterior border of the scapula stands off so as to leave a deep hollow. Another symptom, according to Dr Poore ('Clin. Soc. Trans.,' viii), is an alteration in the shape of the chest on the affected side, as shown by the cyrtometer. He finds that in a healthy person, when the arms are thrown horizontally forwards, the chest becomes flattened in front, and its transverse diameter is increased. Where there is paralysis of one serratus no widening occurs on that side.

More frequent is paralysis of the deltoid and other muscles supplied by the *circumflex* nerve. This, too, may result from direct injury to the shoulder. According to Erb it is sometimes caused by "rheumatic" affections of the joint. One must, however, bear in mind that osteoarthritis (to which Erb probably refers) is commonly attended with destruction of the long tendon of the biceps, and that unless this structure is perfect the arm cannot be raised; while inability to lift the arm from the side is also the principal symptom of paralysis of the deltoid. It may be thought that a sufficient proof of paralysis would be found in atrophy of the muscle. But a considerable degree of wasting may be the result of mere disuse when the joint is diseased.

Paralysis of the *musculo-cutaneous* nerve is indicated by inability to flex the elbow-joint, the biceps and brachialis anticus muscles being paralysed. It must be borne in mind that the supinator longus (which receives its supply from another source) is also a flexor, and the patient must therefore be told to keep his hand supinated while the power of the other muscles is being tested.

Of the three nerves which send filaments to the hand and fingers, the *musculo-spiral*, derived from the fifth to eighth cervical roots, is much more apt than the other two to be separately affected. In a considerable proportion of the cases of paralysis due to the pressure of a crutch, or to lying on one side during the deep sleep of intoxication, the musculo-spiral nerve suffers alone; and as in the latter case the triceps often escapes, it is probable that the nerve undergoes compression where it is winding round the humerus, and after it has given off the branches to that muscle. The extensors of the wrist, of the two joints of the thumb, and of the metacarpo-phalangeal joints of the fingers are the chief other muscles supplied by this nerve; and accordingly one symptom of paralysis of it is a flexed condition of these joints, with inability to straighten them; the two remaining joints of the fingers being nevertheless capable of extension by the interossei and lumbricales, provided that the metacarpo-phalangeal joints are held up by another person. Another consequence of the loss of power in the extensor muscles of the wrist is impairment in the extent to which the fingers can be *flexed* upon the palm. The paralysis of no other nerve interferes so much with the motions of the hand as that of the musculo-spiral.

Isolated paralysis of the *median* nerve (three last cervical and first dorsal roots) is more often due to a wound or injury at the elbow, in the forearm, or at the wrist, than to any affection of the brachial plexus above.

The symptoms in the case of the *median* nerve, are inability to use the flexors of the wrist and fingers, the metacarpo-phalangeal joints being alone capable of flexion by means of the interossei. Indeed, the excessive action of these muscles is apt to cause an over-extension of the two phalangeal joints of the fingers, and especially of the index-finger.

When the *ulnar* nerve, from the last cervical and first dorsal roots, is paralysed the muscles of the little finger and the interossei are the most affected. The fingers cannot be abducted nor adducted, and the patient is unable to flex the metacarpo-phalangeal, or to extend any of the phalangeal joints of the fingers. There being nothing to antagonise the traction of the extensor and flexor muscles, the joints in question become distorted, so that the last two phalanges are over-flexed, while the first phalanges, on the other hand, are over-extended. Accordingly, the knuckles are replaced by hollows on the back of the hand, while they project towards the palm, where the interossei and the two inner lumbricales are atrophied. The fingers of a hand so affected are often compared to the claws of a griffin—*main en griffe, Klauenhand.*

Deformities may be produced by contraction of the muscles which are physiologically the antagonists of those that are paralysed. This often complicates the symptoms of peripheral paralysis. Moreover, after the muscles have been long paralysed they may waste and shorten, so as to produce distortions precisely opposite.

Erb's paralysis.—The anatomical distribution of the nerves is not strictly followed in all cases of peripheral paralysis, and sometimes cases of combined paralysis are observed. The most frequent of these was first observed by Erb. It consists in loss of power in the deltoid, biceps, brachialis anticus, and supinator longus, dependent perhaps on affection of the sixth cervical nerve where it is emerging from between the scaleni. In such patients the arm hangs by the side, and the elbow cannot be flexed. A precisely similar affection was described by Duchenne in newly born children, as the result of traction or pressure upon the shoulder during birth; and Erb also has seen cases of this kind.

Ascending neuritis.—In exceptional cases a morbid process beginning in a single nerve at a particular spot may pass upwards along that nerve, and afterwards extend to others (*neuritis migrans*). Thus Weir Mitchell relates the case of a man who received an injury to the nerve which supplies the pectoralis major; on the third day he was attacked with pain in the course of the median and musculo-cutaneous nerves, and the muscles to which they are distributed subsequently became paralysed and wasted. Another patient bruised his ulnar nerve at the bend of the elbow, and about five months afterwards the flexor muscles of the wrist became affected with paralysis, those supplied by the median nerve as well as those supplied by the ulnar. These, however, were probably examples of peripheral neuritis.

The reaction of degeneration.—Cases of local paralysis due to lesions of the nerve-trunks are all examples of "peripheral paralysis," and the electrical reactions of the affected nerves and muscles present characteristic modifications. These were first minutely studied by Erb and Ziemssen.

Let us suppose that a nerve-trunk is cut across. The result is that throughout the whole length of the nerve and its branches below the affected spot (so far as one can test them without at the same time stimulating the muscles) its excitability undergoes a progressive diminution, both to the make and break of galvanic currents, and to the rapidly alternating faradic currents from an induction coil. In the course of the second week it is entirely extinguished, so that not the slightest contraction can be produced by the application of either form of electricity to the nerves. But the reactions of the paralysed muscles are very different. They, too, fail to respond to a faradic current; but when a galvanic current is employed they contract much more readily than under normal circumstances to the stimulus of opening or closing. The method of determining this fact is first to ascertain what number of cells of the battery are required to excite contractions in the corresponding muscles of the healthy limb; we then perhaps find that only half that number is needed to excite contractions in the muscles supplied by the injured nerve. Indeed, they can often be thrown into vigorous action by a current which is too feeble to be felt.

Moreover there is what is termed a "qualitative" change, *i. e.* the order in which the two poles excite contractions is altered. According to Pflüger's law, stimulation depends upon rise of catelectrotonus or fall of anelectrotonus (*i. e.* in either case the passage of a segment of nerve from a lower to a higher degree of excitability); the descending current has greater exciting power than the ascending, and closing or making than opening or breaking the galvanic current. Accordingly, the order in which currents of successively increased strength produce contraction in the muscle supplied by the motor nerve stimulated as follows:[*]

	CC	AO	AC	CO
Weak current causes . .	c			
Strong current causes . .	C	c	c	
Very strong current causes	C′ or T	C′	C	c

The rule may be shortly remembered thus:—With increasing strength of galvanic current the order in which contractions appear is C.C.C., A.O.C., A.C.C., C.O.C.[†]

[*] Here C.C.c. means that a small Cathodal Closing contraction is produced by a weak current. The current is descending, and the stimulus is the rise (in this case from zero) of catelectrotonus on closing (making) the galvanic circuit. A.O.C. means that an ascending galvanic current produces an Anodal Opening (breaking) contraction, the stimulus being the fall of anelectrotonus (to zero). A.C.c. or A.C.C. means that making contact again with the ascending current will, if it is as strong or stronger, produce a slight or a marked Anodal Closing contraction. Lastly, C.O.c. means that breaking contact with the descending current will, if it is strong enough, act as a slight stimulus and produce a Cathodal Opening small contraction.

[†] The German symbols used by Erb are Ka. for Cathodal, An. for Anodal, S. (Schliessung) for closure, O. (Oeffnung) for opening, and Z. (Zuckung) for contraction. These are wholly or partly adopted by some English writers. If it were important to avoid using the same letter for different words, it might be done by spelling Cathode with a K, and using Break and Make instead of close and open. The normal sequence would then stand: K.M.C., A.B.C., A.M.C., K.B.C. But in such matters whatever is most generally used and best understood is best.

It may be added that c. means a weak contraction, C′. a strong one, and T. a tetanic contraction, that > and < mean respectively *greater than* and *less than*, and that R.D. means the Reaction of Degeneration.

When, however, a muscle is paralysed by severance of its motor nerve in any part of its course, or by destruction of its anterior root, or of its corresponding large ganglion-cells in the anterior cornu, and when this has lasted for a week, not only is the muscle less excitable when the "motor point" of its nerve (*i. e.* the place where it becomes superficial and is most favourably stimulated) is excited by the induced (faradic) current, not only is it *more* excitable when the same point of its nerve is stimulated by the make or break of the direct (galvanic) battery current, but the above physiological order is altered.

So far from contraction at the moment of closure with the cathode (C.C.C.) being now the most marked effect, there is often as much or even greater contraction when the circuit is completed with the anode; while instead of cathodal opening contractions (C.O.C.) being the last of all to appear, they may precede the anodal opening contractions (A.O.C.). Accordingly the normal order for contractions with currents of increasing strength given in the above table is altered, and instead of C.C.C., A.O.C., A.C.C., C.O.C., we have A.C.C. before C.C.C., and C.O.C. before A.O.C.; *i. e.* the anodal closing contraction is for equal currents stronger than the cathodal, and the cathodal opening contraction is for equal currents stronger than the anodal.

Lastly, the contractions themselves are slow and protracted, and resemble tonic spasms rather than the short clonic movements which occur in healthy muscles when stimulated by galvanic currents.

This changed electrical condition of the paralysed muscles and nerves is called the Reaction of Degeneration.

Little progress has hitherto been made towards the explanation of these remarkable facts. The seeming paradox that muscles insensible to powerful faradic currents should nevertheless respond to very weak galvanic currents has, indeed, been accounted for by Neumann, who has shown that it depends upon their requiring a current to pass for some length of time before they can react to it. Momentary galvanic currents, even when of considerable strength, are as inoperative as faradic currents themselves. But why the muscles should be incapable of responding to currents of brief duration, and why they should be sensitive to weaker galvanic currents than under normal conditions, is hard to say.

This, however, is established, that the peculiar reaction in question, as well as the loss of electrical irritability in the trunk of the nerve below the seat of injury, is coincident with and indicative of a remarkable series of degenerative changes which affect the structure of the paralysed nerve. Within a few days after section of a nerve-trunk, the whole length of the nerve below is found to be greatly altered: the medullary sheaths of the fibres break up into fatty granules, the nucleus of the nerve segment proliferates, and finally the axis-cylinders are destroyed. At the same time the perineurium everywhere undergoes a greatly increased development; it becomes crowded with corpuscles, and these develop into spindle-cells and fibres, so that the nerve-trunk becomes denser and harder than before. (See the account in Dr Mott's "Croonian Lectures," 'Lancet,' June, 1900.)

In the muscles, also, degeneration takes place. The fibres diminish in size, and their striation becomes indistinct; but their nuclei multiply, and the perimysium is crowded with cells, which after a time undergo development into fresh fibrous tissue. If the injury to the nerve should remain unrepaired the muscular fibres waste still further, and at length the whole substance of the muscles is converted into a whitish-yellow, flattened mass of dense fibrous tissue, in which no striated fibres can be discovered. It often includes in its interstices a large number of adipose cells.

In some way these changes in nerves and in the muscles which follow depend upon the separation of the affected structures from the nervous centres, in accordance with the law named after its discoverer, the late Dr Waller. The reaction of degeneration is not confined to the paralysis from injury to peripheral nerves now under consideration. We shall hereafter see that it accompanies paralysis dependent on lesions of the motor ganglion-cells of the anterior cornua of the cord and of their homologues in the bulb and mesencephalon. If, therefore, we speak of it as characteristic of "peripheral paralysis," we must extend the meaning of the phrase. The reaction of degeneration and the histological changes in the nerves which follow it are indicative of *severance of the nerve-fibres affected from their trophic centres, either by destruction of these ganglia, or by severance of the nerve in its course.*

It must not be supposed that the commencement of degenerative changes in the peripheral nerves is a proof that the injury of the trunk above is permanent and irremediable. On the contrary, nerves possess extraordinary powers of regeneration. Even after the muscles have for several weeks exhibited the reaction of degeneration it often happens that the connection between their nerves and the spinal cord is at length gradually restored—apparently by outgrowth from the severed ends of the axis-cylinder. This occurs most readily in the case of sensory fibres. Indeed, the above account applies chiefly to motor nerves, in which alone, of course, there can be the full development of the reaction of degeneration. Even in a motor nerve it is only when the original lesion is of a certain degree of severity that the reaction of degeneration presents itself. In the slighter forms of paralysis, such as that due to the pressure of a crutch, or to compression of the brachial plexus or musculo-spiral nerve during sleep, both the muscles and nerves often retain their normal irritability both to faradic and to galvanic currents.

Period of the reaction.—When a motor nerve is completely severed (to take the simplest form of peripheral paralysis), voluntary motion is, of course, at once and completely lost. For about the first week both muscle and nerve are less and less susceptible to all stimuli; but after a few days—while the galvanic and faradic excitability of the muscle, and the faradic excitability of the nerve continue to diminish, until after two or three weeks they are entirely lost—the galvanic excitability of the motor nerve rapidly increases with the other phenomena which accompany degeneration of the nerve-fibres, and atrophy with degeneration of the muscle. In cases which recover, the faradic excitability of muscle and nerve returns as voluntary motive power is re-established, and the abnormal galvanic reactions of the nerve more gradually disappear. In cases which do not recover, the reaction of degeneration continues, though in slowly diminishing degree, for many weeks or months, until at last it also is lost, and the atrophied muscle no longer responds to any kind of stimuli.

The impairment of sensation is with few exceptions—and those chiefly cases of functional paralysis—far less complete than that of motion; and recovery is far more rapid in the case of sensory than of motor paralysis. Probably this depends on a motor fibre having only one trophic centre, whereas a sensory fibre has not only its ganglia in the cerebro-spinal axis, but also its end-organ.

Anæsthesia.—The sensory as well as the motor fibres of compound nerves may be impaired by the same lesions. Peripheral paralysis may completely extinguish the transmission of tactile impressions, and may likewise render the patient unable to feel heat or cold, or the make and break of a galvanic current. This loss of common sensation is termed Anæsthesia; the incapacity to feel pain is distinguished as Analgesia. They do not always accompany one another. As the result of syringomyelia (*infra*, p. 632) for instance, we observe, without the slightest impairment of the sense of touch, great diminution in the susceptibility to pain. One patient of Dr Weir Mitchell only felt a slight pricking sensation when a large needle was run into the palm, and another was scarcely sensitive to the electric wire-brush. This is a most delicate test of sensory perception. The part to which it is applied must be perfectly dry, so as to oppose the current passing through to the underlying muscles and confine its effects to the skin; the numerous minute points of the dry brush help to the same result. Mitchell says that if no sensation can be produced by it, one may generally conclude that the loss is absolute; but that on several occasions it has saved him from despairing of cases which had before seemed hopeless.

From the extreme of anæsthesia there are all possible degrees to a point at which very careful observation is necessary to show that sensation is not perfect. The best way of detecting very slight anæsthesia is to bring the tip of a feather gently upon the surface of the skin, or to touch a single hair with a needle or the edge of a knife. If the patient can feel this on one side but not on the other, sensation is to some degree impaired in the latter.

A pair of compasses may be used after E. H. Weber's method, the object being to determine at what distance from one another the points are felt to be distinct. This "limit of separate perception" varies greatly in different parts of the skin under normal conditions, from 2·25 mm. on the extremities of the fingers to 77 mm. on the arm. But for each region there is an average, and any considerable variation may be taken as proof of some loss of sensibility. Or a direct comparison may be made between the opposite sides of the body in the patient whose case is being investigated. An improved form of the instrument is sometimes employed, in which the two limbs of the compasses slide upon a graduated bar; this is called the "æsthesiometer." Dr Weir Mitchell teaches that, to ensure the most correct results that are attainable, one must cover the patient's eyes, keep the part which is to be tested perfectly at rest, and apply the compass points (which should be rounded) lightly upon the surface of the skin, but with equal force, at exactly the same time, and in a line bearing a definite relation to the axis of the limb. Experience has shown that it is useless to base precise numerical statements upon Weber's method, for widely different results may be obtained from the same spot at an interval of a few minutes. All we can obtain are relative results, but these are after all what we most want.

Clinically we find that when a mixed nerve-trunk is paralysed the loss of sensation is almost always less than the loss of motion. Nay, often there is but little anæsthesia when the loss of power is complete; or sensation may be quickly regained while the motor power is being slowly recovered. Probably the sensory centres are readily affected by few nerve-fibres and by very feeble impulses, whereas a muscle is unable to respond unless the stimulus is conveyed to it by many nerve-fibres at once. As Mitchell remarks, this accords with the well-known fact that by irritation of the ulnar nerve at the elbow the sensation is far more readily excited than motion.

Lesion of a mixed nerve-trunk seems never to produce anæsthesia unaccompanied by motor paralysis.

Beside degrees of anæsthesia there are certain perverted sensations, conveniently grouped together as *paræsthesiæ*—numbness, tingling, burning, &c. These, however, except numbness or "pins and needles," which is the most constant effect of moderate compression of a nerve, are less frequent and less characteristic in cases of injury or disease of nerve-trunks than in spinal and cerebral diseases, of which we shall treat hereafter.

Pain.—A third symptom of paralysis of the nerve-trunks is pain. It is by no means constantly present, and is wanting in most cases of local peripheral paralysis. It shows the features of neuralgia, and is referred mainly to those parts of the skin to which the diseased or injured nerve is distributed. Occasionally the trunk of the nerve may in thin persons be felt to be swollen and tender. In some cases it is periodic, returning every day at about the same hour, generally towards the latter part of the day.

A special variety has been named Causalgia (καῦσις, cautery), on account of its burning character. This may vary in severity up to the most unendurable agony, which the patient compares to a "red-hot file rasping the skin." In cases of peripheral paralysis due to wounds this kind of pain seldom, if ever, comes on until the process of cicatrisation is beginning. Its most frequent seat is—in the upper limb, the palm of the hand; in the lower limb, the instep. Heat aggravates it, and allowing the limb to hang down has the same effect. It is relieved by moistening the skin, and two of Mitchell's patients always carried a bottle of water and a sponge, and would not allow the affected part to become dry even for an instant. Others found some ease in walking by pouring water into their boots. In course of time the severe suffering may lead to a condition like hysteria, with hyperæsthesia of the whole body.

This peculiar kind of pain seems never to occur in parts which have been entirely deprived of sensation by complete division of their nerves; nor does it accompany the subjective sensations that are referred to the distal ends of limbs which have been amputated. From these facts Mitchell argues that its immediate cause is some change in the peripheral extremities of the nerves, rather than in the trunks above.

Trophic changes.—The parts to which the pain of causalgia is referred are liable to a special change, to which Sir James Paget first gave the name of *Glossy Skin* ('Med. Times and Gaz.,' 1864). The surface shines, as though it had been varnished. In Paget's words, "the fingers"—for they are most often affected—"become tapering, smooth, hairless, almost void of wrinkles, glossy, pink or ruddy, or blotched as if with permanent chilblains." An eruption of vesicles is also common; or bullæ may form, which afterwards ulcerate; or painless whitlows, leading to shedding of the nails. Or, the nails may become arched and incurved, and the skin may shrink away so as to leave the sensitive matrix exposed.

Parts which are the seat of causalgia are slightly *warmer* than those on the opposite side of the body; whereas the general effect of injuries of the nerve-trunks is to lower the temperature—in some cases of Hutchinson's by as many as eight or ten degrees Fahrenheit.

Zona, or shingles, is the name given to a remarkable affection of the skin analogous to these results of lesions of trophic nerves. It will be considered in the last section of this work, among Diseases of the Skin.

Another occasional result of morbid conditions of the nerves of a limb is a painful *swelling of its articulations*, which may go on to anchylosis, and greatly increase the deformity and the disablement experienced by the patient. The arthritic effects of injuries to nerves have been well illustrated by Mitchell; they are analogous to the affection of joints in cases of Tabes, described as the arthropathy of Charcot.

Erb also mentions, as sometimes following paralysis of the musculo-spiral nerve, a thickening of the *tendons* at the back of the wrist—a circumscribed painless swelling, as large as a hazel-nut, probably due to their having been exposed to mechanical strain by the flexed state of the joint.

These various conditions are generally due to peripheral neuritis with degeneration of the nerve-fibres. Paget recorded two cases in which glossiness of the fingers developed itself as the result of neuralgia after shingles; Mitchell relates one in which it was due to chronic myelitis; and many years ago the late Dr Moxon had in Guy's Hospital a remarkable case in which a precisely similar state of the fingers was caused by the pressure of a mediastinal tumour on the intercostal nerves.

Diagnosis.—The recognition of local peripheral paralysis is seldom difficult for a good anatomist. Sometimes it might be mistaken for progressive muscular atrophy in an early stage, particularly if the deltoid muscle should be alone affected, or the interossei and the muscles of the little finger. The application of galvanic and faradic currents to the paralysed muscles would clear up the doubt in some cases, but not in all.

When we have ascertained that the paralysis is of peripheral origin, one must search for all possible causes of compression of the nerves. Some years ago a woman came to Guy's Hospital complaining of numbness and loss of power in the right arm, which symptoms (she said) had already lasted six months; she was found to have a cancerous growth in the breast, which had extended to the axilla, and involved the nerves of the brachial plexus.

In the lower limbs the mistake most likely to be made is that of attributing to disease of the spinal cord a paralysis really due to a pelvic tumour compressing the sacral plexus, or to a mass involving branches of the lumbar nerves. In 1876 a woman died in Guy's Hospital who for ten years had suffered from paralysis of the sphincters of the bladder and rectum, and for seven years from loss of power and sensation in the legs below the knee-joints, so that she was unable to walk, and could only crawl about on her hands and knees. The cause was found to be a large abscess in the pelvis, through which the cords of the sacral plexus ran; it depended on caries of the bone, and could probably have now been recognised and successfully treated.

Prognosis.—Most of the cases of paralysis from lesions of the nerve-trunks that come under the care of the physician get well sooner or later;

and he can, by treatment, hasten their recovery. Thus, crutch-paralysis commonly passes off in a week or a fortnight; that which follows compression of the nerves during sleep more often lasts from four to six weeks, and it may be prolonged for several months; that which is caused by severe stretching or bruising of the nerves, generally lasts more than a year, and may never be entirely removed. Even after very long periods improvement sometimes takes place. Mitchell says that only the careful notes previously made could have convinced him of the extent to which function had been restored, in some of the worst cases, at the end of three or four years.

Valuable aid in the prognosis is afforded by an examination of the electrical reactions of the affected parts. In cases which are to terminate rapidly in recovery, the muscles and nerves retain their normal irritability; in those which are to run a protracted course, the muscles present the reaction of degeneration; while in cases which are incurable, susceptibility to every form of galvanic stimulus is extinguished.

Treatment.—Electricity is useful, not only as a guide to prognosis, but also in treatment. There is still much uncertainty as to how it acts, but all writers are agreed that it is beneficial. Sometimes its effects are extraordinary. After a single application of faradic or galvanic currents the patient may suddenly find that he can perform movements which he had for weeks or months been unable to accomplish. In such cases it may perhaps be assumed that the regeneration of the affected parts was already far advanced—that they were, in fact, on the brink of recovery—independently of any treatment. At earlier periods the action of electricity is generally less striking, and an apparent success is still less capable of bearing a critical scrutiny. As regards the choice of one form of electricity rather than the other, Mitchell adheres to a well-known rule laid down by several other writers, namely, that whichever current is found to produce muscular contractions most readily should be used. It has been thought that by stimulating the muscles to contract one helps in keeping them well nourished. But Erb maintains that if the reaction of degeneration is present one cannot by galvanisation hasten the recovery of motor power, although he admits that when recovery has once begun its progress may be accelerated.

When employing galvanism in the treatment of paralysis from an affection of a nerve-trunk, one should use the "labile" method. The anode is placed over the plexus above, or on some convenient "indifferent" spot and the cathode is then slowly moved over the skin, covering each of the affected muscles and nerves in turn. The strength of the current should be such as to excite distinct muscular contractions, but not to cause more than a slight sensation of heat and a little redness of the skin. Its application should be continued for about ten minutes every or every other day.

If the faradic apparatus be chosen, its two electrodes should be held in one hand between different pairs of fingers, and they should be placed in succession over the various muscles at a little distance from one another.

Another method of treatment is that of *massage* or shampooing the affected parts. This manipulation requires strength and endurance as well as gentleness, for it ought to be continued for an hour at a time. Every part of the skin, so far as the paralysis extends, should be lightly pinched and tapped, and moved to and fro on the tissues beneath. Then the joints are to be moved in turn, and lastly the muscles must be gently kneaded and rolled, the power employed being gradually increased. The temperature of the limb is raised by 1° or 2° Fahr., the muscles are less flabby, and respond more readily to faradisation. The patient often feels refreshed and sleeps better, but sometimes is exhausted and otherwise worse for the treatment. If carried on for too long a time it may cause lumbar pain, headache, and nausea.

For persistent anæsthesia Mitchell recommends the application of a rather strong faradic current by means of the electric brush. After two or three sittings he has commonly found sensation beginning to return. He has also employed as counter-irritants rags dipped in hot spirits of turpentine and covered with oiled silk. This affects patients very differently, causing unbearable pain in some, while others scarcely feel it.

He quotes the case of a man who had lost sensation in the whole arm as the result of a fall on the shoulder, and who completely recovered after his arm and back had been severely blistered by the sun.

Causalgia may to some extent be relieved by dressings with cold water constantly renewed. The injection of morphia into the affected part is also useful, and Mitchell advises the repeated application of blisters. Sooner or later this form of pain almost always subsides.

FACIAL PARALYSIS.—The seventh cranial nerve—the *portio dura*, or facial nerve—is very liable to paralysis. Facial paralysis is often called *Bell's palsy*, from Sir Charles Bell having discovered its pathology in 1838.

Ætiology.—This remarkable disease is one of the few that are certainly caused by the direct action of cold. It may result from sitting at the open window of a room or of a railway carriage, sleeping near a damp cold wall, or from anything which chills the muscles supplied by the portio dura. Such cases are spoken of by some German writers as "rheumatic," according to that wide use of the term which really deprives it of meaning. It is supposed that the trunk of the nerve becomes inflamed and swollen so as to cause compression of its fibres in the stylo-mastoid foramen. The rarity of similar paralyses of the spinal nerves from cold may depend on the larger size of the intervertebral foramina.

Inflammation or tumours of the parotid gland, suppuration of the cervical glands, wounds of the cheek, and pressure of the forceps in instrumental delivery, are the chief other causes that may affect the nerve beyond its exit from the skull.

In its course within the aqueductus Fallopii the portio dura is liable to be involved in caries and necrosis of the petrous bone. In fracture of the base of the skull the nerve may be torn across. Dr Moxon has recorded ('Path. Trans.,' vol. xx) a case in which a clot of blood was found compressing and destroying this part of the nerve. Possibly a similar condition was present in an oft-quoted case of Sir Charles Bell's, in which facial paralysis followed a box on the ear; but more likely the tympanum was already diseased.

On the proximal side of the internal auditory meatus the facial nerve may be implicated in tumours and other diseases of the base of the skull; or its origin in the pons may be affected. As a rule, such cases are distinguished by other nerves being involved, or by further symptoms of cerebral disease. But Dr Fagge met with an instance in which simple paralysis of the right facial nerve, in a man who died of granular disease of the kidneys, was found to be caused by a minute spot of softening which existed in the pons, rather to the left of its centre, with a cyst of the size of a pea; and Dr Gowers has "seen two cases of seizure, evidently apoplectic, in which the only paralysis that followed the seizure was seated in the muscles supplied by the portio dura."

Partial implication of the facial muscles is common in ordinary hemiplegia; and it is a constant feature of bulbar paralysis. Facial paralysis has also been seen as the earliest symptom of tuberculous meningitis.[*]

The symptoms of well-marked Bell's palsy are striking. When the patient frowns, or smiles, or laughs, one side remains expressionless; the forehead is unwrinkled, no creases appear round the eye, the cheek and chin are marked by no dimples, the angle of the mouth is undisturbed. When he tries to close the eyelids forcibly, those on the paralysed side are motionless, a condition pedantically called lagophthalmos (hare's-eye); but since there is a physiological association between forced closure of the eye and elevation of the globe, the latter action, which is effected through the third nerve, still goes on; and the eyeball is involuntarily turned upwards, or upwards and inwards, until the pupil is hidden beneath the upper lid. The actions of spitting, whistling, and blowing are impossible; the sides of the mouth cannot be pursed up; the cheek is not held close to the teeth by the buccinator muscle, and bulges out with each expiration. When the patient masticates, the food collects outside the teeth on the affected side, unless he keeps his hand pressed against the cheek while he is eating. In speaking, he slurs or omits the labial consonants *p*, *b*, *f*, *v*, and *m*.

The appearance of the face in repose varies in different cases. In some patients the mouth is drawn far over to the opposite side, the eye stares fixedly, and the tears run from it. This may be partly due to spasm of the antagonist muscles, and is only seen when the nerve is completely destroyed, as in cases of necrosis of the temporal bone. In the ordinary peripheral cases the patient's features are but little disturbed while they are at rest, so that one may scarcely notice that anything is amiss with him. Although he cannot close the eye, the palpebral aperture may appear no wider than on the unaffected side, and the *tensor tarsi* muscle may still be able to keep the lachrymal puncta in contact with the globe, and so prevent the tears from running over the cheek. During sleep, when the levator palpebræ is relaxed, the eyelids approach one another more nearly than when the patient is awake. This protection during sleep may partly explain the not infrequent absence of inflammation from dust settling on the exposed eyeball. No doubt the conjunctiva often does inflame, and the cornea may lose its transparency or ulcerate; but Valleix had a case of facial paralysis of twenty years' duration in which no such results took place. In the daytime, as Trousseau remarks, the patient is able to make up for movement in the eyelids by bringing the globe into various positions so as to wipe it upon different parts of their inner surfaces; or he may push down the upper lid with his finger.

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[*] I once had a patient who was attacked with Bell's paralysis twenty-four hours after the development of an eruption of herpes zoster on the same side of the face. He came to me about seven weeks afterwards; the loss of power to move the facial muscles was then almost complete, and the reaction of degeneration was found to be present when the electrical test was applied. The purple scars caused by the eruption were still visible. No indication of any disease affecting the seventh nerve could be discovered, and I was inclined to regard the paralysis as reflex. He slowly recovered, after the lapse o several months.—C. H. F.

In all severe or protracted cases, independent of their origin, the reaction of degeneration comes on after a few weeks.

Among occasional effects of facial paralysis, impairment of the sense of *smell* is most likely due to loss of the power of sniffing up the air.

A more frequent indirect symptom is a perversion of the sense of *taste*. Trousseau, for example, speaks of a man who said that his food tasted salt, and other patients describe a metallic or sour taste in the fore-part of the tongue on the affected side, or a loss of power to detect acid, sweet, or saline flavours. Or, again, acids and salts may be recognised but syrup and quinine unrecognised. This symptom appears to be due to paralysis of the chorda tympani branch. The secretion of saliva may be diminished from the same cause affecting the submaxillary gland, as the writer once witnessed.

The sense of *hearing* is often unusually acute for all musical notes. The patient hears a watch at a greater distance from his ear on the affected than on the healthy side, and can detect sounds of a very low pitch. Lucæ has shown that this condition (barbarously named "oxyakoia") is the result of paralysis of the stapedius muscle, which receives a branch of the facial nerve; its antagonist, the tensor tympani, being no longer opposed, keeps the membrane too much on the stretch ('Berl. klin. Woch.,' 1874, Nos. 14, 16). Auditory hyperæsthesia should therefore never occur in facial palsy of peripheral origin.

Another branch of the facial nerve, affected in Bell's paralysis, is that which goes to the soft palate. This was fully described by Romberg, and the late Dr Sanders taught ('Edin. Med. Journ.,' 1865) that there is "a vertical relaxation or lowering of the corresponding half of the velum, with diminished height and curvature of the posterior palatine arch," due to loss of power in the levator palati. In certain cases the uvula is turned to the paralysed side; but this seems not to be constant, and lateral deviation of the uvula sometimes occurs in healthy persons. If the levator palati is paralysed, it would follow that the lesion of the portio dura must be on the cerebral side of the geniculate swelling of the nerve in the petrosal bone. But the symptom is more often looked for than seen. It is doubted or denied by Wilks, Ross, Bastian, and Gowers.

Electrical reactions.—From the original observations of Erb and Brenner it appears that in the *mild* form of the affection, which recovers in two or three weeks, the muscles and nerves of the paralysed side of the face react both to galvanic and to faradic currents exactly in the same way as on the healthy side. In only a single instance of rapid recovery could Brenner detect a slight and transient diminution of excitability to both kinds of current. But in the *severe* form (which includes a large proportion of those cases which are due to the direct action of cold) the "reaction of degeneration" is observed. Indeed, it was in a case of facial paralysis that this reaction was first noticed by Baierlacher in 1859.

Diplegia facialis.—Occasionally the portio dura is paralysed on both sides. There is then no distortion of the features under emotion. The face remains without expression, and, as Romberg put it, the patient laughs or cries behind a mask. Double facial paralysis may be caused by a new growth or gumma, involving both nerves at their origin, or by disease affecting the two petrous bones separately, or the action of cold upon both sides of the face in succession, as in eight cases collected by Davaine.

The writer published a remarkable case of this kind—Diplegia facialis with deafness of both ears and vertigo—in the 'Guy's Hospital Reports' for 1895, with reference to cases of Sir William Roberts and Mr Hutchinson, as well as to older ones recorded by Romberg, Bell, and Todd. Davaine's cases were none of them accompanied by deafness. In the case mentioned the cause was almost certainly syphilis affecting the conjoined seventh pair of Willis. Not only the portio dura but the portio mollis was paralysed, including the cochlear nerve and that supplying the semicircular canals, for to the latter lesion the vertigo seemed to be attributable. The patient himself said that he could not walk straight, because he was giddy, his head swam, and he felt as if he were drunk. Whether there were symmetrical lesions of the pons close to the root of the nerve, or of the pia mater, or of the sheath in the internal auditory meatus, was doubtful.

Diagnosis.—This is seldom difficult when facial palsy is fully developed. If other nerves beside the seventh are paralysed, we suspect the presence of some new growth or syphilitic disease at the base of the skull. Even when the loss of power is slight and incomplete, one can for the most part decide its peripheral origin by a little failure of expression limited to one side of the face, and by a narrow chink remaining between the closed eyelids, for these symptoms do not accompany a partial paralysis of the facial nerve from central disease.

It must not be forgotten that some persons have a habit in speaking of moving the mouth awry, sometimes to conceal a defect in the teeth.

When the fact of paralysis of the portio dura is clear, we must try to fix its seat. If above the apparent origin in the pons a lesion will affect most of the muscles on the opposite side of the face, but not the orbicularis and corrugator supercilii; there will probably be hemiplegia or other cerebral symptoms; and there will be no reaction of degeneration. Such a condition will be described among the symptoms of ordinary hemiplegia. The lesion will most often be in the external capsule or adjacent parts; or it may be higher, in the facial centre of the cortex; or lower, in the crus of the opposite side; or, the upper part of the pons before the crossing of the fibres of the nerves. In the "nucleus" of its apparent origin (answering to the anterior cervical origin of a spinal nerve), the facial nerve may be paralysed by hæmorrhage (rarely), or by tumour, or by sclerosis (as occasionally in the progressive wasting palsy known as bulbar paralysis); at its root and in its course to the end of the internal auditory meatus, by local gummatous or other meningitis, when the portio mollis will also suffer. In such cases the paralysis is nearly complete; the upper part of the face is affected as well as the lower, while the stapedius, occipitalis, digastricus (in its posterior portion), stylo-hyoid, and platysma are also involved. Moreover there is commonly auditory vertigo and deafness on the affected side, as well as motor palsy. In its tortuous passage through the aqueduct of Fallopius the facial nerve shares in all the risks of caries and necrosis of the petrosal bone after scarlatina or enteric fever (otitis media, tympanitis).

Again, one or both nerves may be torn across when one or both petrosals are the seat of fractured base of the skull.

The symptoms in such cases are unilateral (or, in the rare cases otitis of both ears, bilateral), paralysis of all the facial muscles, of stapedius ("oxyakoia") and of the stylo-hyoid, &c., without deafness vertigo, and with the reaction of degeneration.

Lastly, if there is complete facial paralysis without paralysis of stapedius, the levator palati, or the digastric and stylo-hyoid, followed by the reaction of degeneration, the condition is due either to injury of the branches of the pes anserinus by an accidental cut or an operation, or by an abscess of the cheek, or by pressure of the forceps in delivery, or more frequently to peripheral neuritis set up by a *coup de vent.*

In one case the late Dr Thomas found actual hæmorrhage into the Fallopian canal in a man who died of Bright's disease with ordinary peripheral facial palsy ('Path. Trans.,' 1869, p. 420).

Course and Prognosis.—The onset of Bell's paralysis is sometimes gradual, but frequently complete within twenty-four hours. Among hospital patients it often happens that what first attracts the patient's notice is his finding himself unable to spit or to whistle; or he may find his face "all on one side" when he gets up in the morning.

The subsequent course of facial palsy varies greatly. When due to destruction of a considerable portion of the nerve, as in cases of necrosis of the petrous bone, it is of course permanent and incurable. Although recovery is possible after an injury, such as the division of the nerve in a surgical operation, yet this seldom occurs before the lapse of several months, and it is apt to remain incomplete. When the affection is the direct result of exposure to cold, recovery to some extent always takes place, but it may last from four to six months. Sometimes it gets well much more rapidly, the patient beginning to regain power over the muscles at the end of ten or twelve days, and recovering completely within a few weeks. The longer the recovery takes the less perfect it is.

When the reaction of degeneration has set in no improvement can be looked for until two or three months have passed, and even then it is often imperfect.*

In severe cases of Bell's paralysis *spasmodic affections* appear after a time. When the patient is just beginning to recover voluntary power over some of the muscles, he often, by what is called irradiation of nervous impulse, moves more than he intended. He may wish to raise his eyebrow, and he involuntarily draws his mouth to that side, or closes his lips, and finds that he also shuts his eye. In a patient who was under Dr Fagge's care in 1877 the distortion of the mouth was so great when an attempt was made to close the right eye, that the clinical clerk supposed the left orbicularis oris to be paralysed. Such mistakes make it needful to be sure when we are told that the left side of a patient's face was paralysed, that it was not really the right. It is curious to observe that Aretæus warns physicians on this very point ('De causis et signis morborum chronicorum,' lib. i, cap. vii).

Hitzig has observed ('Arch. f. Psych.,' 1872) that the involuntary movements sometimes occur while the muscles which cause them are still paralysed. It would seem that the facial nucleus in the bulb may also be in a condition of exalted irritability, for Hitzig has found that touching the affected side of the face sometimes gives rise to spasms which may involve the muscles of the opposite side. In one case of facial paralysis contractions extended to the muscles of mastication, and to those of the limbs. The occurrence of associated movements is commonly a precursor of the return of voluntary power over the paralysed muscles. Such movements generally quickly disappear, but sometimes they seem to delay recovery, and Erb has known them to last as long as thirteen years.

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* Erb describes an intermediate form of facial palsy in which the excitability of the nerve is not completely lost, although the muscles present the characteristic sensitiveness to feeble galvanic currents with absence of faradic contractility; such cases, he says, recover in from four to six weeks.

When the paralysis is to be permanent, *tonic contractions* of some or all of the muscles often show themselves, as first described by Duchenne. The result may be an elevation of the angle of the mouth, a deepening of the naso-labial groove, a narrowing of the palpebral aperture, or a general exaggeration of all the markings of the affected part of the face. The features are sometimes distorted until one might suppose the healthy side was the one paralysed. (See Gower's second vol., fig. 102.) These tonic spasms depend upon degenerative changes in the muscles themselves—a fact established by a case of Hitzig. A patient, who already had contraction of the orbicularis palpebrarum from a former seizure of Bell's paralysis, was attacked a second time on the same side without the orbicularis relaxing.

In facial paralysis of cerebral origin (cortical or nuclear), the prognosis is all but hopeless. In cases secondary to disease of the tympanum it depends on treatment of the ear. In peripheral cases, the persistence of faradic excitability is of good omen. If the palsy persists for three or four months, recovery is unlikely; and the presence of marked reaction of degeneration in addition is of very bad prognosis.

Treatment.—When the discovery was first made that the muscles in many cases show increased reaction to interrupted galvanism, it was assumed that this form of electricity would cure the disease more quickly than the faradism which had before been employed. But thirty years ago Erb, after analysing the cases which had then been recorded, expressed the opinion that the severe cases in which alone the reaction of degeneration is present, cannot be materially benefited by galvanism until the conductivity of the nerves is re-established. The anode should then be placed behind the ear, and the cathode moved over the paralysed half of the face. In mild cases each application of the current is usually followed by increase of voluntary power. Duchenne had reported still better results from faradisation. He first showed that it is capable of introducing secondary spasms, and consequently advised that when the muscles begin to react, the intermissions of the induced current should be reduced to four in the second.

Leeches behind the ear, blisters, and mercury or iodide of potassium have been advised for facial paralysis during the first two weeks. But probably still less is to be anticipated from medicinal treatment than from electricity.

PARALYSIS OF THE MUSCLES OF THE EYEBALL.—Of the movements of the eyes some are caused by single muscles; and the remainder, for which several muscles combine, are capable of a physiological analysis far more exact than has yet been applied to the movements of the limbs. The affections of the three motor nerves cannot be taken separately, because the two eyes are moved together, and a muscle is not always associated with its fellow in the other eye. For example, the external rectus of one side, supplied by the sixth nerve, works with the *internal* rectus of the other side, which received its branch from the third nerve.

Two ocular muscles, the external rectus supplied by the sixth nerve, and the superior oblique by the fourth, are more likely to be separately paralysed than any single one of the rest, which are all supplied by the third nerve; and hence the former muscles are the most important in diagnosis.

Paralysis of the sixth nerve.—Each external rectus is associated with the internal rectus of the opposite side. Their combined function is to carry the eyes to the right or left, without any change in the direction of the vertical axes of the eyeballs. Let us suppose that the patient has his *left* sixth nerve paralysed, he will be unable to look with his left eye at any object to his left. If we hold a candle before him, and move it to his right side, his two eyes follow it until the right one has the edge of its cornea touching the external canthus, and the left has a small part of its cornea concealed beneath the caruncle. If we now carry the candle from in front of him to his left side, the right alone follows it; the left remains motionless, and looks straight forwards, or may perhaps perform a slight zigzag movement to the left under the combined influence of the two obliqui. The further the object is carried to the left the greater is the difference in the directions of the two eyes; and when the paralysis is complete, this *primary deviation* or *squint* at once shows the nature of the case.

When the loss of power is only partial the affected (left) eye lags behind its fellow, but no obvious squint appears. In such a case one can generally make out the nature of the defect by covering the right or healthy eye; the patient then discovers that he has not got his left eye fixed upon the candle, and he accordingly rotates that eye further to the left. This correcting movement may be readily detected by a close observer; but what is more obvious is a movement which is simultaneously made by the right eye. For the left external rectus being partially paralysed, any voluntary movement of that muscle requires the exertion of far more effort than sufficed in health. But whatever amount of nervous energy is put forth is at the same time thrown upon the associated muscle—the right internal rectus—which is in possession of its full vigour; and thus the right eye moves much farther than the left.

This movement of the healthy eye is called *secondary deviation;* and it is of the more importance because it affords a sure means of distinguishing a paralysis of the external rectus from a mere contraction or shortening of one or both of the internal recti, such as exists in cases of ordinary strabismus. When the loss of power is considerable, this distinction is made by the fact above mentioned, that the primary deviation increases as the object looked at is carried further over towards the left side; for if the affection were a mere convergent strabismus the axes of the eyes would remain at exactly the same angle, the one lagging at a fixed distance behind the other, but nevertheless travelling with it—a "concomitant strabismus." But in slight cases this criterion fails, and is replaced by the secondary deviation, the *augmented* extent of which is (as we have seen) dependent on the existence of paralysis; whereas in cases of ordinary strabismus the secondary is exactly equal to the primary deviation, since the muscles on both sides possess equal power.

The other symptoms of paralysis of the sixth nerve are distorted perceptions of the patient, and certain movements to which they lead. When he looks at an object held towards the affected side he sees it double, and this *diplopia* is often the first thing which shows him that something is amiss. We will suppose as before that the *left* external rectus is the muscle paralysed, and we will call the image which is formed upon the retina of the affected (left) eye "the *false* image;" the one which is formed on the unaffected (right) eye "the *true* image." It will be found that double vision occurs only when the eyes are directed to the left, for it is then only that they fail to converge properly upon the object. And a moment's consideration will show that, since the left eye has its axis turned inwards (or to the right), the false image must be formed on the inner (nasal) side of its retina, and not on the yellow spot; and, consequently, that it appears to be *outside the true image* or *further to the left.* Both images are upon the same level, and both are upright. By placing a piece of coloured glass before one of the patient's eyes, we can enable him to distinguish very easily which image is formed by that eye, and which by the other.

It must be borne in mind that diplopia is not always a proof that paralysis of any of the ocular muscles is present. Double images are sometimes formed upon the retina of a single eye; and in other cases they are due to a mere "concomitant strabismus." Making the patient look at the object with each eye separately will exclude the former condition; but the latter can only be dismissed from consideration if it is found that the two images diverge as the object is moved further to the left. One must not assume that diplopia which is of recent origin and which began suddenly cannot be due to concomitant strabismus; for it sometimes happens that a patient whose ocular muscles are imperfectly antagonised is able to keep up the balance between them until he over-fatigues his eyes, or until he is weakened by some illness, when he may abruptly begin to squint.

On the other hand, it would be a mistake to suppose that whenever there is a loss of power in the external rectus muscle the patient must necessarily be conscious of diplopia. There is always the possibility of one eye being defective from abnormal refraction or opacity of the media, so that he has been in the habit of concentrating his attention upon a single retina; in that case all that he is likely to notice is a blurring of the objects at which he looks. One can generally make such a person aware that he really sees double by directing him to fix his eyes upon some small bright object, especially if a coloured glass be held in front of one eye.

Another subjective symptom is that which is known as the "*erroneous projection*" of the visual field. We have seen that a patient who has paralysis of the left external rectus muscle refers the image of any object placed to his left, which he sees with both eyes open, to a position outside its true position, *i. e.* more to the left. The same thing occurs if he is told to look at an object with the right eye closed; because his judgment is then based upon the degree of effort required to bring his left eye to bear upon it, an effort augmented in proportion to the failure in the power of the muscle. One consequence is that such a patient feels giddy, and may even stagger, if he attempts to use his left eye alone—a point of some importance, because the symptom might be mistaken for a sign of serious cerebral mischief. Another result of "erroneous projection" may be employed as an aid in the diagnosis of paralysis of the ocular muscles. The patient is told to close the eye supposed to be unaffected, and to strike at an object on the outer side of his other eye. If the external rectus is paralysed he is sure to miss the object by going to the outer side of it.

Some patients free themselves from the uncomfortable sensations to which paralysis of the sixth nerve gives rise, by keeping the head fixed over towards the paralysed side, so that the images of the objects at which they look may be referred to their right positions.

When paralysis of the external rectus has lasted for a long time, the position of the eyeball often undergoes a further change. It is now drawn inwards by the uncontrolled action of its internal rectus, so that a convergent squint is constantly present, whatever may be the direction in which the patient looks. Von Graefe called this condition the "secondary contraction of the antagonistic muscle," and observed the curious fact that it does not always correspond in degree to the loss of power in the paralysed muscle.

Paralysis of the fourth nerve.—The action of the *obliquus superior* is far from being as simple as that of the rectus externus; and its paralysis is more difficult of detection. The lower rectus, when acting alone, rotates the cornea slightly inwards, beside lowering it; the superior obliquus rotates it slightly outwards; the resultant of their combined action is a straight movement in a vertical plane. The left inferior rectus, by itself, would lower the cornea and carry it towards the right; the left superior obliquus, by itself, would lower it and carry it round to the left. Acting together, they move it straight downwards. The inferior oblique acts in the same way along with the superior rectus.

Suppose the left superior oblique muscle to be paralysed. The result is that when the patient looks downwards the cornea is carried to the right by the inferior rectus of the left eye, which has now no coadjutor to counteract its rotating the globe in that direction. In other words, he will squint to the right and slightly upwards with the affected eye when it is directed upon an object at his feet. The "secondary deviation" of the sound eye will be downwards and to the left. These symptoms, however, are comparatively little marked; so that the subjective symptoms of diplopia become more important in the diagnosis of the paralysis of the fourth than in that of the sixth nerve. The double images are seen chiefly when the patient looks downwards, and their position in regard to one another at once indicates what muscle has lost its power. Thus, first, the false image lies *below* the true one; secondly, it is placed to its *left;* and thirdly, it appears to be *tilted* in a particular manner. This last symptom depends upon the way in which the affected eye is rotated, for the image of the upper end of an object will be formed upon a part of the lower half of the retina outside the vertical meridian of the retina, and so the object seems to that eye to be placed obliquely *with its upper end tilted to the right.* Another point noticed by Von Graefe is that the false image seems to be nearer to the eye than the true one; this, he says, depends upon their both being referred to positions upon a horizontal plane spread at the patient's feet, so that the true image, being the upper of the two, is naturally supposed to be the more distant. Lastly, the patient, in order to avoid a sensation of giddiness when he looks with both eyes at objects before him, keeps his head turned downwards and to the right—a position which is very characteristic.

Paralysis of the third nerve.—Since this nerve, unlike the sixth and fourth, is distributed to four of the ocular muscles, the symptoms produced by its paralysis differ according to the extent of the affection.

In paralysis of the *internal rectus* the symptoms are the converse of those which belong to paralysis of the abducens. If we take, as before, the muscle of the left side, the "primary deviation" occurs when the eyes are directed on an object towards the right; the affected eye cannot then be moved inwards while the other one is being moved outwards, and a *divergent* squint is produced. The "secondary deviation" of the right eye is towards the right. Double images are perceived by the patient when he looks to the right, and the distance between them increases the further the object is moved in that direction. They are both upon the same level and both upright. They are said to be "crossed;" that is, the false image (which is seen by the left eye) lies to the *right* of the true image (which is seen by the right eye). The "false projection" of the image takes place toward the right; so that the patient, if he aims suddenly at an object with the right eye closed, misses it by going too far to the right. To avoid giddiness the patient keeps his head over to the right.

In paralysis of the left *inferior rectus* most of the symptoms are the converse of those which are produced by paralysis of the superior oblique muscle. As in that affection, the affected eye squints a little upwards when the patient looks downwards, but the rotation of the globe is now to the left. Diplopia occurs when the eyes are directed upon an object below them; the false image is below the true one, lies to its right, and has its upper end tilted to the left.

In paralysis of the left *inferior oblique* the affected eye squints a little downwards when the patient looks upwards; the globe is rotated to the right. The false image lies above and to the left of the true one, and has its upper end tilted to the left.

In paralysis of the left *superior rectus* the affected eye squints a little downwards when the patient looks upwards; the globe is rotated to the left. The false image lies above and to the right of the true one, and has its upper end tilted to the right.

Lastly, if the *whole of the third nerve* on the left side is paralysed, there is complete loss of power to move the eye inwards or upwards. It cannot be moved straight downwards, but it can be slightly lowered if also carried a little outwards.* In the direction horizontally outwards its play is perfectly free.

The visual range of the affected eye is reduced to about one twelfth of its normal extent; it is limited by a straight horizontal line above, but below by a curved line which sweeps downwards and then outwards.

The direction of the "primary deviation" of the left eye, and that of the "secondary deviation" of the right eye, will, of course, vary according to the position of the object at which the patient looks; the relations of the false to the true image of course undergo corresponding variations, and so also does the "erroneous projection" of the objects that meet his eye on different sides. The consequence of this is that giddiness is a far more marked symptom when several of the ocular muscles are paralysed than when one alone is affected; and it cannot be obviated by adopting any particular posture for the head, nor, indeed, by any method except that of shutting the affected eye (the left, as we still suppose). It is only when the patient looks at an object in the extreme left of the visual field that he ceases to squint and sees a single image with the two eyes.

In paralysis of the entire third nerve, beside the affection of the four ocular muscles, there is loss of power in certain other muscles which are also supplied by that nerve. One of these is the levator palpebræ, and the result is that the upper eyelid droops, a condition termed *ptosis*. The patient is altogether unable to open the eye in the ordinary way; at most he can only separate the lids by wrinkling the forehead—by means of the occipito-frontalis. Ptosis may exist without paralysis of any other muscle; indeed, it is much more common than an isolated affection of any other branch of the third nerve.

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* In this movement the globe is made to revolve on its antero-posterior axis by the uncompensated action of the superior oblique muscle. And the rotation is so marked—especially when the eye is allowed to move as little outwards as possible—that it affords a striking confirmation of the modern views with regard to the action of the ocular muscles

Again, the sphincter muscle of the pupil receives filaments from this nerve, and paralysis of them leads to dilatation of the pupil, or (as it is termed) *mydriasis*. The aperture of the iris never becomes so wide from paralysis as it does under the influence of atropine; but it is often large enough to interfere seriously with the accuracy of vision, owing to the formation of "circles of diffusion" upon the retina from the spherical aberration more or less present in an average eye not being corrected by the iris acting as a diaphragm.

Lastly, the ciliary muscle is also supplied by the third nerve, and hence *paralysis of accommodation* may be one of the symptoms of disease of that nerve. Except in very short-sighted persons, this greatly interferes with distinctness of vision for small print and other near objects. To detect it, one must test the range of accommodation in the usual manner.

The common signs, therefore, of paralysis of the third nerve are—external strabismus, ptosis, dilated pupil, and loss of accommodation for near objects.

Ophthalmoplegia externa,—a term used by von Graefe. When all the muscles of the eyeball are paralysed together (from affection of the third, fourth, and sixth nerves) there is immobility of the globe with ptosis, but without strabismus, and without double vision for distant objects—unless one eye only is paralysed. The paralysis is usually double.

The term *Ophthalmoplegia intima vel interna* has been applied by Hutchinson to paralysis of the iris and ciliary muscle only ('Med.-Chir. Trans.,' 1878, p. 215, and 1879, p. 307).

Ætiology of ocular paralysis.—With regard to the causes of paralysis of the third, fourth, and sixth nerves, a point of great practical importance is that almost all those cases in which the affection remains limited to a single nerve, or to a single branch of the third nerve, terminate sooner or later in recovery, excepting cases of traumatic lesions or of basal meningitis, where the sixth nerve is most often affected. In the absence of dissection after death, oculists say that they are either syphilitic or "rheumatic" (*i. e.* not syphilitic).

In 1876 a man died in Guy's Hospital of aortic aneurysm who had about nine months before been in another ward suffering from ptosis of the left eye and pain in the left side of the head. An old meningeal apoplexy was found; the termination of the left internal carotid artery was dilated, and its coats much thickened; moreover the third nerve on that side was adherent to the side of the artery and stained of a deep brown colour. Sir William Gull used to say of the third nerve that it ran a "dangerous course," on account of its liability to compression in passing between the posterior cerebral and the superior cerebellar arteries, if these vessels should become diseased; and this view certainly accords well with the fact that ptosis is very apt to occur in old people whose arteries are dilated and tortuous.

Of the fourth and sixth nerves it must be remembered that their long and exposed course along the base of the brain renders them very liable to be affected by tuberculous or other effusions in the diamond-shaped space.

When two or more nerves are simultaneously affected, one suspects th presence of some malignant growth or aneurysm or gumma at the base the skull. An intra-cranial carotid aneurysm is very likely to compres the sixth nerve for some time before it reaches the rest. Not infrequentl suppuration or tumour in the orbit presses on one or more branches of th motor oculi.

A syphilitic affection of isolated nerves is not always a gumma, for ther may be ptosis of each eye, while the ocular muscles entirely escaped, and it is difficult to suppose that the corresponding parts of the third nerves wer both occupied by a gumma. Probably the lesion is often syphilitic neuritis. Von Graefe is said to have traced to syphilis about one third of all cases of ocular paralysis.

We shall hereafter find that ocular paralysis may accompany various lesions of the base of the brain, as fracture in the anterior fossa and basal meningitis, and that they are sometimes early symptoms in locomotor atary or insular sclerosis; but when an organic lesion of the brain or spinal cord is present, one can almost always discover other symptoms which exclude the supposition of peripheral paralysis.

Prognosis.—Most cases of ptosis or of paralysis of the various ocular muscles recover, except when they are due to aneurysm or malignant growth compressing the affected nerve. But it must be added that recovery is sometimes slow, taking many months; or imperfect, so that one or more of the affected muscles remain permanently weak.

Treatment.—When the palsy can be traced to syphilis, treatment by mercury is usually successful. Iodide of potassium is probably less useful. In other cases the application of blisters behind the ear is believed to be serviceable. Benedikt and Erb have used galvanism for two or three minutes at a time, with the anode on the temple or the back of the neck, and the cathode on the closed eyelids. The improvement is said to be often instantaneously manifested, and Benedikt thinks that if no good is effected within the first fortnight there is no object in continuing this treatment; but according to Erb many cases require to be galvanised for several months before any result is discoverable.

Paralysis of the ninth or hypoglossal nerve is a common symptom of cerebral, pontine, and bulbar paralysis, but as a lesion of the nerve-trunk it is rare. It is attended with one very remarkable effect, wasting on th corresponding side of the tongue. Dr Fagge recorded two cases of thi kind: one was in an old woman, in whom a cancerous nodule (seconda to a cancer of the breast) involved the nerve where it passes through t base of the skull; the other in a boy, who had extensive caries and necros of the atlas and neighbouring bones. In both patients the paralysed ha of the tongue was remarkably flaccid and wrinkled, and its tip was curv round towards the affected side. Similar cases have been recorded Dupuytren, Sir James Paget, and the late Mr Fairlie Clark. In Paget case ('Clin. Trans.,' vol. iii) the affection was caused by necrosis of a pa of the occipital bone from an injury. Several pieces of dead bone we removed by operation; a few days afterwards the wasted part of the tongu began to grow larger, and within a month it had nearly regained its form- size and muscular power.

Cases of double hypoglossal paralysis are on record, apart from tho due to cerebral hæmorrhage and to narcotic poisoning, as in the administr tion of chloroform. The tongue lies motionless and low between the rami of the mandible, and articulation and deglutition are impeded.

Paralysis of the fifth nerve.—The principal symptom of this affection is anæsthesia of the face. The loss of sensation or the corresponding subjective sensations of numbness and painful tingling may in some cases be traced with great accuracy to the median line of the forehead, nose, and mouth. Most often what first attracts the patient's notice is that when he puts a cup to his lips it seems to him exactly as though it were broken. The eyelashes and conjunctivæ are perfectly insensible; so also is the nostril, and liquor ammoniæ may be applied to it without causing sneezing; but there is no impairment of the sense of smell, except from diminished secretion of tears and consequent dryness of the mucous membrane. The sense of taste has in several cases been found absent in the anterior portion of the tongue, as long ago observed in three instances by Romberg. It is the same part of the tongue which is deprived of the sense of taste in cases of facial paralysis; so that probably the chorda tympani is a true gustatory nerve, and in different parts of its course runs with the fifth and also with the seventh nerves. The great petrosal nerve appears to be the connecting branch.

Another effect of lesion of the fifth nerve is paralysis of the masticating muscles on the affected side. This only deprives the patient of one movement completely, that in which the lower jaw is carried forwards and towards the *healthy* side by the external pterygoid muscle. His power of chewing food is much less impaired than might have been expected, but he masticates only with the muscles of the unaffected side. If he is thin, one can see that the temporal and masseter do not swell out when the mouth is forcibly closed; or one can easily feel that they do not harden.

In certain cases of facial anæsthesia the circulation in the affected parts is disordered, or their nutrition is to some extent impaired. Sponginess of the gums, ulceration of the mucous membrane of the nose and mouth, and hæmorrhage from their surfaces have been observed. But such trophic changes are rare and comparatively unimportant, whereas there is another affection which is of frequent occurrence, namely, inflammation of the eye, leading to sloughing of the cornea. It is now agreed, after prolonged investigations in the laboratory, that the inner portion of the trunk of the fifth nerve contains all the trophic fibres of the eyeball; but there is still a difference of opinion as to whether the result is due to a mere interruption of their conducting power, or to some directly irritant influence. Snellen's view, that the anæsthesia is its immediate cause, the eye being exposed to mechanical injuries from the loss of the protection afforded by the sensitive nerves, is now generally abandoned; indeed, Charcot brought instances in which the cornea sloughed as the result of affections of the fifth nerve, unattended with loss of sensation.

We have seen that in affections of the great nerve-trunks of the limbs, when the continuity of their fibres is not completely destroyed, anæsthesia, if present at all, soon passes off. We should therefore expect that a nerve running so protected a course as the fifth would be seldom paralysed except by lesions of a destructive kind. And this appears to be the case.

In the great majority of cases paralysis of the fifth nerve is due to destruction of the trunk or of its sensory ganglion, by caries or necrosis of the skull, syphilitic disease of the pons or meninges, cancerous or sarcomatous growths, or, lastly, by aneurysm of the internal carotid artery.

A man aged forty-two, who formerly saw service in India, was a patient of the writer in John Ward (November, 1897). He had signs of chronic tuberculous disease of the lungs, and also of spastic paresis; but the most striking symptoms were those of paralysis of the fifth nerve. There was anæsthesia over the distribution of the first and second divisions, the forehead, cheek, and front of the ear, the conjunctiva and the mucous membrane of the nose and upper jaw on the left side. The chin and lower lip supplied by the third division escaped, but there was complete motor paralysis (as usual more complete than the sensory paralysis) of the muscles of mastication. The left masseter and temporal muscles were wasted as well as powerless, and when the patient drew the lower jaw forward it was dragged over to the paralysed side, showing that the external pterygoid shared in the disease, and so no doubt did the internal pterygoid also. There were no trophic symptoms present beside this muscular atrophy. There was a history of syphilis, and the lesion seemed most likely to be meningitis affecting the fifth nerve at its superficial origin.

Paralysis of the nerves of special sense.—The affections of the auditory and the optic nerves are too important to be treated adequately in a work on general medicine. Auditory vertigo with deafness points to affection of the cochlea and semicircular canals, as in the case above mentioned of *diplegia facialis*. We shall meet with it again with other cerebral affections.

Paralysis of the optic nerve, or *amaurosis*, is an important symptom of various lesions of the retina, optic nerve, and brain, but as a symptom of nervous disease neuritis and consequent atrophy of the optic nerve is far more important even when not accompanied by blindness, and we shall meet with it again and again in the following chapters. Limitation of the field of vision, colour-blindness, and illusions of both form and colour are other optical symptoms which we shall meet with hereafter.

When loss of smell (*anosmia*) is unilateral, the patient is very likely to remain ignorant of his loss unless one is particular to test the power of the olfactory nerves separately, carefully closing each nostril in turn. We shall hereafter see that this form of anosmia accompanies some forms of hemiplegia. When both nerves are affected, the patient is sure to notice the defect. His inability to perceive odours is then complete; he is altogether insensible to the most fragrant perfumes and to the strongest stinks. But he retains his susceptibility to pungent vapours, such as those of ammonia or acetic acid, and snuff is still capable of making him sneeze. The impressions from all these substances are conveyed to the brain [illegible] the branches of the fifth nerve to the nasal mucous membrane; and it only when that nerve is paralysed that they fail to be felt.

But the symptom which forces itself upon a patient suffering from complete anosmia is not the loss of the sense of smell (as he understands it) but impairment of the sense of taste. He can still recognise bitter and sweet, sour and salt; and he can distinguish the rough or smooth character of the food that he takes into his mouth. But in all other respects his sense of taste appears to be extinguished. He cannot tell one kind of meat from another; apples, onions, and turnips appear the same to him; [illegible] kinds of wine seem to have lost their flavour, tasting merely like sour [illegible] sweetish water, except that they are more or less rough to the palate. it were not from habit and prejudice he would probably be indifferent as [illegible] his food. Evidently, therefore, physiologists are right in maintaining that the true gustatory sense is limited to four tastes—sweet and bitter, acid and saline; and that the other perceptions known as tastes belong to the sense of smell.

The pathological significance of anosmia was worked out by Dr Wm. Ogle ('Med.-Chir. Trans.,' 1870). He records five cases in which this condition resulted from blows upon the head, the part struck being generally, if not always, the occiput. He believes that in such cases the olfactory nerves are torn across as they pass through the ethmoid foramina. Or perhaps the olfactory bulbs themselves may share in the bruising of the anterior lobes of the brain, as the result of injuries to the back of the head.

The remaining causes of loss of smell are independent of lesion of the olfactory nerves, but it may be well to mention them here. One is an absence of the pigment in the mucous membrane of the upper part of the nose; at least, a case has been recorded in which a negro boy lost the power of smell more or less completely at the same time that his skin became white. Dr Ogle cites some other evidence to show that there is a relation between the presence of this pigment and the olfactory sense. Another cause of anosmia is closure of the passage into the posterior nares by adhesion of the palate to the pharynx; this prevents the patient from drawing air through the nose, and so deprives him of susceptibility to odours, and also to flavours. When the latter remains while the former is lost, there is found to be an obstruction to the passage of air upwards through the anterior nares, while the passage through the posterior nares is open. The usual cause is thickening of the Schneiderian membrane by chronic catarrh, so that one part of the lateral wall of the nose is brought into contact with the septum. Dr Ogle mentions the case of a woman who for several years had entirely lost smell from this cause. A patient of Dr Fagge said that for ten months she had lost both smell and taste as the result of a severe cold; but probably what she had really lost was the power of appreciating flavours. Some arsenic was prescribed, and in less than a fortnight she perfectly recovered. In one case the writer found the sense of smell in both nostrils completely destroyed as the result of an attack of influenza in a healthy man of about fifty.

With respect to the loss of the sense of taste, those cases on record, which are certainly of nervous origin, are almost all entirely referable to paralysis of the chorda tympani. Taste is sometimes affected on one side in hemiplegia; but it is unaffected in bulbar paralysis.

MULTIPLE PERIPHERAL NEURITIS.*—In describing neuralgia and peripheral paralysis we have taken the most uncomplicated forms of each as types: a primary or idiopathic neuralgia, and paralysis like that caused by direct injury severing the nerve from its centre. But we have already stated the many cases of neuralgia are pathologically interstitial neuritis; the trophic disturbance of the skin called zona or shingles and the neuralgia which follows it are due to neuritis of the posterior roots and their ganglia; reflex paralysis is due, as Leyden showed, to ascending neuritis; and the Wallerian degeneration of a nerve severed from its trophic ganglia is due to parenchymatous neuritis.

Progressive neuritis of extensive tracts of peripheral nerves is now known to occur symmetrically, affecting the upper or lower limbs and sometimes the muscles of the trunk of the head in addition. Such cases are, as a rule, toxic, due to mineral or animal poisons.

History.—Chomel appears to have first recognised the clinical features of this affection, which occurred epidemically in Paris in the spring of

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* *Synonyms.*—Symmetrical neuritis—Polyneuritis—Peripheral paralysis—Neuritis acuta progressiva (Eichhorst)—Névaite parenchymatose (Goffroy).

1828, and his account was corroborated by Graves, who saw it there in the summer following. The great Irish physician described it as beginning with pricking and severe pain in the hands and feet, followed by excessive tenderness to the touch. This was succeeded by anæsthesia, and finally by loss of motor power, which spread up the arms and legs; but after the patients had lain helpless for weeks, they in most cases gradually recovered.

Duménil described a case of the disease in 1864. The clinical symptoms leave little doubt of its nature, and the microscopical examination of the nerves (but not of the cord) by Pouchet confirms that opinion.*

Dr Buzzard described in the 'Clinical Transactions' for 1874 a case of paralysis affecting both facial nerves as well as others, which recovered under antisyphilitic treatment; he has since regarded it as due to multiple peripheral neuritis; but there was no increased excitability of the paralysed muscles to interrupted galvanism, and of course no anatomical proof of the nature of the lesion. There was no mention of the disease as now recognised in Erb's article in 'Ziemssen's Handbuch' (Bd. xii, 2te Auflage, 1876). But in 'Virchow's Archiv' for 1877 (vol. lxix, p. 265), Eichhorst described, under the title "Neuritis acuta progressiva," a remarkable case occurring in a woman of sixty-six, with successive invasion of the nerves of the legs and arms, accompanied by pyrexia. She was suffering from chronic Bright's disease. The nerves were examined after death, and were found in a state of neuritis (pl. viii). Lancereaux in 1871, and Leyden in 1880, contributed cases.

Professor Grainger Stewart published three cases of the same affection in the 'Edinburgh Medical Journal' for April, 1881, as "Paralysis of the Hands and Feet from Disease of the Nerves." There was an excellent account of the disease under the title "Progressive Multiple Neuritis" in the late Dr Ross's treatise (1883, vol. i, p. 354), and Dr Buzzard published an interesting monograph in "Paralysis from Peripheral Neuritis" (1886). He showed that many cases of alcoholic paralysis which recover belong t this pathological group, especially those described by Dr Wilks in his we known 'Lectures on Diseases of the Nervous System' (p. 272).† T excellent article on the subject in 'Allbutt's System' (vol. vi, pp. 671—72 is by Dr Judson Bury.

Course and symptoms.—Multiple symmetrical neuritis (called peripher to exclude affections of the nervous fibres which run in the brain and cor and of the roots of the vertebral nerves) usually begins somewhat abruptl and runs a rapidly ingravescent course, though less acute than that of infantile paralysis. Pyrexia is only occasionally present. Pain in the course the nerves affected is quickly followed by more or less complete loss power in the muscles they supply; this paresis is accompanied by tinglin numbness, and loss of tactile sensibility. Trophic disorders frequentl ensue, particularly vesicles and bullæ.

The *pain* is that of neuralgia, often the peculiar burning pain describ as causalgia (p. 574). The *anæsthesia* extends over broad patches skin, sometimes independently of a particular nerve-trunk, and the affecte regions are frequently bordered by a zone of hyperæsthesia. The *loss of power* is variable in degree, from the slightest paresis to complete akinesia.

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* 'Gazette hebdomadaire,' 1864, p. 203. The same writer contributed a monogra on the subject, with several other cases, to the same Gazette in 1866 (pp. 51—84).

† Other cases of paralysis due to alcohol, *e. g.* those recorded by Dr Broadbent ('Med Chir. Trans.,' vol. lxvii, 1884), are clearly different in clinical, and probably also in pathological character. See also 'Brit. Med. Journ.,' Jan. 1, 1887.

Faradic contractility is lost, as was noted by Duménil in his first case; and most often, as we should expect, the galvanic contractility is increased, with the other characters of the reaction of degeneration. But Dr Buzzard has found this not to be a constant feature, and it was wanting in one case of peripheral neuritis which recovered under the writer's care. As a rule it is present except in the cases which in other respects resemble progressive muscular atrophy; in these reaction to both currents disappears as the muscles waste.

The *distribution* is symmetrical, and affects the extremities more often than the face or the trunk; and the hands or feet, or both, rather than the proximal parts. When the legs are affected, the extensors of the foot suffer most, so that the patient has a characteristic gait, in which the toe drops and catches the ground.

The distribution varies in the several varieties. Alcoholic neuritis usually begins symmetrically below the knee, then spreads upwards, then attacks the hands, and only occasionally the neck and trunk. Plumbic neuritis begins symmetrically in the hands and rapidly reaches the forearms, where all the extensor muscles supplied by the musculo-spiral nerve undergo loss of power and wasting, except the supinator longus. Occasionally it spreads to the upper arm and shoulder; still less frequently it affects the lower extremities. The writer has seen only two cases of the latter event; in one the feet were "dropt" after the hands, in the other the legs were alone affected.

But motor, sensory, atrophic, and vaso-motor symptoms vary much in their comparative frequency and degree, and are not even always constant to one ætiological form of the disease.

Pain and tenderness are absent in some forms of neuritis; thus, lead palsy seems to attack the motor or some of the motor nerves with little disposition to affect the sensory.

Diagnosis.—The pains *are* neuralgic; but anæsthesia (often present to a slight degree in neuralgia) is well marked, and there is motor paralysis as well. The course and clinical features are different from those of mere neuralgia, and probably the histological condition of the affected nerves is different also.

The symmetry distinguishes multiple neuritis from ordinary hemiplegia and from crossed hemiplegia; while lesions of the pons which affect both sides of the trunk and both sides of the face, without producing rapid death, have rather a theoretical than a practical existence. More often there is difficulty in distinguishing multiple neuritis from symmetrical affections of the cord and its membranes, particularly spinal meningitis. Some cases of cervical pachymeningitis, in particular, may closely resemble peripheral neuritis. So also do certain central lesions which run an acute course, like Landry's ascending paralysis, and some forms of essential spinal paralysis in adults.

The absence of implication of the bladder and rectum, and of bedsores, distinguish it from destructive transverse lesions of the cord. Hysterical paraplegia does not affect the hands, and is not limited to the feet; while hysterical anæsthesia is unilateral and without akinesia. The severe pains of multiple neuritis, along with disturbance of sensation and voluntary movement, are the symptoms which most resemble spinal meningitis.

Apart from the characteristic distribution, and the relation to definite nervous tracts, the most distinctive symptom is deep-seated tenderness on pressure, and particularly tenderness of the affected nerve-trunks. The usually favourable course, and the absence of rigidity in the affected muscles, are additional helps to diagnosis; and so is the existence of such known causes as alcohol, diphtheria, or plumbism.

In some cases there are striking exceptions to the usual symptoms. As above stated, the reaction of degeneration may be absent or imperfect. There may be some loss of control over the bladder, as in a case of the writer's above noticed; and there may be, as Leyden has recorded, paraplegic bedsores. Atrophy of the muscles may be slight, instead of well marked; but if rigidity of the affected muscles supervenes, probably the lesion is not confined to the peripheral nerves. Loss of knee-jerk is very common, particularly in alcoholic cases, and with the pains and unsteady gait has led to the diagnosis of Tabes. The gait, however, differs from that of locomotor ataxia, as well as from that of cerebellar disease, and is very characteristic of one of the forms of alcoholic paraplegia. If we call the tabid gait "unsteady" or "staggering," and the cerebellar "reeling," we may apply the term "halting" to the dragging of the feet described above.

Histology.—To the naked eye the affected nerves appear normal. The microscopic degeneration begins in the fibres themselves (parenchymatous neuritis, v. *supra*, p. 471), and leads to breaking up of the axis-cylinder, segmentation and extravasation of myelin, and muscular degeneration. Sclerotic atrophy of the nerves with loss of myelin and thickened perineurium, as described by Leyden, are probably secondary conditions.[*] The characteristic change begins in the branches within the muscles and thence spreads upwards, but never reaches the roots of the nerves. In the typical cases no secondary changes are found in the nerve-roots or in the cord. By the administration of lead to guinea-pigs, Gombault (quoted by Ross) produced similar changes, not continuous, but in segments of the nerves.

Ætiology.—Many of the best marked cases of peripheral neuritis are certainly due to *alcohol*, particularly in women, others are decidedly *gouty*, others are caused by *lead*, and others again are *sequelæ* of enteric fever, measles, smallpox, influenza, and other acute diseases, particularly *diphtheria*. The cases which recover cannot, of course, be tested by anatomical investigation; but judging by clinical features, most cases of lead palsy, most cases of alcoholic, and certain cases of syphilitic paralysis may be grouped with those described by Chomel and Graves, Dumeril, Eichhorst, Leyden, Stewart, and Buzzard, while diphtheritic paralysis seems always to be of this nature. It is probable that the so-called "ascending paralysis of Landry" depends in most cases on acute changes in the peripheral nerves.

Other less frequent antecedents of multiple neuritis are certainly arsenic, and perhaps other metallic poisons beside lead, also diabetes and tuberculosis. The anæsthesia and trophic lesions of the skin in *leprosy*, and probably the effect of *ergot* on the limbs, are due to multiple neuritis. So also are the nervous symptoms of the remarkable disease called Beri-beri, of which a brief account was given above (p. 303).

However, we must not forget the important differences between neuritis due to different causes, and must still speak of alcoholic, syphilitic, plumbic, influenzal, or diphtheritic paralysis.

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[*] See plates 3, 4, and 5 in Mr Bowlby's 'Injuries and Diseases of Nerves'

Of the cases of more doubtful ætiology clinically conforming to the character above stated, which have fallen under the writer's observation, one was apparently idiopathic, and occurred in an elderly man with great acuteness and severity; he gradually and slowly recovered. Another, more certainly genuine, occurred soon after an attack of gout, and subsided without local treatment. It was confined to the hands and feet, and showed all the features above described. A third was also in a gouty patient.

In Murchison's 'Treatise on Fever' (p. 191) he narrates as a case of "general paralysis" that of an army surgeon, who, after suffering from the hardships of the siege of Sebastopol, caught typhus fever. He soon after recovery was attacked by pricking sensations in the toes and fingers, followed by numbness, and at last by complete loss of power and also of sensation, "although the slightest handling of the calves of the legs excited the most exquisite pain." The muscles of deglutition were partially paralysed, but there were no pelvic symptoms, and the mind was unimpaired. This is clearly a case of multiple peripheral neuritis following typhus.

Prognosis.—This is generally good; the most typical cases appear to become rapidly worse, then to remain at a standstill, and then slowly to recover. But in practice our prognosis depends greatly on the clinical features of the case, and especially on its origin. The best cases are those due to alcohol, the worst those of neglected lead palsy. Diphtherial paralysis, though often very threatening, may be recovered from even in extreme cases. In one under the writer's care, a girl of thirteen lost power of deglutition, and in all her limbs, in the neck and back. In fact, the only parts moved were the eyes, the tongue, and the diaphragm, together with faint movements of the lips and face, and of the abdomen and chest. Yet she recovered perfectly.

The following varieties of peripheral neuritis claim (if needful) particular mention.

Alcoholic neuritis.—This form of paralysis was mentioned by Lettsom, and was recognised by Wilks as marked by its usual limitation to the lower limbs, its frequency among women, the absence of vesical disturbance, the presence of local tenderness of the muscles, and the remarkable recoveries often seen.

In severe cases the arms as well as the legs are paralysed, and there is great prostration, with loss of flesh.

Similar effects have been observed in cases of poisoning by ether-drinking, particularly in the North of Ireland. In Manchester Dr Judson Bury says that excess in the use of beer is as frequent a cause as excesses in spirits. In London, and particularly among women, the antecedent would seem almost always to be brandy- or whisky-drinking. It is a somewhat rare complication, even in patients of confirmed intemperate habits.

The most frequent age for this form of neuritis is between thirty and sixty, and it is certainly more common in women than in men. Beside the muscular weakness and anæsthesia there are shooting neuralgic pains and numbness, "pins and needles," formication, and other paræsthesiæ, particularly the feeling of clumsiness which gives the impression of the fingers being clothed in thick woollen gloves, and the feet in thick, solid, elastic boots, which prevent treading firmly on the floor. In addition to tenderness of the muscles on pressure, there are sometimes severely painful cramps. There is also increasing atrophy of the affected muscles.

If the symptoms spread beyond the legs and arms they affect the tongue and lips, or the diaphragm, intercostal and other respiratory muscles; but they seldom or never attack the face generally, the muscles of the eye, or those of the bladder. Nor do they appear to affect the cardiac muscles or those of respiration. The pains are often severe, and the tenderness of the paralysed muscles extreme. The cerebral functions are unaffected, and there are no pelvic symptoms (retention or incontinence of urine, cystitis, and bedsore); but the knee-jerks are completely lost, and the reaction of degeneration is marked.

These cases of alcoholic paraplegia are of remarkably favourable prognosis, and almost always recover under treatment. The first necessity is to cut off alcohol, the second to feed the patient as plenteously as possible with starchy and fatty food, and the third to give strychnia. This drug is useful whether given as tincture of nux vomica in a bitter infusion, or as the alkaloid in acid solution. But in severe cases it is better to use it by injection under the skin, or into the affected muscles, until improvement takes place. If any stimulant is necessary, porter is by far the best to prescribe. Under this plan of treatment, followed up by shampooing the affected limbs when the subsidence of tenderness allows of it, and by moderate application of interrupted galvanism, and afterwards of faradism, the writer has seen very severe cases completely recover. Indeed, as Wilks long ago remarked of "alcoholic paraplegia," the patients generally do well.

The special characteristics of diphtherial paralysis have already been described (p. 328); and we have referred to the cases of peripheral neuritis which sometimes occur in the course of diabetes (p. 445). Another form of the lesion will come under notice in the chapter on Leprosy.

Epidemic multiple neuritis.—It has now been ascertained (chiefly by the accurate investigations of Scheube in 1884) that multiple neuritis is the lesion which causes many of the symptoms of the remarkable disease called Beri-beri [*] (cf. *supra*, p. 303).

There are pains in the course of the nerves with patches of anæsthesia, cramps, numbness, and loss of knee-jerks. The paralysed muscles suffer atrophy as in other cases of peripheral palsy, and are very slow in recovering. The cord, brain, and cranial nerves are unaffected, and there appears to be absence of trophic lesions of the skin or joints.

The distribution is first and chiefly in the calves and other muscles of the legs, the thighs, and the buttocks (the hip muscles not escaping as they do in alcoholic paralysis), next in the arms and hands, and less frequently in the trunk. The muscles of the face and eyes escape, but the diaphragm and the cardiac muscle may suffer, and sudden death from syncope is not uncommon, as in cases of diphtherial paralysis. The reaction of degeneration is present from the first, and the histological changes in the affected nerves and muscles are those of acute parenchymatous neuritis with muscular atrophy. Pekelharing of Utrecht confirmed in 1887 the anatomical facts discovered by Scheube, and the constant presence of the specific microbe described by Ogata, a Japanese pathologist.

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[*] *Synonyms.*—*Kakké* in Japan—*Kaki-lembut* (*i. e.* weak legs; a Malay term). The origin of the word *Beri-beri* is said to be a word in Ceylon meaning severe sickness. See Mr Wm. Anderson's 'Lectures on Japanese Beri-beri,' 1879; Morehead ('Br. and For. Med.-Chir. Rev.,' 1855), Fayrer ('Med. Times and Gaz.,' 1880), and Vilette ('Brit. Med. Journ.,' April 23rd, 1887), and a paper by Melotti abstracted in the 'London Medical Record' for January, 1889, p. 13. Dr Buzzard quotes Harada ('Die Japanische Kakké,' 1882) and Palm ('Edin. Clin. and Path. Journ.,' September, 1884). A full account of Beri-beri is given by Dr Manson, who has had extended opportunities for observing its various forms ('Allbutt's System,' vol. ii, p. 439). See also Surgeon-Major Brodie's account of an epidemic of Beri-beri in Madras ('Brit. Med. Journ.,' July 20th, 1889), and Dr A. W. Sinclair's of the disease at Singapore and Sarawak at the same meeting of the British Medical Association at Leeds in 1889.

*Peripheral neuritis from lead.**—The most common effect of chronic poisoning by lead upon the nervous system is a peculiar form of motor paralysis, known as "the dropped wrist."

This attacks the upper limbs by preference, and the extensors of the hand are the chief ones to suffer. The patient's hand hangs powerless from his wrist, and his fingers are more or less forcibly flexed. When the forearm is laid prone upon a table he is unable to turn the palm uppermost, but the supinator longus generally escapes. The muscles which form the ball of the thumb are affected very frequently and sometimes before any others; also those of the little finger, the lumbricales and the interossei. In some cases the deltoid muscles are first attacked, and with them the lower part of the trapezii† and the muscles which cover the dorsal surface of the scapulæ. The affected muscles always become atrophied. Thus the ball of the thumb, instead of being rounded, is sunken; the loss of substance in the lumbricales causes the flexor tendons to be visible in the palm of the hand; and the back of the forearm is hollowed out and flaccid. If the shoulder is attacked, it loses its roundness, and the outlines of the bones can be felt plainly. In some cases the whole upper limb may be weak and all its muscles more or less wasted, but the biceps and triceps in the arm and the flexor muscles in the forearm are never so much affected as the extensors.

Most frequently both upper limbs are attacked, but often one more than the other. Sometimes the paralysis is limited to one arm and hand.

The paralysis which occurs in chronic plumbism is not always confined to the upper limbs, and twice the writer has seen lead-palsy confined to the legs: here the peronei are first attacked. In two instances at Guy's Hospital the whole body was affected, so that the patient could not walk nor raise himself in bed.

The affected nerves and muscles are tender on pressure, but lead paralysis is less painful than that of alcohol. It was formerly a question whether lead-palsy is due to an affection of the nervous centres in the cord, or to the direct action of lead upon the muscles. John Hunter examined the tissues of a painter paralysed hand and arm, and he found that the muscles were cream-coloured. Some years ago a man died in Guy's Hospital who had been attacked with lead paralysis seven years before, and had never completely recovered from it. Dr Moxon found that the affected muscles—especially the deep extensors of the forearm—were represented only by loose watery connective tissue. This muscular atrophy is, however, probably always secondary.

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* *Synonym.*—Saturnine palsy. This term and the corresponding one of Saturnine gout are survivals of the astrological chemistry which connected the seven principal metals with the "seven stars"—gold with the sun, silver with the moon (witness "lunar caustic"), iron with Mars, quicksilver with Mercury, tin with Jupiter, copper with Venus (*diva potens Cypri*, the island being so named from its copper mines), and lead with Saturn.

† This curious immunity of the upper part of the trapezius also obtains in progressive muscular atrophy.

The tenderness of the muscles, and the marked electrical reaction of degeneration shown by the paralysed muscles, shows that lead palsy is due to peripheral neuritis (perhaps beginning in the anterior roots of the nerves) and not to disease of the cord, or to primary atrophy of the muscles. And the microscopical features of the affected nerves confirm this view.[*]

There is a close clinical relation between the symptoms of lead palsy and those of chronic atrophic spinal paralysis and primary muscular atrophy. One distinction between them—on which Sir William Gull used to lay stress—is that in chronic plumbism the patient complains of pain when one grasps the affected muscles with one's hand. This we now know to be due to peripheral neuritis. There are no fibrillary tremors such as accompany progressive atrophy. But the most remarkable distinction is the reaction of degeneration. In lead paralysis faradism causes but slight contraction or none at all, whereas interrupted galvanism gives rise to movements more readily than in health,—that is, a smaller number of cells is required to excite the muscles to contract, and the other peculiarities of the reaction of degeneration are well marked. They are absent in primary muscular atrophy.

The exemption of the supinator longus is so constant as to be often of help in diagnosis. The gums must be examined for the pathognomonic *blue line ;* its peculiar characters will be described in the chapter on Lead Colic. The patient may have suffered from abdominal pain with constipation; but, on the other hand, he may have been slowly absorbing the metal in minute quantities for a long time, until paralysis appears without other symptoms, and these are precisely the cases which are difficult of diagnosis. In most cases of chronic plumbism there is a peculiarly sallow and anæmic aspect of the patient.

Another nervous affection which is said to be an occasional result of lead poisoning is a *tremor*, resembling that caused by mercury. It has hitherto only been observed by Brockmann, among the miners on the Hartz. It is generally limited to the arms and hands, but attacks also the lips and the angles of the mouth. Very rarely it attacks the legs and the muscles of the head and trunk.[†]

Treatment of peripheral neuritis.—In gouty and syphilitic cases the treatment should be directed to their respective causes. In "idiopathic" cases no therapeutical measures are at present generally accepted, and it is most rational to treat them, not "on general principles," but according to the "indications of the disease" as successive symptoms demand relief. One plan, however, seems to be almost always beneficial, namely, the application of slowly interrupted galvanic currents.

In alcoholic cases, strychnine is the most important drug, and often acts better when injected under the skin than when taken by the mouth. In one severe and protracted case in a clergyman's wife, the writer saw its prolonged use result in complete recovery. Abundance of fattening food, and entire abstinence from spirits are essential, but in some cases porter with meals helps both appetite and sleep and does not appear to do any harm.

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[*] See a paper on "The Histology of the Nerves and Muscle-spindles," by Drs Laslett and Warrington in 'Brain,' 1898, p. 224.

[†] Other effects of plumbic intoxication are referred to under the headings of Epilepsy and Insanity, Lead Colic, Chronic Interstitial Nephritis, Saturnine Gout (p. 475), and *Anæmia*. It is also a cause of abortion.

In cases of peripheral neuritis depending on plumbism the most useful treatment is by iodide of potassium. It probably acts partly as a diuretic, and partly by forming with the lead a proteid compound which can be absorbed into the blood. The lead can be found in the urine when the iodide has been given, and when there was before no blue line visible on the gums it may appear there (or at the anus) as a granular deposit of plumbic sulphide. Sulphur baths have been supposed to be serviceable. They lead to the excretion of some of the lead which has been accumulated in the body; and a blackish discoloration of the skin and the nails is observed, evidently due to the conversion of some of the lead into a sulphide.

Locally the application of a galvanic current, just sufficiently powerful to excite contraction of the affected muscles, is very useful. As soon as they will react to faradism, that form of stimulation may be substituted.

AFFECTIONS OF THE SPINAL CORD

PARAPLEGIA

Παραπληγίη δὲ πάρεσις ἁφῆς καὶ κινήσιος, ἀλλὰ μέρεος, ἢ χειρὸς ἢ σκέλεος.—ARETÆUS.[*]

Arrangement of diseases of the cord proposed.—Anatomical points.
Symptoms of Paraplegia generally—Localisation, transverse and longitudinal—Sensory symptoms—Physiological interpretation of symptoms—Incomplete paraplegia—Reflex symptoms—knee-jerk and clonus—Muscular atrophy and muscular rigidity—Pelvic symptoms—the bladder and urine in paraplegia—the rectum and genitalia—bedsores.
EXTRINSIC PARAPLEGIA—*Compression of the cord from vertebral caries or cancer—from aneurysm, hydatid cyst, or meningeal tumour, or hæmorrhage—Symptoms, Diagnosis, Prognosis, and Treatment of paraplegia from compression.*
INTRINSIC PARAPLEGIA—*Myelitis—Pathology and histology of acute myelitis—Localisation, transverse, ascending, disseminated, diffuse—Ætiology—Course and symptoms of acute myelitis—Diagnosis—Prognosis—Treatment.*
Paraplegia due to Tumours within the cord—Syringomyelia—Hæmorrhage—Anæmia and Congestion of the cord—Divers' palsy—Concussion of the cord.
Acute Ascending paralysis of Landry—Alcoholic and Syphilitic paraplegia.
Reflex and Hysterical paraplegia—Neurasthenia spinalis—Rachialgia.
Hemiparaplegia—Its pathology and symptoms, origin and course.
Chronic Myelitis or Sclerosis of the cord.
SPASTIC PARAPLEGIA—*in adults—in children—Primary and secondary—Anatomy—Mixed forms—Symptoms—Prognosis and Treatment—Cases.*
SPINAL MENINGEAL AFFECTIONS—*Hæmorrhage—Meningeal tumours—Acute and chronic spinal meningitis.*

OUR knowledge of affections of the spinal cord has of late made great progress; positive diagnosis can now be made of lesions which were unknown to the pathologists of the last generation. But the exact diagnosis of many forms of spinal paralysis still remains a matter of presumption rather than of certainty, for it is based mainly upon our knowledge that under given circumstances one kind of disease is more frequently met with than another. No doubt the same interpretation of symptoms by probability often guides our diagnosis of diseases of the brain or the abdomen;

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[*] Paraplegia is a palsy of touch and of movement, but of one part only, as hand or leg.

but there is no region in which the results of an autopsy are so often unexpected as the spinal cord.

Classification.—The arrangement of diseases of the cord is not easy. Very few can be grouped on an ætiological system, since their origin is often most obscure. The pathological processes are comparatively few, and vary greatly in the symptoms they produce according to their exact seat. Anatomical lesions of the cord are peculiarly difficult to determine, from the frequent slowness of their course, from the difficulties of histological examination, and from the rarity of some of the most obscure. Moreover certain affections of the cord have, so far as the most skilled observers working with modern methods can determine, no anatomy at all. A clinical classification is, therefore, in the present state of our knowledge, the best; but the distinction between functional and organic affections, the local distribution of chronic myelitis, and such ætiological facts as bear upon treatment or prognosis must also be regarded.

The plan here adopted will be to describe, first, the most frequent and obvious result of lesions of the cord, namely, Paraplegia. Next will follow a more particular account of paraplegia as the result of direct compression of the cord *from outside.* We will then discuss the special characters of Myelitis, and of other causes of paraplegia which operate *within the cord.*

The anatomical and clinical characters of *spastic paraplegia* will follow; and an account of the symptoms produced by affections of the spinal *meninges* will complete the present chapter.

The *atrophic* forms of spinal paralysis—some paraplegic, but others not—are conveniently grouped together, and will form the subject of the succeeding chapter.

Most of these various spinal paralyses are defined by the anatomical lesions to which they have been traced; but there remain two remarkable nervous disorders, which also (while associated with definite changes in the cord) have far more constant and characteristic clinical features than the preceding affections, so that each may be regarded as a natural and connected series of events. These two "diseases"—Tabes and Insular Sclerosis—complete the list of spinal maladies, and will occupy the third and fourth chapters of the present section. The latter, being cerebral as well as spinal in its anatomy, forms a natural transition to the next section on Diseases of the Brain.

PARAPLEGIA.—This form of paralysis has two essential characters: first, that it affects both sides of the body, generally to an equal extent and in the same degree; and secondly, that it affects all parts, up to a certain level, according to the functions of the several pairs of spinal nerves, ascending from the sacral to the cervical.*

A "segment" of the cord is included between two adjacent horizontal sections. It takes the whole thickness of the cord, and a single pair of nerves. We thus recognise eight cervical, twelve dorsal, and many lumbo-

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* This is the modern meaning of the word, dating from the last century. Thus, Cullen defines "Paraplegia" as *paralysis dimidii corporis transversim sumpti.* But Hippocrates and Galen applied the word *παραπληγια* or *παραπληξια* (for both forms are used) to paralysis of a single limb, and distinguished it from apoplexy as a "side-stroke," *i. e.* a partial palsy only. See the quotation from Aretæus on chronic diseases printed at the head of this chapter (Lib. i. cap. vii). In 1703 paraplegia is defined as *paralysis quæ omnes corporis partes capiti subjectas occupat* (Blancardi 'Lexicon Medicum').

sacral segments, although the last are too closely packed together for us to be able always to distinguish their several lesions.

It must be borne in mind that there are important differences between the segments, in the proportion of grey to white matter, and the size of the anterior cornua; and also in the development of the several "tracts" of the cord.

Thus, the anterior cornua are very large in the cervical and lumbar enlargements, very small in the dorsal region; while the important group of cells known as Clarke's vesicular column is well represented in the dorsal (especially the lower dorsal) region, and is almost absent in the cervical and lumbar. Again, as we pass downwards, the crossed pyramidal tract becomes smaller and more superficial; the direct cerebellar and direct pyramidal tracts gradually disappear; and the postero-median column of Goll becomes smaller and deeper.

It must also be remembered that the lumbar or lumbo-sacral enlargement is so named from its giving origin to the nerves of the lumbar and sacral plexuses, but that it does not, like the cervical and dorsal regions, correspond to the vertebræ of the same name. The cord ends, in the adult, at the first lumbar vertebra, and from the mid-cervical region the nerves pass downwards to their foramina of exit from the vertebral canal, with increasing obliquity, until they are collected together to form the cauda equina. Thus, for example, the origin of the great sciatic nerve is in the upper lumbar region of the spine, and its constituent strands pass through the sacral foramina twelve inches or more from their origin.

Moreover the dorsal spines slope downwards so that their subcutaneous apices felt by the finger are one, one and a half, or two vertebræ below the exit of the corresponding nerves. Dr Reid ('Journ. Anat. and Phys.,' vol. xxiii, 1889) found the seventh cervical spine on the level not of the seventh cervical but first thoracic nerve-root, and the sixth thoracic on a level not with the origin of the sixth but of the ninth thoracic nerve.

Localisation.—The lesions which produce Paraplegia are various—mechanical pressure, acute and chronic inflammation, new growths, cysts and hæmorrhage, some originating in the cord itself, others outside it. They also vary as regards their distribution in the length of the cord. They may be strictly confined to a single segment, or may extend from the cauda equina to the cervical region, or may transcend the limits of the cord and invade the bulb, the pons, or the cerebrum itself.

The *upper limit* of a spinal affection is indicated by the extent of the paralysis to which it gives rise. If a total transverse lesion affects the lumbar enlargement, it causes paralysis of the lower limbs; a similar lesion in the dorsal region causes in addition paralysis of the abdomen and chest, corresponding with the level to which it reaches; one affecting the cervical enlargement adds paralysis of the upper limbs; and disease still higher up produces paralysis of the diaphragm and of the cervical muscles in addition to that of all the parts below, and must be rapidly fatal, by preventing the respiratory movements.

Between the paralysed and normal districts there is often a zone of hyperæsthesia, which is a good mark for the upper limit.

The *lower limit* of a spinal affection is more difficult to fix, since a complete transverse lesion of the cord will paralyse all the fibres below. Inasmuch, however, as the cord is a collection of centres as well as of conducting tracts, we can to some extent define the lower limits by tudy of its reflex functions. Thus in some cases the feet and legs ome powerless before the thighs and the hips. This is generally sup- ed to mean that the lesion is at first confined to the extreme lower end the lumbar enlargement; but, since Woroschiloff found that in the eral columns of the lumbar cord of the rabbit the motor paths for the tal parts of the lower limbs lie outside those for the proximal parts, it been thought that the symptom in question may sometimes be due to dual extension of disease from the surface inwards.

When paralysis attacks the upper extremities, as the result of disease eading upwards through the cord, the muscles of the hands are as a e affected before those of the elbow and the shoulder. The fact that nerves to the ulnar side of the hand come chiefly from the lowest part the brachial plexus would lead one to expect that the inner fingers ld suffer earlier than the outer fingers or the thumb.

Apart from their distribution in the length of the cord, its lesions er in their transverse extent. Most of those which cause complete para- gia are total transverse lesions. Others affect the region around the tral canal, the anterior cornua, the lateral or the posterior columns; these partial lesions cause only partial paralysis.*

Sensory symptoms.—When any segment of the cord is completely des- red, there is of course an absolute loss of *sensation*, as well as of ion, in all parts of the body below the lesion. And when a total dis- anisation spreads upwards through the substance, the gradual progress he disease from day to day can be determined from the extension of the esthesia with far greater accuracy than is possible from that of the or paralysis alone. But in most cases of paraplegia the sensation of the ts affected is but little impaired.

This preponderance of loss of motion (*akinesia*) over loss of sensation *esthesia*) is not only met with in spinal affections, but also appears in ases of the brain, and sometimes in those of the peripheral nerves.

Beside the loss of touch in paralysed limbs, there is loss of the sense emperature, of the muscular sense, and of the sense of pain (*analgesia*). ny paraplegic patients complain of "pins and needles," "pricking" or ngling" in the toes or in paralysed parts, "creeping" or "crawling" *rmicatio*), or subjective sense of heat or of cold—perverted sensations ich are conveniently called *paræsthesiæ*.

Course of fibres in the cord.—All physiologists are agreed that volitional tor impulses pass mainly along the crossed pyramidal tracts of the ero-lateral columns. With regard to the paths for sensory impulses, re still is doubt. One opinion, maintained by Brown-Séquard, was that y are all situated in the grey matter. Another, which was first taught Schiff, is that tactile sensations are transmitted through the posterior mns, but sensations of pain through the grey matter. There is good hological evidence, as we shall hereafter see, for the latter statement. it seems probable that the track of common sensation lies not in the matter nor in the posterior columns, but in the deeper part of the ral columns. The sensory tracts do not cross at one level like those of

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According to Erb ('Ziemssen's Handbuch,' xi, 2, p. 62) paralysis of all four limbs, of the trunk muscles generally, sometimes occurs without any interference with the ratory movements, as the result of an exceptional limitation of disease to a part of egmental area of the cord in the cervical region, leaving the lateral columns intact. tly *speaking, such an affection is not* paraplegic in the sense defined above.

motion, but gradually pass over, so as to reach the brain on the opposite side to their periphery; the passage is begun at the line of the posterior roots of each nerve, and probably most of the sensory fibres have crossed at the level of two or three segments higher.

The motor tract then, starts from the cortical centres in the ascending frontal and parietal convolutions and parts adjacent, on, say, the left side, passes down the corona radiata through the left corpus striatum by the fibres of the external and internal capsule, but without direct communication with the grey matter (caudate or lenticular), and, continuing through the crus of the same side, passes "under" the pons to the bulb, where most of the fibres cross over to the opposite side, pass down the lateral columns as the crossed pyramidal tract of the right side, and end by joining the large cells of the anterior cornua. Taking a fresh start from these, the motor tract leaves the cord by the anterior roots, and continues through the mixed nerves to supply the muscles and blood-vessels of the right half of the trunk.

Occasionally in man, as in animals also, the crossing of the anterior pyramids is more or less incomplete. And normally a small band of fibres do not cross at all, but continue down on the same side as the direct pyramidal motor tract or antero-median column of Türck, to the anterior cornua and anterior roots on the same (left) side.

The sensory tract starts from the skin, the joints, and other end-organs, passes into the cord through the posterior roots, traverses the corresponding root-zone, crosses at once or soon after to the opposite side, runs deeply in the lateral white columns and the grey substance up to the bulb, where it joins the corresponding motor tract. It passes "under" the pons and through the crura cerebri to the corpus striatum, where it lies to the inner and posterior side of the motor tract, and finally runs through the corona radiata to the sensory centres in the cortex, of which (except those of sight, hearing, smell, and taste) the locality is at present undetermined.

The descending and ascending tracts of the cord do not completely correspond with the motor and sensory tracts. They are so named because the progress of secondary degeneration follows in certain regions of the cord, and either downwards or upwards, the latter process corresponding to that of degeneration of a motor nerve when severed from the nuclear part of its neuron (*i. e.* one of the large cells of the anterior cornu), and to that of degeneration of the sensory nerve when severed from the fusiform cells of the ganglion on the posterior root.

The descending tracts are—(1) the crossed pyramidal tract in the lateral column, deeply situated in the cervical and thoracic regions, but becoming superficial as it becomes smaller in the lumbar cord, until it disappears in the last sacral nerves.

(2) The direct pyramidal tract as above described, a small column on each side of the anterior median fissure, which becomes deeper and vanishes at the level of the last thoracic nerve.

(3 and 4) Two slender tracts: one in the antero-lateral part of the cord, which does not touch the surface at all, and is only present in the cervical and dorsal regions: the other still smaller, more slender and deeper, is best marked in the cervical region, where it lies deeply in the "posterior" columns between the "posterior" (or superior) grey horn and the "posterior" median fissure.

ascending tracts are (1) the posterior-median columns of Goll,
un up the whole length of the cord and end in the posterior
s of the bulb, and so by the inferior peduncles are probably con-
ith the cerebellar centre; (2) the direct cerebellar tract, a slender
al band of coarse medullated fibres which occupies the postero-
urface of the cord from the upper lumbar nerves upwards, and
more extensive, though not thicker, as it passes up to become
us with the corpus restiforme or inferior peduncle of the bulb, and
the cerebellum; and (3) a thin tract named after its discoverer,
which continues the cerebellar tract forwards. It is nearly iden-
h one described by Stilling.

ial paraplegia.—Complete motor paraplegia, in which the affected
are altogether incapable of voluntary movement, is, as a rule, due
y. As the result of disease, the paralysis is usually incomplete,
y degree of loss of power may be noticed. One patient can just
s toes, another can flex the knees so long as they lie horizontally
ed; a third can draw the thighs up to the abdomen; a fourth can
r an instant and then falls down; a fifth can walk a few paces;
n, up to a condition in which all that can be detected on the most
xamination is a slight uncertainty of step, with a tendency to catch
against any slight obstacle.

describes the gait of paresis,* as partial motor paralysis is called,
ollowing terms:—"The foot hangs down in walking, the toes are
, the sole is brought to the ground clumsily, and for the most part
s outer edge; the knee is too much raised or carried forwards
being bent; there is generally a kind of stiffness about the legs.
ient uses one or even two sticks, or he is held up by crutches or
arms of other persons; he totters but slightly, and stands quietly
dily. When left unsupported he sinks upon the ground. The gait
e varies somewhat according to the number of muscles which are
d. When only those below the knees are affected, it is waddling
y peculiar."

lysis of sensation likewise admits of every degree, and, in addi-
difference in kind, as sensations of touch, of temperature, of pain,
scular action are affected.

ough, according to the rule above stated, in cases of slight para-
næsthesia is as a rule still less marked than loss of motion, yet in
cases it is much more affected, particularly in those of syphilitic
unctional ("hysterical") origin.

me cases of motor partial paraplegia with impaired sensation the
feels when he stands upon his feet as though they were wrapped in
or covered with thick woollen stockings; or a hard floor may seem
ike a thick felt carpet. In such cases he requires the guidance of
enable him to stand firmly or to walk evenly. When a patient
his feet close together totters or falls as soon as he is made to
s eyes, he is often assumed to be suffering from locomotor ataxy.
ough this symptom is present in tabes, it is also met with when-

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term paresis (*πάρεσις, παρίημι*) is in Greek a mere synonym of *παράλυσις*, both
to the Latin term *nervorum resolutio*, and carries no connotation of diminution;
e modern application of it is arbitrary, and not of much practical use, since all
vary in intensity. Aretæus limits the word quite as arbitrarily to loss of power

ever there is an imperfect transmission of those sensory impulses from the feet by which equilibrium in the erect posture is maintained.

Reflex symptoms.—We have said the lower limit of a lesion of the cord cannot be determined with any great accuracy. Disease in the cervical region renders the legs as well as the arms paraplegic, and this equally whether the affection is confined to a small part of the length of the cord or involves the whole of it down to the cauda equina. If, however, reflex movements can be excited in the legs, it proves that the lumbar enlargement is still intact. On the other hand, their absence is not conclusive evidence that the disease extends throughout the whole of the lower part of the cord. On the contrary, it often happens in cases of very limited lesions that reflex movements can only now and then be elicited. For example, surgeons find, when there is fracture with displacement of the upper dorsal or cervical vertebræ, that for a few days after the accident there is an entire absence of reflex excitability in the cord; it seems to have been in some way deprived of its functions by the shock.[*] Again, reflex movements are, as a rule, less readily obtained when disease has begun within the cord than when it is merely compressed from without.

Reflex movements are more or less co-ordinated. There seems no reason to suppose that muscles are individually represented in the spinal centres; and whatever may be the machinery in the brain by which the several groups of muscles are harmonised and co-ordinated in their actions so as to produce definite movements, it is certain that cross-connections exist within the cord by means of which a similar result can be brought about. We do not see such marked purposive reflex movements in a paraplegic patient as are observed in decapitated frogs; but a near approach to them was seen in a case of Dr McDonnell's, recorded in the 'Dublin Quart. Journ.' for 1871. The patient was paralysed in all four limbs; when a catheter was passed into his bladder his left arm would "wriggle up and down, and the hand come fluttering over the genital organs, although he was absolutely unconscious of the operation, and even when his eyes were shut." Another curious case is related by Virchow. It is that of a man who was paraplegic, and who generally lay in bed with his lower limbs rigidly flexed. When he wanted to stand upright he would pinch the right thigh sharply, or give it a smart blow on its outer surface; this threw both the legs into a position of extension; still the gastrocnemii remained contracted, and before he could put his foot to the ground he had to bring down the heels with both hands ('Gesammelte Abhandlungen,' p. 684). Another illustration of the way in which one set of reflex actions may be made to counteract another is afforded by a case of Dr Budd's. The patient was recovering from paraplegia and could walk a few steps, until the contact of the soles of his feet with the ground would make his knees bend beneath him; this he was able to overcome by rubbing the surface of his belly, when the legs were extended with a jerk.

As a rule, when reflex movements are excited by impressions upon the cutaneous surface, they remain confined to the limb on the same side. Leyden says that they do not spread to the opposite limb except when the lesion is in the bulb, but many instances to the contrary may be found

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[*] See the instructive remarks of Prof. Goltz on this subject in his 'Verrichtungen des Grosshirns,' S. 78—81, and the important paper by Dr Bastian ('Med.-Chir. Trans.,' vol. lxxiii).

The movements generally consist in a rapid series of intermittent (*clonic*) spasms; but sometimes the contraction is continuous (or *tonic*) in character.

The following reflexes are of diagnostic value for localising symptoms in cases of paraplegia.

(1) Cutaneous reflexes are obtained by touching, pinching, or stroking the skin so as to produce contraction of the underlying muscles. The following are the most important of these superficial reflexes:—The *plantar*, tickling the sole of the foot so as to cause the leg to be drawn up; *cremasteric*, tickling the skin supplied by the femoral branch of the anterior crural nerve, and causing the testicle on that side to be drawn up; the *abdominal*, when the external oblique muscle contracts; the *epigastric*, when tickling the skin over the fourth to the sixth ribs produces contraction of the upper segment of the rectus; the *gluteal*, when irritation of the skin of the buttock leads to dimpling over the insertion of the gluteus maximus into the fascia lata; and the *scapular*, a slight movement of the teres major in the fold of the axilla when the skin between the shoulders is pinched. All these are most easily produced in children; and others may be observed at an early age, as the *palmar* reflex or closure of the fingers produced by gently touching the palm of a sleeping infant. In elderly people several are absent, and all are less readily elicited.

As criteria of the seat of a spinal lesion, the *plantar* reflex corresponds to the sciatic nerve (first, second, and third sacral) and lower part of the lumbar enlargement of the cord, situated in the adult opposite the last dorsal spinous process; the *gluteal* reflex corresponds to the fourth and fifth posterior lumbar branches, the lumbar enlargement, and inferior gluteal nerve; the *cremasteric* to the first and second anterior lumbar branches; the *abdominal* to the lower three or four intercostal nerves and dorsal cord; the *epigastric* to the middle intercostals (fifth to seventh), and the upper dorsal cord; and the *scapular* to the upper bundle of the brachial plexus (last cervical and first dorsal nerves), and the cervical enlargement of the cord.*

In most cases tickling of the sole, besides causing retraction of the whole limb, is followed by arching of the foot; but in young children the great toe is not flexed but extended; and this "extension" form of the plantar reflex is a symptom of some importance when it occurs in adults.

(2) There are what may be termed Visceral Reflexes:—*vesical*, when the bladder is caused to contract by irritation of the urethral orifice, of which the incontinence of urine in children, by reason of a long prepuce, is an example; *penile*, when erection is produced by the passage of a catheter or by the slightest contact, a condition sometimes seen in cases of partial paraplegia, short of that which produces priapism; *rectal*, as when contraction of the bowel follows the introduction of a suppository; lastly, the familiar reflex actions of *vomiting* from tickling the fauces, *sneezing* from a draught of cold air or a brilliant light, *blinking* from touching the cornea, and contraction of the *pupil* to light and during accommodation for a near object. The movement of the ribs in *respiration* enables us to judge of the integrity of the connection of the intercostal nerves with the brain, or of the point where it is severed by transverse lesion of the cord. The act

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* See Dr Gowers' useful diagram ('Diagnosis of Disease of the Spinal Cord,' p. 58).

of *swallowing* depends on the centre situated in the bulb, and that of *contraction of the iris* on the centrum ciliospinale in the same region.

(3) A closely connected set of phenomena have been called *deep reflexes*, but it is still doubtful whether they are reflex or due to direct stimulation of the muscles involved.*

The most important was first described in 1875, by Westphal and Erb independently; it was called by the latter the "patellar tendon-reflex," by the former the "knee-phenomenon." † The best English name is *knee-jerk*, and it has long been known to schoolboys. A smart tap is given with the side of the hand upon the tendon of the great quadriceps extensor muscle of the leg, above or below the patella, while the knee is crossed over the opposite thigh; and the foot is jerked upwards. A similar *elbow-jerk* may be obtained in most healthy persons by striking the stretched tendon of insertion of the triceps extensor brachii.

The absence of the knee-jerk is usually, perhaps always, a morbid sign; but different persons vary much in the readiness with which it is procured. It is exaggerated in most cases of paraplegia, but contrary to what we should expect, is as a rule diminished in the paralysed leg of hemiplegia. In morbidly excitable conditions the jerk may sometimes be obtained by stretching the ligamentum patellæ and striking the subcutaneous surface of the tibia; or, by stretching the tendo Achillis and tapping the tibialis anticus and extensors of the toes, an extension of the foot may be obtained (Gowers' front-tap contraction).

The so-called deep reflexes do not always increase or diminish in correspondence with the superficial reflexes.

Ankle-clonus is the name given to the clonic contractions produced in some patients by forcibly bending up the foot so as to make the tendo Achillis tense. It was first described by Charcot, and is included by Erb and Westphal among tendon-reflexes. It usually goes with exaggerated knee-jerk, and is probably always a morbid phenomenon. But a somewhat different kind of clonic spasm of the gastrocnemius can be obtained in most healthy persons by bending the knee and ankle with the ball of the foot resting on the ground, and moving the knee rapidly up and down.

The paralysed muscles.—The condition of the centres in the cord ha direct influence on the nutrition of the paralysed muscles.

When disease destroys the entire substance of the cord up to a cert level no reflex movements of any kind can be obtained in the limbs bel Under these circumstances the muscles rapidly lose their electrical tractility, and they undergo marked *atrophy*. Before complete loss susceptibility to galvanic currents supervenes, there is generally a per in which the reaction of degeneration is present.

When paraplegia is due to a lesion limited to one of the upper segme of the cord, so that reflex movements persist in the paralysed limbs, muscles, as a rule, retain their normal electrical reactions, and they rem tolerably well nourished for months and years, or show merely such sl flaccidity and wasting as may fairly be attributed to disuse. They still in connection with their trophic centres in the anterior cornua.

In time such paraplegic muscles usually become more or less *rigid* that the legs are either forcibly extended, or drawn up in a state of flexion. The knee-jerk is exaggerated and clonus is present. Cases of this kind will be described under the name of "spastic paraplegia."

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* See papers by Tschirjew ('Arch. f. Psychiatrie,' 1877, and Du Bois's 'Arch.,' 1 Gowers ('Lancet.' 1876, i, p. 156), Waller ('Brain,' July, 1880), and De Watteville (' Med. Journ.,' 1882, p. 736); also by Bowditch and Warren ('Journ. of Physiology, xi, 1890).

† Gowers, "*myotalic* movements," from μῦς, a muscle, and *τατικός* (*τείνω*), stretc

We may therefore distinguish two kinds of paralysis:

(1) *Atrophic*—with reaction of degeneration, loss of reflexes, and rapid atrophy of muscles, the conditions which we have seen obtain in cases of peripheral neuritis.

(2) *Spastic*—with no reaction of degeneration, only moderate atrophy from disuse of muscles, and marked rigidity with increased reflexes and ankle-clonus, the symptoms present in most cases of chronic paraplegia and also in chronic hemiplegia.

The bladder in paraplegia.—Unstriated as well as striated muscles can be excited to reflex contractions. Erb mentions a curious case of paraplegia, in which a fluid fæcal evacuation was passed whenever a large bed-sore was dressed. In other instances micturition was directly produced by pressure over the bladder, or by the introduction of the finger into the rectum. It does not follow that impulses were reflected to the vesical muscular fibres after being conveyed to the cord by cutaneous nerves, for the slightest compression of any part of the distended bladder appears to be a sufficient direct stimulus to its muscular fibres. Indeed, in the apparently volitional act of micturition in health the influence of the will is really limited to relaxing the sphincter, so that the pressure of the urine on the walls of the bladder may set in action its reflex machinery. The experiments of Goltz upon young dogs ('Pflüger's Archiv,' 1874) have shown that in these animals the bladder is capable of emptying itself at regular intervals, and in a perfectly normal manner, after the cord has been completely cut across.

Thus we might expect that reflex micturition would take place naturally in those cases of paraplegia in which the lesion is limited to the upper part of the cord. In practice, however, it is found that retention of urine almost invariably occurs, even when reflex movements in the lower limbs prove that the lumbar centres are intact. The explanation probably is that an inhibitory influence is transmitted downwards upon those centres from the diseased parts above. Goltz, in fact, states that in some of his experiments it was not until several days after the section of the cord that the animal was able to micturate properly; and he suggests that in cases of injury to the spine in man the bladder would after a time be found to have regained its functions if catheterism were not continued as a matter of routine. However this may be, the accuracy of Goltz's views with regard to micturition is established by some exceptional cases of paraplegia, in which the urine is passed quite naturally. In one of Dr William Budd's cases the urine at first began to flow in interrupted jets, but as the reflex spasms of the legs grew more feeble its stream became continuous. Another patient, at a certain period in his illness, emptied his bladder involuntarily and with a sort of jerk about every two hours. In a third case, one of fracture of the spine, in which there was at first retention, the power of voiding urine was regained on the fifth day, whereas no volitional movements in the legs occurred until the ninth day. So, again, one of the cases related by Ollivier, in his classical work,* is that of a man who had complete paralysis of his lower limbs as the result of caries of some of the higher dorsal vertebræ, but in whom the bladder performed its functions normally.

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* 'Traité de la Moelle Épinière et de ses Maladies,' par C. P. Ollivier: 1st ed., 1821; 3rd, 1837, with plates.

The vesical symptoms not infrequently precede the paralysis of the legs, sometimes by a considerable interval; but it is doubtful whether the lumbar centre for micturition is ever the only part of the cord diseased, so that while the bladder is paralysed the lower limbs permanently retain the power of movement. If such a condition exists, it is very rare.*

In some instances one can distinguish paralysis of the *sphincter* from that of the *detrusor urinæ*. It might have been supposed that in the former case there would be complete incontinence, the fluid dribbling away incessantly. But physiologists have found that even after destruction of the lumbar cord the neck of the bladder can withstand a pressure of six inches of water—as compared with one of twenty inches when the cord is intact; and this result is entirely confirmed by some observations of Hutchinson ('Brit. Med. Journ.,' 1877) upon persons who have the remains of a shrunken spina bifida, involving the fourth pair of sacral nerves, so that the sphincters of the bladder and rectum are paralysed without other symptoms. In such cases he finds that the bladder can hold a large quantity of urine, but if once its reflex contractions are excited the will is, of course, powerless to restrain them. So in many cases of partial paraplegia the patient says that as soon as he feels the inclination to micturate he is obliged to do so at once on pain of an accident. When the detrusor is paralysed a patient complains that he has to strain for a minute or two before he can pass any water, that it comes away slowly in a feeble stream, and continues to trickle after voluntary effort has ceased.

When the "micturition centre" in the lower part of the cord is wholly deprived of its functions by disease or injury, the sphincter and the detrusor muscles are of course paralysed together. The result is that the organ becomes distended, and that the excess of urine beyond what it can hold dribbles away through the urethra. The Germans call this condition "ischuria paradoxa." It has often been mistaken for incontinence; an unskilled nurse is apt to suppose that there can be no retention of urine in the case of a patient who is constantly wet, and her statements may mislead a careless practitioner; but one can always avoid such an error by passing one's hand over the lower part of the abdomen. In cases of incontinence from paralysis of the sphincter alone, the bladder keeps firmly contracted and almost empty, while its walls become greatly hypertrophied. It seems probable that this kind of incontinence always depends upon an exalted reflex activity of an intact lumbar centre.

The urine in paraplegia.—We have still to consider certain changes in the urine, which are of the highest clinical importance. Sir Benjamin Brodie sixty years ago stated (in the 'Med.-Chir. Trans.' for 1836) that in cases of injury to the spine the urine is sometimes voided alkaline, ammoniacal, and turbid as early as the second or third day; and in most cases of paraplegia due to disease of the cord the same thing occurs later. Ammoniacal urine, unlike that which is alkaline from salts of sodium and potassium, is always morbid, and always due to decomposition of urea in the urine already secreted, usually in the bladder, and occasionally in the renal pelvis. Not only those patients who have complete retention pass ammoniacal and fœtid urine, but the same may occur when only slight impairment of the power of the bladder prevents it from emptying itself completely. The difficulty is to understand how the decomposition in the urine is brought about, apart from the access of air and of the *Micrococcus ureæ.* No doubt in former days the cause was the passage of a dirty (or let us say a not aseptic) catheter to relieve retention, as Traube long ago taught. But since then the catheters and the surgeons' hands are alike scrupulously pure, and yet accidents sometimes happen; and now and then a case occurs where the catheter was never used and the urine has become putrid.

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* I have notes of more than one case in which retention of urine has preceded all other indications of the onset of paraplegia by an interval of a few days, but I do not remember any instance in which it has continued to be the sole or even the main symptom of a spinal affection. Some years ago I saw a gentleman who was one of the sufferers in the Thorpe railway collision, and whose chief complaint was that he could pass his water only in a very feeble stream; he said that "it flowed from him like oil," and it quickly became ammoniacal and very fœtid. He had pain and tenderness in the back, but the only sign of any loss of power in the lower limbs was that in walking he sometimes seemed to totter, especially if his foot caught against any slight obstacle; he also had "twitchings" in the legs three or four times a day. He was not long in getting perfectly well.—C. H. F.

Decomposed ammoniacal urine is capable of exciting inflammation of the bladder and of the urinary passages; but in other cases cystitis is no doubt the primary, and ammoniacal urine the secondary event.

After death one finds intense cystitis and suppurative nephritis. The mucous membrane of the bladder is ecchymosed, infiltrated with inflammatory products, lined with a "diphtheritic" layer or with a granular deposit of phosphates, or actually sloughing. Its muscular wall is sometimes greatly thickened; in one case at Guy's Hospital it measured at least a twelfth of an inch across, although the disease had lasted only four or five weeks. The kidneys are enlarged, intensely congested, and full of suppurating points and streaks; in other words, they present all the characters of an "ascending inflammation."

Rectum and genitalia.—Some other symptoms connected with the pelvic organs may be conveniently mentioned here. One of them belongs to the *rectum.* The peristaltic movements of the intestine are not directly dependent on the spinal cord, but the sphincter ani is under the control of a lumbar centre. Consequently it may become paralysed along with the bladder; and incontinence of the fæces sometimes follows retention of urine.

Another symptom concerns the male genitalia. *Priapism* is of frequent occurrence in paraplegia, as one result of the reflex activity of the lower part of the cord. Sometimes it is constant; sometimes it only appears when the bladder is distended, or when a catheter is passed. Goltz, in his experiments on paraplegic dogs, traced out carefully the various ways in which this symptom could be induced by irritating the skin of the abdomen, or of the thighs, or the sheath of the penis, and he found that he could easily inhibit it by simultaneously irritating the sciatic nerve, or by pinching the hind foot. When the lumbar enlargement of the cord is destroyed, priapism is of course altogether absent.

As regards the female organs, it is worthy of mention that *parturition* may take place naturally in a woman suffering from paraplegia, provided that the lesion is above the lumbar centres.

Bedsores.—A fact in favour of the conclusion advocated by Charcot—that morbid changes in the bladder and urine are at least sometimes directly dependent upon an influence transmitted from the nervous centres—is that they often coincide with gangrene of the skin over the sacrum and the gluteal regions. An "acute bedsore" sometimes begins to form as early as the fourth day. Many instances of this kind are quoted by Charcot; but none is more striking than a case recorded by Sir William Gull, of a man who on November 22nd, 1856, was working in the docks, when he felt a sudden pain in the back after lifting some deals; he became paraplegic on the 24th, and on the 26th was admitted into the hospital with a small bedsore already present. It seems clear that in such cases the affection cannot be regarded as a mere result of pressure upon the skin, or of the irritant action of excreta, like the superficial and chronic bedsores which do not appear until after the lapse of weeks or months. An acute bedsore may sometimes be made to heal if carefully attended to; this occurred in the case just quoted, but afterwards another one formed at the same spot, and it rapidly increased in size, so that, when the disease ended fatally, six weeks from its commencement, the whole of the sacrum was exposed. A bedsore may destroy life, either by exhaustion, or by pyæmia, or by extension of inflammation to the vertebral canal.

The four symptoms last described—retention of urine, incontinence of fæces, priapism, and acute bedsores—are apt to go together, and may be conveniently grouped together as the "pelvic symptoms" of paraplegia.

Paraplegia from Compression.—The most simple and "experimental" example of this form of paralysis is destruction of a segment of the cord by sudden violence, as when the back is crushed between two railway carriages or the neck broken low down. In the cases, however, with which we are here concerned, the compression is gradual and slow, so as first to produce pain by interference with the posterior roots of the nerves, then to excite secondary myelitis with softening, and finally to destroy the cord completely at the point affected. Ollivier and Cruveilhier described separately paraplegia which is produced by the "slow compression of the cord;" and, though the importance of this grouping seems not to have been recognised by their immediate followers, Charcot worked out the idea in detail. He included in his description tumours lying in the interior of the cord, although, as he admitted, some of the most characteristic symptoms are absent in such cases.

Causes.—In the first place it is to be remarked that some affections of the spine never cause paraplegia. Thus the cord seems always to escape compression in cases of lateral curvature (*scoliosis*), however extreme may be the deformity.[*] The vertebral canal is seldom, if ever, so narrowed in cases of *osteo-arthritis* as to cause paraplegia.[†] Gowers mentions two cases of vertebral *exostoses* compressing the cord; but syphilitic *nodes* seem never to grow from the bodies or arches inwards, so as to cause symptoms.

1. *Caries of the spine; Pott's disease.*—Putting aside direct violence, the most frequent, and therefore by far the most important cause of "compression-paraplegia" is that which in this country is known as "angular curvature." On the Continent it is universally called after a great English

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[*] We had lately an extreme case of lateral curvature with rotation of the vertebræ in Philip Ward, following a neglected empyema in childhood; but, though much deformed, the patient, a young man of twenty-three, had not the least symptom of paraplegia.

[†] In 1838 Aston Key related in the third volume of the 'Guy's Hospital Reports' two cases of paraplegia, in each of which Wilkinson King—a pathologist of deserved reputation—found an intervertebral substance projecting backwards, with raised lips upon the edges of the two adjacent vertebræ, and in one case with an ossified posterior common ligament bridging over the space between them. But whether this was the cause of the paralysis may perhaps be doubted.—C. H. F.

surgeon of the last century, Percival Pott, of St Bartholomew's Hospital. It consists in the destruction by caries, with or without necrosis, of the centrum of one or more vertebræ, until they give way beneath the weight of the head and upper part of the trunk. The result is displacement of the corresponding arches and spinous processes, which become bent into a sharp angle or more rounded curve.

The first step in this morbid process is still a point of dispute. Perhaps it does not always begin in the same tissue. German pathologists agree in stating that it begins in the bodies of the vertebræ, the discs escaping or being implicated only at a late period; and undoubtedly one may see vertebræ with caseous masses in their interior, or even irregular cavities, which nowhere touch the discs. But, as Wilks long ago pointed out, there is in some cases a source of fallacy in the fact that when a disc is completely destroyed, the adjacent vertebræ often lose each about half of its substance, and the remaining halves, coming together, look exactly like the fragments of a single bone ulcerated through by the disease. Dr Fagge repeatedly found intervertebral discs above and below the main seat of mischief presenting early changes of such a kind as to convince him that in the particular case under observation they, rather than the bones, were primarily affected. Thus, in an instance recorded by Sir William Gull ('Guy's Hosp. Rep.,' 1856, p. 179), death occurred from an affection of the cord at a time when the only change was softening of three of the discs, with the formation of a cheesy substance in the middle one, and a little "absorption" limited to the adjacent part of the bone.

Another question formerly debated, whether caries of the vertebræ is to be regarded as "scrofulous," is now decided—partly by the presence of tuberculous lesions elsewhere, partly by the presence of the characteristic bacilli of Koch. Cases of Addison's disease or tuberculous pyelitis are now and then accompanied by caries of the adjacent vertebræ; and a similar affection occurs in cases of pulmonary phthisis, or tuberculosis of the testis, or disease of other bones or joints. Like tubercle of the joints, caries of the spine can often be traced to accidental injuries.

In most instances of permanently cured angular curvature with ankylosis, the active stage of the disease was passed through in childhood.*

So great is the deformity in many of these cases, and so much does it alter the relations of the vertebræ to one another, that it would not be surprising if the displaced bones often compressed the cord directly. That this does sometimes occur appears clear from a case of Brown-Séquard's, in which paraplegia which had set in suddenly was removed in twenty-five hours by extension of the spine. But every pathological museum contains specimens which show that the spinal canal usually remains of its full width, however much its direction may be altered. Moreover, as Charcot observed, paralysis often occurs in cases of vertebral caries in which there is no curvature at all; while in other cases, in which curvature exists, the patient regains the use of his limbs, although the state of the bones remains unaltered. The immediate cause of pressure on the cord when there is caries of the spine is, as Gull showed in 1856, a mass of caseous material, which collects between the carious vertebræ and the dura mater, after destruction of the posterior common ligament. This material is identical with that found in phthisical vomicæ, in Addison's disease of the adrenals, and in old tuberculous abscesses; it consists of dried-up pus, tubercle bacilli, and phosphates and carbonates of calcium and magnesium.

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* Out of sixteen cases, all causing fatal paraplegia, in fifteen the patients were adults, four between twenty and thirty years of age, three between thirty-one and forty, seven between forty-one and fifty, and one fifty-six. The only case in which ankylosis existed was one in which the spinal disease began when the patient was six years old, and he died at thirty-two.—C. H. F.

Much of the pus secreted by the carious body of vertebra passes forwards to form a collection behind the anterior common ligament.

2. *Malignant disease of the spine.*—This is the only other frequent cause of extrinsic paraplegia. It may be secondary to carcinoma of the breast, or to cancer of the œsophagus or colon, the uterus or testicle, or to sarcoma in the lymph-glands of the neck and loins, or in one of the long bones. The nature of the disease is usually obvious if it follows a primary affection; but it is otherwise when the growth is seated internally,—as, for instance, in the mediastinum. Sometimes the vertebræ become affected with malignant disease by direct extension, as from a cancer of the kidney. Sometimes the spinal lesion is itself a primary new growth. In these cases it is generally a sarcoma, and not infrequently it is found in the vertebræ of different regions, as well as in other bones, those of the limbs, the ribs, the pelvis, or the skull. Thus a careful search during life may lead to the discovery of a tumour in some distant part of the body, and so may clear up a case otherwise obscure.

Sometimes one may feel a projection of one or more spinous processes, or a mass of growth can be felt within the substance of the erector spinæ muscle. Sometimes a tumour connected with the front of the spinal column is discovered when deep pressure is made; or, if the cervical vertebræ are diseased, manipulation of the neck may reveal an enlargement on one side corresponding with the transverse processes. But in the great majority of cases one can feel nothing abnormal.

In two of the writer's cases the primary growth was in the œsophagus. It was in the vertebræ in five, in the bladder or prostate in four, in the breast, uterus, colon, or adrenal in the remainder. These fifteen patients were of all ages, from sixteen to sixty-eight. In one case in Philip Ward a correct diagnosis was possible, from the age of the patient making caries unlikely, while the severe pain and its increase on pressure of the spine downwards pointed to the vertebræ. It was confirmed before death by the detection of a tumour in the abdomen, which proved to be a cancerous gland. In another case the paralysis was very slight, and the pain and other symptoms subsided so much after rest in bed that the patient, a man of fifty, was allowed to get up. The same day he was attacked with renewed pain, and paraplegia became complete. Death speedily followed, and we found that while secondary cancer of the vertebræ had compressed the cord, the weight of his body in walking had crushed the bones together and destroyed a segment of the cord.

3. *Erosion of the vertebræ by aneurysm.*—Common as it is for an aneurysm of the aorta to eat away the bodies of the vertebræ with which it comes into contact, it very rarely penetrates the spinal canal so as to interfere with the cord. We have in Guy's museum specimens from two such cases, in each of which paraplegia developed itself at a period long after the discovery of a pulsating tumour in the back.

A third remarkable instance occurred in 1871. A man, aged thirty, was admitted into the hospital for paraplegia, which had begun three months previously. He had first complained of pain in the shoulders, then his left leg became weak and numb, and afterwards his right leg, and he experienced a pain as though the abdomen were constricted by a cord

He died without our suspecting the real nature of his disease. When the erector spinæ was cut into, a large mass of laminated clot was found in its substance on the left side. This belonged to an aneurysm of the descending aortic arch, which had destroyed several ribs, and laid open the spinal canal for a space two inches in length. Some of the clot adhered to the outer surface of the dura mater; the cord itself was flattened, white, and very soft.—C. H. F.

4. *Erosion of the vertebræ by a hydatid.*—This also is extremely rare. A striking instance is recorded by Cruveilhier. A large echinococcus lay behind the vertebræ, and filled the groove on each side of the spine beneath the lumbar muscles, so as to form two elongated pouches, which were connected by a narrow neck. A somewhat similar case occurred to Dr Moxon in 1871, a few months before the case of aneurysm just quoted. The parasite formed a large elastic swelling on the left side of the spine, and made its way into the canal through the second and third lumbar vertebræ. It compressed the cord, but did not penetrate the dura mater. The patient, a woman of fifty-eight, had been paraplegic for six weeks.

5. *Meningeal tumour.*—New growths occasionally form in the spinal membranes, and have been known to press on the cord so as to produce paraplegia. Sometimes a lipoma or an enchondroma is formed in the connective tissue outside the dura mater of the cord, more frequently within the sheath. According to Charcot, tumours are particularly apt to grow from the inner face of the dura mater—generally sarcoma or psammoma.

Sir William Gull described three cases of this kind in his paper on paraplegia in the 'Guy's Reports' for 1856: one (? a myxoma) grew from the inner surface of the dura mater at the first dorsal vertebra; the second, a sarcoma, had the same origin a little lower down; the third (probably a gumma) grew from the pia mater in the lower dorsal region.

Dr Fagge recorded a tumour of perfectly developed fibrous tissue which lay close to the foramen magnum and extended downwards for more than two inches, so as to press upon the back of the cord on the left side; also a soft, granular, reddish-grey growth, smooth and lobulated on the surface, which lay loosely attached between the arachnoid membrane and the pia mater, in the dorsal region. It measured an inch and a quarter in length, and consisted partly of fibrous, partly of spindle-cell tissue.

Such new growths of the spinal membranes, although rare, are much less so than those which begin in the cord itself.

6. *Meningeal gumma.*—This seems to be very rare, more so than meningeal tumour. Virchow has related a case in which the dura mater in the lower cervical region was increased to three times its normal thickness, and was bound down to the bodies of the fifth and sixth cervical vertebræ by a large quantity of firm connective tissue, which was believed to originate in a gumma. Wilks once found a hard, irregular, yellow mass three quarters of an inch long, and probably syphilitic, lying on the right side of the cord within its sheath, and adherent to the pia mater and to the posterior roots of the spinal nerves, which were compressed by it. In a case of Dr Moxon's ('Guy's Hosp. Rep.,' 1871) there were several brownish or blackish patches, from the size of a barleycorn to that of a pea, with soft yellowish centres, penetrating into the substance of the cord from the pia mater. Heubner, in 'Ziemssen's Cyclopædia,' described a gelatinous mass extending from the floor of the fourth ventricle into the bulb for a depth of one twelfth of an inch; while in the cervical region the dura mater and the other membranes behind the cord were pressed together into a callous

Meningeal *tumours* are described by Erb as slower in their effects; some cases end in eight or ten months, others in from two to five years; others last longer still, even to a period of fifteen years. In one of Dr Fagge's two cases the duration of the disease was sixteen months, and in the other two years, if we reckon from the time when pain in the back was first complained of; one patient died of pneumonia, the other of renal suppuration.

The only kind of paraplegia from compression in which recovery of power over the limbs appears to be possible, is that due to *caries* of the spine. Of such cases many do well; among six which came under Erb's observation in one year, only one ended fatally, the remaining five being cured or greatly relieved. Charcot refers to two patients, who had their lower limbs completely paralysed for eighteen months and two years respectively, and who nevertheless recovered perfectly. A favourable result after loss of power for seven months is mentioned below.

One might well have doubted whether in these cases of caries of the vertebræ, in which recovery takes place from compression-paraplegia, inflammation of the cord itself has ever occurred. But an observation of Michaud, which is cited by Charcot, shows that this really is so.

A woman who had regained the use of her legs for more than two years, after suffering from compression paraplegia with caries, died of hip-joint disease. The cord, at the level of the spinal affection, was found reduced to the thickness of a goose-quill, its sectional area being not more than one third of that of a healthy cord in the same region. It was of firm consistence and grey in colour; in other words, it seemed to be affected with sclerosis in an advanced stage. The microscope, however, showed that a considerable number of nerve-fibres possessing medullary sheaths were embedded in the thick dense fibrous material which gave it these appearances. Only one of the grey cornua remained, and this displayed but a small number of uninjured nerve-cells.

Treatment.—The most important means of treatment in cases of paraplegia from angular curvature is complete rest in the recumbent and, as much as possible, in the prone position.

When this is impracticable, or when it has been continued with good results, and the patient is first allowed to sit up, a valuable means of securing rest for the diseased part is Sayre's method of applying a plaster-of-Paris bandage, rolled round the body while the patient is suspended from the head, chin, and armpits by a suitable apparatus.

Cod-liver oil should be administered, and every effort made by fresh air and abundant food, particularly of a fatty nature, to combat the tuberculous process. The use of iodide of potassium in very large doses may sometimes be followed by such remarkable recovery as in the following case.

In 1890 the writer had under his care a young woman of twenty-three, with complete motor and sensory paraplegia from vertebral caries. She had been the subject of angular dorsal curvature since childhood, with tuberculous disease of one hip-joint; and for seven months had been completely paralysed up to the waist, with some vesical symptoms in addition. After the example of some American physicians, large doses of iodides of potassium and sodium were prescribed, increasing in amount and frequency until half a drachm was taken every two hours; much distress from iodism was produced, and the drug was stopped every ten or fourteen days for a short time; but the patient persevered with the unpleasant treatment, and was rewarded by finding first sensation and then motion return in one foot. Continuance of the iodide treatment ended in both feet and ankles recovering, and after about two months sensation was completely regained, and she was able to move her legs freely in bed.

She continued taking iodide of potassium in ten-grain doses every four hours while

Associated with the pain there is often extreme hyperæsthesia. The patient may be unable to bear the slightest touch without crying out, while every movement is excessively painful. On the other hand, there is sometimes impaired sensation or actual anæsthesia over more or less of the surface to which the affected nerves are supplied. The muscles which they supply may show tonic or clonic spasms, or become paralysed and atrophied, with loss of faradic contractility and other reactions of degeneration. An eruption of zona has now and then been observed in the course of some of the nerves involved.

These pains are symptoms of compression paraplegia only indirectly; they depend on the nerve-roots being pressed upon, and may occur whenever spinal nerve-roots are involved in disease, though the cord may remain intact. Thus lateral curvature of the spine, which probably never causes paralysis, is frequently accompanied by a fixed pain in one or more of the ribs or intercostal spaces, which seems to be due to pressure upon nerves as they are passing through the intervertebral foramina. In the following remarkable case of medullary sarcoma of the vertebræ there was at no time any well-marked paralysis.

The patient was an errand-boy, aged sixteen, who, five months before his death, began to experience pain first in the loins, then in the shoulders, and then in all his limbs. His sufferings became almost unbearable. The whole surface of the body was excessively tender, but especially the lower part of the back. The pain was constant, but it varied in position. There was numbness and a sensation of tingling in each hand; this was first noticed in the fingers supplied by the ulnar nerve on either side. He became wasted to the most extreme degree. The only way in which he got any ease was by lying flat on his back, with his legs drawn up. Every movement of his neck, and of any part below it, caused him severe pain. One day he became insensible and had a series of epileptiform seizures, in one of which he died. A mass of white medullary new growth (with microscopical characters such as are now known to belong to the sarcomata) occupied the lumbar glands, and spread from them to the intervertebral discs, eating also into the lumbar vertebræ themselves. It passed up in front of the spine into the neck, where it involved part of the brachial plexus on each side; but nowhere penetrated into the vertebral canal or implicated the membranes of the cord.—C. H. F.

There seems to be no doubt that pain is more marked as an early symptom in cases of malignant disease of the spine than in those of other forms of compression paraplegia. Cruveilhier long ago brought together under the name of *paraplégie douloureuse* certain cases attended with intense lancinating pains in the nerves of the sciatic and lumbar plexuses, and with paralysis of their muscular branches. It has been since ascertained that a large majority of the cases in question are instances of malignant disease of the spine, involving the roots of the nerves for the lower limbs in the cauda equina.

The reason for this distribution of symptoms seems to be that whereas the lumbar and lowest dorsal vertebræ frequently become cancerous, they are not often affected with caries. On the other hand, in fourteen consecutive cases of paraplegia, due to Pott's disease of the vertebræ, collected from our *post-mortem* records at Guy's Hospital, the caries did not once occupy this lower part of the spine. The few other cases in which it did occupy this position were without paralysis: thus in one instance the third and fourth lumbar vertebræ were found "extensively diseased," and in another the eleventh and twelfth dorsal vertebræ had entirely disappeared, so that on straightening the spine a large space was seen in front, in which the sheath of the cord was exposed; but in each case the disease had been entirely latent during life, for the patient had been kept in bed for a considerable time by phthisis or some other malady, and thus no doubt the cord escaped being compressed. In the report of the second case it is expressly noted that repeated questions failed to elicit any complaint of pain, whether in the back or elsewhere.

Pain in the back itself—rachialgia—although not infrequently present as an early symptom of compression paraplegia, is far less constant than one might have expected. It may assume different forms. Certain of the spinous processes may be tender on pressure, or over-sensitive to a slight blow or jar, or to a hot sponge, or the patient may be conscious of a feeling of stiffness in moving the back or the neck, according to the seat of the mischief.

Intrinsic or spinal symptoms.—After the "extrinsic" symptoms have lasted for weeks, months, or even for years—and perhaps been regarded as neuralgic or "rheumatic"—they are succeeded by others of which the spinal origin is obvious. These "intrinsic" symptoms are not mere results of mechanical pressure on the cord; for, though gradual external pressure may probably alter the shape of the cord, symptoms only begin when the pressure interferes with its circulation and nutrition.

The course of the paraplegia is such as might be inferred from the nature of the lesion causing it. Sometimes its development takes place very rapidly—within two or three days, or even in a few hours; much more often it occupies several weeks, or months. The patient finds his legs more and more heavy, especially in going up and down stairs; he becomes unable to stand; at last he cannot even move his toes while lying in bed. Subjective sensations of numbness or pins-and-needles often precede the motor symptoms; but, later on, loss of voluntary power over the muscles generally preponderates over loss of sensation. In some cases when sensation is decidedly affected the perception of temperature and of pain is abolished, while tactile sensation is good. The upper limit of the spinal lesion is commonly well marked, especially if there is much anæsthesia. It corresponds more or less exactly with the seat of the early "extrinsic" pains and tenderness, which indeed often persist after paralysis has set in. A point of great importance is that secondary changes in the cord seldom spread upwards beyond the point of compression: occasionally the local myelitis from compression leads to ascending sclerosis of the posterior median columns without special symptoms; but much more frequently to descending sclerosis of the lateral columns.

Reflex contractions in the lower limbs are usually more readily excited than under normal conditions, but this is only after an interval of reflex paralysis following immediately on a total transverse lesion.[*]

The state of the bladder is very variable; it often continues to act naturally for some time after the legs have begun to be paralysed; but as soon as the paraplegia is complete, retention of urine at once follows.

Some forms of *paræsthesia*, although not peculiar to compression paraplegia, are observed frequently in these cases. One is a retardation in the transmission of sensory impressions when there is not absolute anæsthesia; fifteen, twenty, or even thirty seconds may elapse between the application of a stimulus to the cutaneous surface and the perception of it by the patient. This we shall meet with again as a symptom of Tabes. Another is a peculiar dysæsthesia, a distressing "vibration," which is excited by slightly pinching a limb or touching it with anything cold, and lasts for several minutes. A third is an "associated sensation" which is referred to a spot upon the opposite limb corresponding exactly with that to which the stimulus is applied. These symptoms, like the numbness and tingling above mentioned, disappear when anæsthesia is complete.

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[*] See on this point Dr Bastian's paper ('Med.-Chir. Trans.,' lxxiii, p. 151), and remarks by Dr Jackson, Mr. Bowlby, and Dr Buzzard ('Med.-Chir. Proc.,' February 25th, 1890).

It is important to notice that paraplegia from whatever cause is not always symmetrical, one leg is often rather worse than the other.

Diagnosis.—The determination of compression as the cause of paraplegia may be either very easy or very difficult. The simplest cases of all are those in which there is obvious spinal deformity, for the short round bend or sharp angle produced by Pott's disease is unlike anything else. But often the natural configuration is preserved.

Leyden says that if caries occurs in either the lumbar or the cervical region there is rarely any projection of the spinous processes, the reason being that the natural curve of those parts of the column is in the opposite direction; but if in these cases the patient stoops or bends the head forward, some one of the cervical or lumbar spines may appear unduly prominent. It is important to observe whether the vertebral column retains its flexibility, and whether in the neck there is any lateral thickening to be felt.

In malignant disease, if there is anything to be detected on manipulation, it is the presence of a new growth; but the pain, the seat of the disease, and the age of the patient may help diagnosis (pp. 617-18).

Cruveilhier, in his hydatid case (p. 615), discovered between the twelfth dorsal and first lumbar vertebræ a spot the size of a sixpence, which felt like a depression surrounded by a bony ring. This he took for the remains of a spina bifida; but, as he said with regret, if he had rightly interpreted what he observed, and had ventured upon making a puncture, the patient might perhaps have been cured.

It is chiefly at an early period that a case of compression paraplegia might be mistaken for functional *rachialgia.* Considering the frequency with which this spinal neurosis is associated with hysteria, it is fortunate that rachialgia seldom accompanies hysterical paraplegia. Careful examination of the patient would probably show a want of correspondence between the apparent upper limit of the lesion in the cord, and the seat of tenderness and pain in the spinal column.

It is often difficult to distinguish cases of caries of the vertebræ or of meningeal tumour from those of primary *transverse myelitis*, or of any other affection of the interior of the cord limited to a particular segment. Myelitis is often accompanied by painful sensations referred to various parts of the body, and by the peculiar "girdle-feeling"—symptoms which are strictly comparable with the "extrinsic" symptoms of compression paraplegia, and which do not always differ from them even in degree. We must admit that in some cases diagnosis between the two forms of disease (in fact, between primary and secondary myelitis) is impossible.

Prognosis.—Most forms of paraplegia by compression end in the death of the patients, but their duration is very variable.

According to Leyden, *cancer* of the vertebræ commonly runs a course of some months, sometimes of more than a year. Among fifteen cases in Guy's Hospital the writer found the duration, from the earliest symptoms till death, was three months in six, under a year in eight, and fifteen months in one patient.*

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* When the upper cervical vertebræ are affected, the fatal issue is sometimes preceded by delirium and stupor, for which no explanation can be found in the autopsy.—C. H. F.

Meningeal *tumours* are described by Erb as slower in their effects; some cases end in eight or ten months, others in from two to five years; others last longer still, even to a period of fifteen years. In one of Dr Fagge's two cases the duration of the disease was sixteen months, and in the other two years, if we reckon from the time when pain in the back was first complained of; one patient died of pneumonia, the other of renal suppuration.

The only kind of paraplegia from compression in which recovery of power over the limbs appears to be possible, is that due to *caries* of the spine. Of such cases many do well; among six which came under Erb's observation in one year, only one ended fatally, the remaining five being cured or greatly relieved. Charcot refers to two patients, who had their lower limbs completely paralysed for eighteen months and two years respectively, and who nevertheless recovered perfectly. A favourable result after loss of power for seven months is mentioned below.

One might well have doubted whether in these cases of caries of the vertebræ, in which recovery takes place from compression-paraplegia, inflammation of the cord itself has ever occurred. But an observation of Michaud, which is cited by Charcot, shows that this really is so.

A woman who had regained the use of her legs for more than two years, after suffering from compression paraplegia with caries, died of hip-joint disease. The cord, at the level of the spinal affection, was found reduced to the thickness of a goose-quill, its sectional area being not more than one third of that of a healthy cord in the same region. It was of firm consistence and grey in colour; in other words, it seemed to be affected with sclerosis in an advanced stage. The microscope, however, showed that a considerable number of nerve-fibres possessing medullary sheaths were embedded in the thick dense fibrous material which gave it these appearances. Only one of the grey cornua remained, and this displayed but a small number of uninjured nerve-cells.

Treatment.—The most important means of treatment in cases of paraplegia from angular curvature is complete rest in the recumbent and, as much as possible, in the prone position.

When this is impracticable, or when it has been continued with good results, and the patient is first allowed to sit up, a valuable means of securing rest for the diseased part is Sayre's method of applying a plaster-of-Paris bandage, rolled round the body while the patient is suspended from the head, chin, and armpits by a suitable apparatus.

Cod-liver oil should be administered, and every effort made by fresh air and abundant food, particularly of a fatty nature, to combat the tuberculous process. The use of iodide of potassium in very large doses may sometimes be followed by such remarkable recovery as in the following case.

In 1890 the writer had under his care a young woman of twenty-three, with complete motor and sensory paraplegia from vertebral caries. She had been the subject of angular dorsal curvature since childhood, with tuberculous disease of one hip-joint; and for seven months had been completely paralysed up to the waist, with some vesical symptoms in addition. After the example of some American physicians, large doses of iodides of potassium and sodium were prescribed, increasing in amount and frequency until half a drachm was taken every two hours; much distress from iodism was produced, and the drug was stopped every ten or fourteen days for a short time; but the patient persevered with the unpleasant treatment, and was rewarded by finding first sensation and then motion return in one foot. Continuance of the iodide treatment ended in both feet and ankles recovering, and after about two months sensation was completely regained, and she was able to move her legs freely in bed.

She continued taking iodide of potassium in ten-grain doses every four hours while

awake, and after three months walked out of the ward with complete restoration of sensation and movement in her limbs.

It is probable that mercury and iodide of potassium are both capable of curing paraplegia which is due to syphilitic gummata pressing on, or growing into, the cord; and this treatment should never be omitted in any case of paraplegia from pachymeningitis, which may possibly be of venereal origin.

Charcot was convinced of the value of the actual cautery. This ancient and formerly much-used remedy has been applied in two ways,—by searing the skin on each side of the spine, or by making incisions and cauterising the wounds. After witnessing a trial of each the present writer would dissuade from both.

Of late years antiseptic surgery has successfully dealt with paraplegia from compression by the products of vertebral caries. Many cases in the hands of MacEwen have been now recorded in which the laminæ have been exposed, trephined, or resected, and the caseous material cleared out; and this severe operation has again and again proved successful.

Mr Horsley published a remarkable case in which he resected the spine in a patient of Sir Wm. Gowers, who had diagnosed the existence of an intra-vertebral tumour pressing on the cord. The tumour was found and removed, and the patient was not only relieved from excruciating suffering, but was restored to power of locomotion ('Med.-Chir. Trans.,' 1888). So brilliant a result of the combination of the skill of the physician in detecting and the skill of the surgeon in removing an organic lesion can scarcely be more than a rare event; but even one such case encourages efforts to localise and define a lesion, and justifies the boldest operations when so directed.

MYELITIS.—Among the intrinsic lesions of the spinal cord which cause paraplegia, the most important and by far the most frequent is one which is now believed by most pathologists to be inflammatory, and is therefore termed *myelitis*.

*Acute diffuse myelitis.**—It has long been known that in many cases of paraplegia the cord is found after death to be more or less extensively softened; and from the time of Ollivier the opinion has been maintained that the morbid process in question is of an inflammatory nature. There are, indeed, obvious objections to such a view. It involves the admission that a remarkable contrast exists between softening of the cord and a similar change in the brain. The latter (as we shall hereafter find) is most frequently the result of a deficient supply of arterial blood to the affected part of the cerebral substance; and when it is inflammatory it is almost always traceable to some definite local irritation. The former, on the other hand, can very seldom be shown to be secondary to interruption of the blood-supply, and its chief causes are supposed to be functional exhaustion or exposure to cold. A more serious difficulty is that the histological appearances correspond only partially with those which are seen in inflamed tissues generally, and that even the naked-eye changes are not altogether such as are met with in the spinal cords of animals when inflammation has been artificially set up. Leyden, for example, induced myelitis in dogs by injecting solution of arsenic, and obtained greenish patches of purulent infiltration, surrounded by vascular zones; but, in human pathology, the

* *Synonyms.*—Acute softening of the cord—Red and yellow softening—Myelomalacia.

presence of suppuration in the cord is quite exceptional. On these and other grounds Dr Bastian regards most cases called acute myelitis not as strictly inflammatory, but as a process of softening secondary to thrombosis or other vascular changes.

The late Dr Moxon, in his Croonian Lectures, drew attention to the scanty vascular supply of the lower part of the cord owing to the fewness, small calibre, and prolonged course of its arteries, and to its not having, like the brain, an internal set of vessels in addition to those of its pia mater. He justly argued that this anatomical condition may explain some of the peculiarities of anæmia, thrombosis, and inflammation as they affect the spinal cord.

Anatomy.—The colour of the spinal cord when softened is very variable. Sometimes its tissue is pink or bright *red* from vascular injection; sometimes it is mottled with small ecchymoses; or it may be so saturated with extravasated blood (hæmato-myelitis) that it looks like pure hæmorrhage ("spinal apoplexy" if there be such a thing). Probably all these appearances are confined to an early stage of the inflammatory process; for afterwards the blood undergoes changes which give to the affected parts a tawny *yellow* tint, due to the presence of hæmatoidin crystals and of fatty degeneration in the form of "compound granule corpuscles." If no hæmorrhage has occurred, the softened spot as a rule is *white;*[*] or in rare cases of a greenish tint.

We must not forget that one part of the spinal cord being softer than the rest, or looking diffluent, may result from bruising with the bone forceps during the autopsy, even though made by a practised assistant. The microscope here affords valuable assistance. As Dr Frederick Taylor remarks, the more softened the cord, the more difficult it is to get satisfactory results from the usual hardening processes, and it is therefore generally desirable to examine some portion of the cord under the microscope in the fresh condition ('Allbutt's System,' vol. vii, pp. 8, 9). In most cases one has only to spread out a little of the softened tissue between a slide and a cover-glass in order to find numbers of opaque mulberry-like aggregations of fatty granules, the well-known compound granule masses or "corpuscles of Gluge." These structures, when present in large numbers in one part and absent in others, are undoubted evidence of local lesion. They are fully developed at an early period. Thus there were large numbers of them in the spinal cord of a patient who was under the care of Dr Frederick Taylor in Guy's Hospital in 1873, and who died within eleven days from the commencement of his symptoms, and not more than fourteen days after the occurrence of a slight injury that perhaps set up the disease. In such cases they are not generally accompanied by an obvious infiltration of leucocytes, or by the presence of any diffused granular matter, but lie embedded among nervous elements.

Granule masses are not infrequently discovered when there is no obvious softening, and even when there is no marked change of colour, or, at most, only a pinkish-grey tint, with some blurring of the pattern on a cut surface. In a fresh frozen section the blood-vessels are seen to be distended, their lymphatic sheaths full of leucocytes and larger (? endothelial) cells, and en blood-discs are seen extravasated. In many cases after the cord has n soaked in a dilute solution of chromic acid for two or three weeks one n see at a glance what parts are diseased, since they remain colourless tead of turning green. A microscopical examination of thin sections ws that the nerve-fibres have their axis-cylinders greatly swollen and icose, that the ganglion-cells are much enlarged and granular, or perhaps uolated, and the axon thickened. Subsequently the ganglion-cells dis-ear, the fibres atrophy, and little remains but Deiters' corpuscles and ge's granule masses.

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[*] Is white softening ever observed when death has occurred at a very early stage? I have not found any instance among cases which have proved rapidly fatal at Guy's Hospital, although Jaccoud speaks of a *myélite suppurée* ('Les Paraplégies,' p. 545).—*C. H. F.*

The network of the neuroglia is thickened, while its cells are increased size and augmented in numbers, and sometimes contain more than one leus. More or less infiltration of leucocytes is seen in the interstices ween the nerve-fibres; but actual suppuration is very rare, and probably always secondary to septic meningitis. Such appearances seem to be clusive as to the inflammatory nature of the process, and so is the sence of lymph in the tissue of the pia mater of the cord or in the sub- chnoid space, where it is sometimes in sufficient quantity to be visible the naked eye.

Acute myelitis is never limited to the anterior or posterior cornua of e cord, nor to any one of its columns. It is a diffuse or “indiscriminate” ocess, not what the German pathologists call a “system-lesion,” or, we may say, a “columnar” disease. It may begin from the surface, sequence to meningitis (“annular” myelitis), or it may occupy the grey atter around the neural canal (“central” or “peri-ependymal” myelitis); may affect the entire breadth of the cord, including both cornua and lumns (“transverse” or “segmental” myelitis); it may be “dissemi- ated” in minute patches; or, lastly, it may rapidly spread over large por- ons of the cord as a progressive ascending myelitis.

Course.—The clinical course of myelitis is not always that of an acute flammation, although it always begins as one. Sometimes, indeed, it ts in with fever and ends fatally in a few days, but in many cases it is attended with febrile disturbance, and goes on for months before it rminates, either in the death of the patient or in his more or less com- ete recovery. What justifies the name of acute softening or acute elitis is the fact that its onset is more or less sudden and its develop- nt rapid, so that the paralysis is complete within a week or two.

Ætiology.—According to Erb, inflammation of the cord is more frequent tween the ages of ten and thirty years than in older persons. But enty-five consecutive cases of fatal primary diffused myelitis at Guy's ospital were pretty uniformly distributed over the several decennial riods from ten to fifty, and three of them occurred in patients between ty and sixty years old. It is certainly a rare disease in children and in d people above sixty—as would be supposed from the nature of its ante- edents. Most of the above cases occurred in men, the proportion being 9 to 6. This may also depend on the fact that men are more exposed han women to its probable exciting causes.

Sometimes myelitis follows as the result of exposure to cold, particularly o wet. Many instances of this have been published,* and our records at Guy's Hospital supply many others.

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* See, for example, the first three of Dr Bristowe's cases ('Brit. Med. Journ.,' 1888, ii, 1369), and one described by Dr Duffin in the 'Clinical Journal,' where the cause peared to be a draught of cold air through the open porthole of a ship.

Thus, in 1876, a custom-house officer was admitted into the hospital with nearly complete paraplegia, who had been well until eight days previously, when he was obliged to remain for twenty-four hours in a barge, where everything was soaking wet. On the very next day he was attacked with pains in the joints, which he believed to be rheumatic; his urine began to run away; and two days later his feet and hands and back felt numb.

Another patient, who was brought in on January 29th, 1875, had on the 18th of that month got wet and caught cold. Three days afterwards he passed his water into his bed without knowing it, and his legs became numb and powerless.

Cases of acute myelitis have followed sleeping on the damp ground or in snow, and prolonged immersion in water, as when a person narrowly escapes drowning. Ditchers and other men who work up to their knees in water, and women who have tramped through rain till their skirts are soaked through, seem particularly liable to this disease.

Great bodily fatigue also seems to play an important part in the ætiology of the disease. When it occurs in soldiers on active service, as is said to be frequently the case, it has been ascribed to exhaustion of the spinal cord from forced marches, or to exposure to cold and wet, or to both causes combined. The following case in point was recorded by Sir William Gull ('Guy's Hosp. Rep.,' 1856, Case 10).

A young man, after walking twenty-eight miles to seek for work, passed the night of July 8th, 1855, in a brick-field. On the 15th he walked thirty-two miles; the day was wet, and he allowed his clothes to dry upon him. Next day he felt as well as usual, but in the afternoon, while he was sauntering in a garden, his legs suddenly gave way under him, and he fell down. He was admitted, six days later, completely paraplegic. He was healthy and florid-looking; but it should be stated that he had, about a fortnight previously, been unable to pass his water for a period of twelve hours. He died in less than a month.

A great muscular effort, such as causes a pain in the back, has been followed, after an interval of a day or two, by paraplegia. An instance of this has already been cited in the case of a dock labourer.

Another example of it occurred in Guy's Hospital in 1863: a railway porter, aged thirty-two, was pushing a carriage with his back, when he suddenly gave a jump to escape falling into a pit, and so ricked his spine; for a moment he felt powerless, but was able to resume his work, and worked as usual on the next day. On the day after, however, he was walking in the street, when he suddenly became paralysed and fell down.

Both these cases ended fatally in about six weeks. At the autopsy no injury to the bones or the ligaments could be discovered; but in the dock labourer there was softening of the cord in the dorsal region, and the affected part had a greenish colour, from the presence of extravasated blood. The microscopical examination revealed no further proof of inflammation; but if modern methods of investigation could have been used, we might have been more successful.

Acute paraplegia occasionally appears as a sequel or complication of a specific fever. Virchow has recorded ('Ges. Abhandl.,' p. 683) an instance in which chronic myelo-meningitis came on a few months after recovery from enteric fever. Westphal examined the bodies of two patients, who became paraplegic during the eruptive stage of smallpox, and in both cases he found disseminated myelitis. Myelitis does not appear to follow typhus or relapsing fever, and is unknown as a sequel of scarlatina, measles, pneumonia, or acute rheumatism. In fact, syphilis is the only specific fever of which acute paraplegia is more than an exceptional sequel.

That acute paraplegia often follows *syphilis* there is no doubt; the only question is whether it is always due to myelitis.

In one remarkable case of acute myelitis, which ended very quickly in death, Baumgarten found the characteristic bacilli of anthrax in the *blood* as well as in the spinal cord ('Arch. f. Heilk.,' 1876). The paraplegic n of rabies normal in rabbits and occasionally seen in man, as in Dr towe's case (*supra*, p. 408), is probably due to acute myelitis.

Either pregnancy or the puerperal state may be attended with paralysis he lower limbs. A woman was under the writer's care in December, 9, suffering from complete paraplegia, probably due to myelitis, which wed severe puerperal convulsions with albuminuria; she slowly vered.

There is some reason to believe that sexual excesses have a share in ætiology of diffused myelitis, although it is very difficult to say how e a one.*

None of these "causes" can be regarded as constant or pathogenic, and r influence is at most predisposing. Nor has the attempt to discover a ific infective cause of acute myelitis yet been successful. Its following e specific fevers, and particularly syphilis, septic endocarditis, and other ns of pyæmia, suggests this hypothesis; but only occasionally have ptococci or diplococci been found after death (Buzzard and R. Russell, in. Trans.,' 1898, p. 185).

Clinical history.—The symptoms of *primary*, *acute*, *diffused* myelitis first those of paraplegia as above described, including retention of ne and other pelvic symptoms.

Beside, however, causing akinesia, impairment of sensation, dysæs-sia, disorder of the reflex functions of the cord, and the like, myelitis often attended with another set of symptoms, namely, painful sensations erred to various parts of the body. In some instances the seat of pain in the back; it there varies greatly in intensity; it may either be nfined to one or two spinous processes, or diffused along the whole length the cord; there may be extreme tenderness on pressure, or this may altogether absent. The symptom of pain is usually referred to the nal meningitis which so often accompanies acute myelitis, but the lowing cases prove that this is not always the case. In other instances pain is referred to the front of the chest, or to the epigastrium, or to or more of the limbs.

ne patient, a girl of nineteen, was attending Guy's Hospital for pain in the chest. she one day fell down in the waiting-room, struck with paraplegia. Another, a man forty-nine, had complained of a fixed pain in the left hip for eight weeks before any ite sign of spinal mischief showed itself. A third, a man aged fifty-one, suffered at from severe burning pains in the soles of the feet, which continued night and day for siderable time. These cases all terminated fatally, and in each of them the bones and ents and meninges were found to be healthy, and the disease to be limited to the sub-e of the cord itself.—C. H. F.

Sometimes pain is accompanied by a sense of constriction, as if a cord tightly bound round the waist, or as if the chest were fixed in a vice. noticed that this symptom, to which German writers give the special e of *Gürtelgefühl* (girdle-feel), is particularly marked in cases of ex-sic paraplegia due to compression of the cord. It may also occur in nary myelitis, but the majority of cases are unattended with either pain girdle-feeling.

The *course* of myelitis differs widely, not only according to the nature seat of the morbid change in the cord, but also in individual cases which appear to resemble one another closely. The disease sometimes sets in with malaise and fever, and even with rigors. A case in point occurred at Guy's Hospital in 1872.

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* Men who have been indulging their passions too freely, especially if they are weakly ery young, often experience pains in the back and limbs, which seem to be due to ation of the lower spinal centres, and it is possible that these symptoms may in some s denote the first step of *myelitis*.

A man aged fifty-one, who was much exposed to weather, one day began to shiver, and was attacked with severe pains running down the back of the thighs and calves, and the insides of the arms. He remained in bed for four days, after which the pains left him, and he went to work again. Next day he lost all power in his limbs, and he died of dyspnœa about eight days afterwards.

Paralysis often shows itself suddenly, and this, as Erb remarks, is almost the only reason for calling such cases "acute myelitis," since they are often unattended with febrile symptoms, and their duration may be prolonged over a period of many months. In most cases of acute myelitis, however, death takes place in a few days or two or three weeks. It is comparatively rare for the disease to be prolonged in a chronic form.

Prognosis.—Many patients suffering from acute paraplegia get perfectly well, but whether there was inflammation of the cord is in most cases uncertain.*

An observation made by Michaud, on a woman who died from caries of the hip and vertebræ, shows that, in spite of the occurrence of well-marked myelitis, recovery from paraplegia due to external compression may take place, though the affected part of the cord retains signs of a morbid change. This case may perhaps be taken as proving the possibility of complete subsidence, with return of normal function, of those local and slight forms of myelitis which we may suppose to be present in partial and transitory forms of spinal paralysis. This conclusion is further supported by the fact that in such cases recovery is often incomplete: since it is more reasonable to suppose that an organic affection of the cord may subside than that a merely functional disorder should be permanent.

The prognosis is generally unfavourable when there is complete paraplegia with pelvic symptoms; but even to this rule there are some remarkable exceptions. Two have been recorded by Wilks in his 'Lectures on Diseases of the Nervous System.'

One is the case of a gouty man, aged fifty-two, who had been losing power over his lower limbs and his bladder for a fortnight, so that on admission into Guy's Hospital he could not move his legs, and had partial loss of feeling up to the umbilicus. Reflex movements persisted. The urine, which was ammoniacal, had to be drawn off twice daily. Afterwards he had a feeling of tightness round the lower part of the chest and the abdomen, and he experienced numbness passing down the arms into the fingers. He gradually got worse; and about a fortnight after his admission he became feverish, with a quick pulse, a red tongue, rigors, and hiccough; a bedsore was forming, and the urine ran away from him; moreover his mind became clouded. Dr Wilks thought that he was suffering from suppurative nephritis, and had not many hours to live. For some days he remained in a precarious state, and then his symptoms abated. He began to regain some degree of power in his legs, and from this time he made a rapid recovery; he sat up in bed, he ceased to require the catheter, he got into a chair, he asked for crutches, he walked about the ward, and finally he left the hospital: this was exactly two months after his admission, and six weeks from the time when his paralysis became complete. This patient showed himself several weeks later, still quite well.

The other case is that of a woman of middle age, who came in suffering from almost complete paraplegia, which had begun a few days before. It was accompanied by pain and swelling of the joints, with some febrile disturbance. She complained of great pain in the limbs, twitching of the muscles, and of a sense of constriction round the waist. The urine was ammoniacal, and the sphincter ani was paralysed; a bedsore formed, which ended in a deep slough. Yet after three weeks she began to recover, and at the end of five months she was discharged perfectly well.

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* Eichhorst and Naunyn found in experiments upon young dogs, that after they had cut through the spinal cord in the lower dorsal region the lesion was slowly and partially repaired; a few nerve-fibres with double contour were developed in the new material which filled up the gap, and motor power and sensation were to some extent regained. These results, however, have not been confirmed by the more recent investigations of Goltz and Freusberg, and it is certain that in man the cord has no such powers of repair.

The following case of apparently acute anterior myelitis also ended in recovery.

The writer saw in January, 1888, with Dr Cock, of Peckham, a boy thirteen years old, who a week before had been unable to pass his water. It was drawn off, and there had been more or less incontinence since. Three or four days before, his legs began to feel weak; and when seen he was almost completely paralysed below the waist, with sensation unimpaired, the knee-jerk abolished, and the plantar and cremasteric reflex almost absent. He had scarcely any power to expel either urine or fæces. There were no convulsions, and neither stiffness nor pain of any kind. Sensation was apparently normal, so that it was difficult to fix the upper limit of paralysis. The next day priapism came on and bullæ appeared on the paralysed legs. The temperature remained normal or subnormal. There was no tenderness along the spine either to pressure or to contact with a warm sponge. The paralysed muscles reacted to faradism as well as to interrupted galvanism. In a week from the onset of the attack there was some return of power in the legs. The pelvic symptoms gradually disappeared, and he at last recovered perfectly after about two months' illness.

Diagnosis.—The decision that myelitis is the cause of partial or complete paraplegia is generally effected by a process of exclusion.

For practical purposes we must first exclude mere shamming, functional and hysterical paraplegia, and alcoholic paraplegia due to symmetrical peripheral neuritis (p. 595). The presence of "pelvic symptoms" (p. 612) will generally suffice for this. Next we must exclude "extrinsic" cases due to compression of the cord, and chronic and spastic cases of slow development.

Lastly, we have to decide which intrinsic disease of the cord is most likely to be the cause of the paralysis. Myelitis is at once the most frequent of these affections, and the most varied in its symptoms and course.

The occurrence of paraplegia in a person known to be suffering from phthisis or any other tuberculous disease would lead one to suspect *caries of the vertebræ* producing compression of the cord without deformity. It is impossible to distinguish the paralysis caused by a *tumour* within the cord from more common cases due to circumscribed transverse myelitis, but the latter is the more likely. Severe and long-continued pain would probably lead one to suppose that the lesion was in the *bones* or in the *membranes*; but, as we have seen, acute myelitis sometimes causes pain. In two of the cases of paraplegia on which this chapter is founded *hæmorrhage* was, in fact, correctly diagnosed in spite of its extreme rarity, chiefly from the suddenness of onset of the symptoms. As regards *syphilis*, its recognition as the cause of paraplegia must be based upon the clinical history and upon the presence of other syphilitic lesions rather than upon any peculiarities in the spinal symptoms themselves; and no doubt there are many instances of syphilitic paraplegia in which one can only guess their real nature. When there is evidence of syphilis mercurial treatment is sometimes followed by recovery.

There is one positive indication of myelitis that must always be carefully sought, namely, gradual creeping up of the paralysis and anæsthesia from the thighs to the groins, and from thence to the abdomen; it warrants a confident diagnosis of diffuse ascending myelitis. Its early stage is probably indistinguishable from Landry's paralysis (*infra*, p. 637);

but after a time the absence of pelvic symptoms, and the other negative characters, would differentiate the latter.

The diagnosis between myelitis and other lesions which may cause acute paraplegia will be further referred to under their several heads.

Treatment.—The question remains, whether we can by medical treatment influence the course of myelitis so as to increase the proportion of cases in which recovery occurs; and here we have little positive knowledge to guide us. Of late years it has not been the practice in England to adopt active measures for the acute form of myelitis. The modern German plan seems to be to use leeches or cupping-glasses freely, to apply bags of ice along the spine, to rub in blue ointment, or to give calomel internally, —in fact, to carry out the "antiphlogistic" treatment very much as English physicians used to employ it fifty years ago. Erb recommends blistering the back, and even the actual cautery, in spite of the risk of setting up bedsores, which he fully recognises.

It is of great importance for the patient from the first to be placed upon a water-bed. He should not be allowed to lie constantly on his back, but should be shifted from time to time. It is sometimes dangerous to allow him to sit up. Some years ago a man, who had recently been admitted into Guy's Hospital with symptoms of acute myelitis limited to the lower part of the cord, was taken out to have his bed made; when put back he seemed much exhausted, and soon afterwards he died.

The utmost care must be taken to prevent the formation of *bedsores*. The sacral region must be kept dry and clean. The back, hips, and buttocks should be sponged with brandy or with lead lotion, so as to harden the skin. Or if a spot is already reddened, a large piece of felt plaster may be applied, having a hole in the centre. When an ulcer has formed, Hammond, of New York, speaks in the highest terms of Golding Bird's plan of placing a thin plate of silver over it, of exactly the same size as the sore; a wire six or eight inches long is carried from this plate to another made of zinc, which is laid on some part of the skin above, but separated from it by a piece of wash-leather soaked in vinegar ('Guy's Hosp. Rep.' for 1876, 3rd series, vol. xxi, p. 341). Galvanic action is set up; and Hammond states that he has "frequently seen bedsores three or four inches in diameter, and half an inch deep, heal entirely in forty-eight hours." Sir Spencer Wells is said to have obtained no less striking results.

Retention of urine must be relieved. A soft catheter should be used as soon as interference is necessary, but not before, and care must be taken to depress the free end of the instrument between the thighs, so as to draw off the whole of the urine; for if any should be left it will quickly decompose. The reason of this untoward event, however, is, there is only too good reason to believe, the conveyance of bacteria from an imperfectly cleansed catheter to the urine in the bladder. Unless the instrument is as scrupulously clean as a surgeon's fingers it is one of the most dangerous to life.

When a patient has unfortunately, by this or other means, already contracted cystitis with ammoniacal urine, the decomposition may be checked and the cystitis controlled by the administration of salicylates, or of some other antiseptic medicine, by the mouth. This practice seems to have been first proposed by Fürbringer. We have seen excellent results since Dr Pavy introduced it at Guy's Hospital. From half a drachm to a drachm must be taken in the course of the twenty-four hours; it often restores the urine from an offensive alkaline condition to one of normal acidity and odour. Or benzoate of soda or ammonia may be used, in ten- or fifteen-grain doses three times a day. When much pus is discharged the bladder should be regularly washed out with a weak antiseptic solution. One of the best is Sir Henry Thompson's borax lotion.

Next to the applications of cold, of heat, or of counter-irritants to the spine, various drugs are generally regarded as of value in acute paraplegia. One of these is *ergot;* Hammond relates a case of what he calls "congestion of the cord," in which it seemed to be of service. The patient had become affected with partial paraplegia, and afterwards with paralysis of the bladder, "from exposure to cold and damp." He had a constant dull pain in the loins, and occasional starting of the legs while in bed. Under treatment by the extract of ergot he recovered in a month. He had a relapse a few weeks later, but recovered in ten days under the same treatment. The writer at one time had a few cases of apparent benefit from the administration of ergot, but more frequently has found it to be quite useless. The tincture of *belladonna* in fifteen-minim doses has often been given in cases of paraplegia; and many writers recommend full doses of iodide of potassium, not only when there is probability of syphilis, but in other forms of paraplegia. *Strychnia* is of doubtful value in chronic myelitis, and is certainly harmful in the acute stage.

Galvanism should not be employed early in cases of myelitis, but it is probably useful and certainly harmless after the paraplegia has lasted for several weeks and acute symptoms have subsided. The electrodes should be large sponges placed at a considerable distance apart, one upon the neck, the other over the lumbar vertebræ; they should be moved slowly up and down. When there is reason to believe that the disease is limited to a segment of the cord, one pole may be placed on the affected spot, the other over the front of the chest or of the abdomen. Faradisation of the paralysed muscles may be useful if they are flabby or wasted.

On the Continent certain spas are reputed to be efficacious in the treatment of chronic paraplegia, with what justice it is hard to say. Erb says that hot baths are apt to be injurious, except at advanced periods of such cases. Among the places most frequently visited by patients suffering from paralysis are Schlangenbad, Ragatz, Pfeffers, Gastein, Wiesbaden, Teplitz.

Hæmorrhage into the cord ("*Hæmatomyelia*").—As a primary cause of paraplegia this is so rare, that Hayem* endeavoured to show that in all the supposed cases which have been recorded since those of Ollivier and Cruveilhier there was antecedent softening, the result of myelitis. This view would dissociate it altogether from the common cerebral hæmorrhage of old age; but there certainly seem to be a few instances in which extravasations of blood into the spinal cord are strictly comparable with those into the brain. The following case is Jaccoud's.

A woman, aged sixty-two, was brought into the hospital with complete paraplegia, which had suddenly appeared four days before. The diagnosis was given that there was hæmorrhage into the lumbar enlargement of the cord. Six days afterwards she was found dead in her bed. The autopsy showed that the grey matter of the cord, up to the highest roots of the lumbar plexus, was occupied by a clot which was beginning to undergo disintegration. The cause of death was an immense effusion of blood into the left lateral ventricle, the crus cerebri, the pons, and the bulb.

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* His monograph, 'Des hémorragies intra-rachidiennes,' 1872, contains a collection of thirty-two cases with copious references. Goltdammer finds about thirty cases recorded of non-traumatic hæmatomyelia, most of them hæmorrhagic myelitis ('Virchow's Archiv,' lxvi).

Hæmorrhage seems to be less rare in the central grey substance than in the columns of the cord. It is met with more often in men than in women, and between the ages of fifteen and thirty-five than at a later age. A striking instance is recorded by Goltdammer ('Virchow's Archiv,' 1876).

A girl, between fifteen and sixteen years of age, was one day about noon sitting quietly on a chair, when she suddenly felt a severe stabbing pain in the back between the shoulder-blades, which compelled her to cry out. The pain quickly passed into the right arm, and then into the left arm and the chest; at the pit of the stomach it extended round the body like a girdle. She fell from her seat, and at once noticed that she could not move her right leg; half an hour later her left leg also was paralysed; within two hours she was taken into the hospital, and was found to be perfectly paraplegic, with anæsthesia up to the nipples, and loss of power over the bladder. Reflex movements could be excited in the lower limbs. The case was diagnosed as one of hæmorrhage into the cord below the cervical enlargement. She lived almost exactly a year. At the autopsy there was found a firm cicatricial mass in the grey and white substance of the upper part of the dorsal region of the cord; the nervous structure was completely destroyed; hæmatoidin crystals and granular pigment were so abundant as to give a reddish-brown colour.

A few years ago a healthy labourer was admitted into Guy's Hospital under the writer's care, who after a sudden and severe effort felt his legs give way under him, and became completely paraplegic within an hour or two. There was no pain, no sign of local injury, and he slowly and steadily recovered. The suddenness of the attack, the complete paralysis, and the complete recovery seemed to point to hæmorrhage in the cord or its meninges.

Hæmorrhage into the cord, though often fatal, may be completely repaired by absorption of the clot. A remarkable case in a young man, which after severe symptoms terminated favourably, is recorded by Ross (vol. ii, p. 154).

It is doubtful whether any special treatment is of service.

Tumour within the cord.—With glioma and sarcoma we may include under this head syphilitic gumma and tubercle of the cord. Even then the group is a very small one.

A *gumma* very rarely begins in the cord itself; it is far more frequent (or rather, less rare) in the meninges.

Tubercle in the cord forms a rounded or elongated mass, which seldom reaches any considerable size. Two cases were observed in Guy's Hospital in the year 1870; in one the tubercle was no larger than a pea, in the other as large as a small cherry.[*] The main substance is firm and caseous; its centre may be softened into a creamy liquid; its periphery is sometimes grey; it is surrounded by a soft, vascular, pinkish zone, so that it readily slips from its bed when it has been cut across.[†] In some cases there is extensive pulmonary phthisis; in others the peritoneum or the mesenteric glands are tuberculous.

If not tubercle, a growth in the cord is probably a *glioma*, for an intrachordal sarcoma is extremely rare; one that was found in the body of a woman aged twenty-six, at Guy's Hospital in 1860, was an oval soft mass made up of spindle-cell tissue. A glioma may undergo softening (myxo-glioma). This is no doubt the origin of some of the remarkable cysts which are occasionally discovered as an elongated cavity in the cord (*syringomyelia*).

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[*] It is worthy of note that in one of them no sign of the lesion was visible on the surface of the pia mater. I made a series of sections at the usual distances from one another, and missed it altogether. It was afterwards found by Dr Reginald Stocker when cutting across the cord in fresh places.—C. H. F.

[†] Leyden says that tubercles more often occur in the upper than in the lower part of the cord. But of three cases of which I have notes there are two in which the lumbar region was the seat of the disease, while in the third it was in the lower dorsal region. Two of the patients were women, aged twenty-eight and fifty-eight; one was a man aged thirty-six. Dr Frederick Taylor has told me of a case that he saw in a child.—C. H. F.

The symptoms produced by tumours of the cord vary widely in different cases. As a rule paraplegia is present, and differs in no respect from that dependent on any other cause. But sometimes the paralysis is of a more limited kind. In a case under the writer's care in Stephen Ward (Nov., 1878) the tumour (a glioma) occupied the cervical region of the cord and caused acute hæmorrhagic myelitis around it. The symptoms were paraplegia and cystitis with paresis of the right arm, and the temperature rose to 108·5° F. shortly before death. Theodor Simon has recorded several remarkable cases in which there would appear to have been a complete absence of all symptoms ('Arch. f. Psych.,' 1874).

Tumours of the meninges producing paraplegia by compression of the cord are not quite so rare as growths in the cord itself.

Syringomyelia.—There is a condition in which the central canal of the spinal cord remains of the large size it is in the fœtus during the whole of adult life. This arrest of development is known as Hydromyelus. Probably some cases are not congenital, but have their origin during adult life in a "dropsy" and dilatation of the central canal. Such a case was described and figured by Gull in 1862 ('Guy's Hosp. Rep.,' 3rd series, vol. iii, p. 250) as "chronic cervical hydromyelus comparable to a chronic hydrocephalus." The canal in this case and in others was lined by its characteristic columnar epithelium, and much of the central grey matter was atrophied around it.

Hydromyelus seldom produces any spinal symptoms during life. In children suffering from spina bifida it is not uncommon. We now confine the term hydromyelus to dilatation of the central canal, and apply to cavities of other origin, mostly posterior to the canal, the name *syringomyelus*, originally invented by Ollivier, though not with the object of conveying this distinction.

In such cases a cavity is found in the cord, usually posterior to the central canal, which is intact, and often situated in the median line so as to be naturally regarded as due to persistence of the primary neural cleft which remains as the posterior median fissure of the cord. In other cases syringomyelia is certainly not a congenital malformation, but a morbid product formed in adult life, and sometimes the result of cystic degeneration of a glioma. Even when no gliomatous tissue can be demonstrated in the wall of the cyst, syringomyelia is found associated with multiple gliomata of the cord, as in a case under the writer's care in 1890. Dr Frederick Taylor showed a beautiful specimen in which no clear indications of the presence of a new growth could be made out, and collected ten other cases.*

The first case in which atrophic paralysis of the arms was observed in connection with a cavity in the cord was recorded by Gull in 1862, although this was one of hydromyelus rather than what we now recognise as syringomyelia.

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* See his two papers in the 'Pathological Transactions' (vols. xxix, p. 21, and xxxv, p. 36). Also Dr Bullard's case of glioma with syringomyelia in the 'Reports of the Boston City Hospital,' 1899, p. 197.

The patient, a journeyman tailor aged forty-four, had suffered for thirteen months from loss of power in the little and ring fingers of the right hand, and for a shorter time from a similar affection of the corresponding fingers of the left hand. The muscles of the hands had undergone extreme wasting. He caught typhus in the hospital, and died of that disease. In the cervical and upper dorsal regions of the cord the place of the grey matter was taken by a large quadrilateral cavity which was supposed to be a dilated part of the central canal.

Cases were soon afterwards recorded by Schüppel, Hallopeau, Westphal, Th. Simon, and many others.

The symptoms of syringomyelus are sometimes very slight or absent, so that the condition is accidentally discovered after death; but when well marked they allow of a probable diagnosis. The most characteristic of these symptoms is a dissociation of the sense of touch from that of pain, analgesia without anæsthesia. Along with ordinary tactile sensations the muscular sense is preserved, and, along with the sense of pain, that of temperature is lost. Numbness, formication, delayed sensation, and other forms of paræsthesia may sometimes be observed.

The horizontal distribution of the lesion is "central," *i. e.* in the deep part of the cord, the grey matter around the central canal and the posterior cornua, while the posterior root zones are unaffected. This localisation agrees with the results of histological inquiry, and of experiments on the lower animals as to the afferent paths in the cord. The symptom, not infrequent in these cases, of muscular atrophy with loss of power, points to an extension of the cavity so as to involve in pressure or in degeneration the anterior cornua.

The vertical or longitudinal distribution of syringomyelus is usually in the cervical region. Hence the upper limbs are commonly affected by muscular atrophy and paresis, particularly the shoulder and the hands. The hands are often found in the clawed condition caused by paralysis of the ulnar nerve. The muscles of the trunk sometimes share in the wasting, and, usually after the arms have been affected, the lower extremities also.

More frequently, however, the chronic lesion in the upper region of the cord causes a secondary descending sclerosis of the crossed pyramidal tract in the lateral columns, and consequent spastic symptoms in the legs.

There are seldom any pelvic symptoms, and in most cases no cerebral symptoms either; but some writers have met with eclampsia, and others with limitation of the field of vision or want of perception of certain colours. Much more constantly observed are certain trophic lesions, bullæ, and painless sores on the fingers or the feet, pigmentation of the skin, and chronic affections of the joints. A monograph on the subject by Schlesinger has been translated by the New Sydenham Society: the trophic arthroses have also been described by Mr Targett in his Erasmus Wilson Lectures before the Royal College of Surgeons in 1895. There is a specimen of a joint in syringomyelia in the museum of the College, and another in the Guy's Hospital Museum, which occurred, but was not recognised, in 1884.

The course of syringomyelia is very slow—measured by years, not months.

The diagnosis is often impossible, but becomes easier as more cases are recorded. The diseases which have been confounded with it are Pachymeningitis cervicalis hypertrophica, and other forms of progressive atrophic paralysis of the arms, central myelitis, amyotrophic lateral sclerosis, multiple peripheral neuritis, glioma of the cord without central cavitation, and Morvan's disease, if this last exists.

The fact is that several diseases which have been given different names agree in the parts of the cord or in the nerves which are affected, and therefore produce the same symptoms, *i. e.* they are clinically identical; while the difference in their cause and origin can often be only ascertained after death.

The cases of painless whitlow and other trophic lesions which occurred in Brittany, and were described by Dr Morvan as a new disease in 1883, were probably, as one was certainly proved, cases of syringomyelia. The multiple neuritis produced by the poison of the *Bacillus lepræ*, with its marked dystrophic effects, resembles syringomyelia in its result, but is totally different in pathology. Possibly, as Dr Zambaco believes, from his experience in the East, some of the cases called Morvan's disease among the fishermen of the Brittany coast may be really leprous, like the cases of anæsthesia with painless bullæ and sores among the fishermen of Norway.*

Anæmia of the spinal cord.—Unless the modern interpretation of a very old observation is incorect, one of the possible causes of paraplegia is an arrest of the arterial blood-supply to the lower part of the cord. Nicolas Steno in 1667, and Haller afterwards, stated that after tying the abdominal aorta in animals the lower limbs became powerless, and remained so until the ligature was removed. This experiment was long afterwards repeated by Sir Astley Cooper and by others; but until 1869 the explanation universally accepted was that the muscles were paralysed because they and the peripheral nerves were deprived of blood. Schiffer, however, rightly argued that if this were the case an interval would elapse, instead of the effects being observed a few seconds after the operation. Not only the heart, but the skeletal muscles retain their contractility for some time after removal from the body. The cause of the paralysis is no doubt anæmia of the lumbar cord.

The abdominal aorta is sometimes completely obstructed by an embolus; but the clot is usually impacted close to the bifurcation of the vessel—too low to interfere with the circulation in the lumbar enlargement of the cord.

In the 'Guy's Hospital Reports' for 1857 Sir William Gull gave the clinical history of a patient whose aorta was from some cause or other obstructed, and who was attacked with paraplegia. The man was a shipwright, and in the beginning of March, 1855, while apparently in perfect health, he was suddenly seized, being in a stooping position, with pain in the loins. This went off after he had rested for a few minutes; but on his resuming his work it returned and extended down the legs, with a sense of numbness, soon followed by entire paralysis, both of sensation and motion, from the loins downwards. After a few days sensation returned, and he gradually improved, but the legs remained unsteady. The paraplegic symptoms led to his coming under Gull's care in June of the same year. It was found that there was no pulsation in the abdominal aorta, nor in the arteries of the lower limbs. The right superior epigastric artery was already distinctly enlarged, and could be seen pulsating. In the course of the two following years he regained power to walk tolerably well, but at the end of that term his gait was still languid, and from time to time he had slight returns of weakness and numbness in the legs. His feet also were cold and damp. Using the legs brought on increased weakness and numbness.

An intermittent form of paraplegia has been noticed in horses, as a result of obstruction of the aorta; a loss of power in one or both of the fail in their turn; the act of swallowing becomes impossible, and death by suffocation closes the scene. Towards the last it is sometimes noticed that the speech is embarrassed, that liquids regurgitate through the nose from paralysis of the palate, and that the masticating muscles and those of the face are weakened. The pupils are sometimes unequal, and transitory diplopia has twice been observed. There may also be very rapid action of the heart when the lesion has reached the upper part of the cervical cord.

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* Compare on this curious subject a valuable article by Dr J. J. Pringle, 'Journ. of Dermatolog.,' 1893, vol. v, p. 193, and a review by Dr Pernet (ibid., vol. ix, p. 454).

In some cases the order in which different parts are paralysed is said to be reversed. The fatal illness of the great naturalist Cuvier was of this kind, and terminated in less than seven days. His first symptom was a sensation of discomfort at the epigastrium. Next morning he experienced a difficulty in deglutition, and in the evening he could swallow nothing, and had marked loss of power in the upper limbs. The paralysis gradually became absolute, and affected the lower limbs also. Such a course of events can only be explained on the supposition that the morbid change (of whatever nature) descends the cord, instead of ascending; and that it is limited to grey matter, leaving the white columns unaffected, which of course contain at each level fibres belonging to all parts of the body below. It is doubtful whether these cases can fairly be brought within the narrow definition of Landry's paralysis recognised by systematic authors; they only resemble the typical cases in there being no recognisable lesion, and in the sensibility of the paralysed parts remaining perfect.

The most characteristic symptoms of Landry's palsy seem to be the following. The affected limbs lie flaccid and free from spasm. At first reflex movements can be excited; after a few days both superficial and "deep reflexes" are lost; but the electrical contractility of the muscles persists to the last, nor do they waste. The functions of the bladder and rectum are unimpaired; there may for a time be retention of urine, but this quickly passes off, so that a catheter has seldom to be used. The patient may complain of slight feelings of numbness, or of formication, but the paralysed parts are still sensitive to touch and to painful impressions. No pains are experienced in the affected limbs, nor is there any tenderness of the spinal column. The patient may be so completely free from discomfort, and from all the ordinary signs of serious illness, that it may be difficult for those about him to realise the gravity of his state. In one of Landry's original cases M. Gubler thought for a day or two that the paralysis was feigned, nor was there any apprehension of danger until the patient was within a few hours of death.

Pathology.—Morbid anatomy has hitherto thrown no light whatever upon the nature of this disease. Westphal ('Arch. f. Psych.,' 1876) examined the spinal cord microscopically in three typical cases with absolutely negative results; and the same conclusion followed autopsies by Vulpian and Hayem. In other cases enlargement of spleen has been noticed.

Before, however, accepting any but an organic change as the origin of the symptoms, it would be necessary to examine the whole of the motor tract, including the anterior roots of the nerves and their trunks.

Westphal supposes that the affection of Landry may be due to the operation of some hitherto unrecognised poison; others assume the presence of a microbe. Nauwerck and Barth have published, in the fifth volume of Ziegler's 'Pathologische Beiträge,' a case of Landry's acute ascending paralysis, in which they found after death the cord normal, but to prove it; for such gaseous emboli are only formed after liberation from a far higher atmospheric pressure, and far more suddenly, than is possible in cases of divers' paralysis.

The autopsies have not thrown much light on the pathology of the disease. In most "congestion" of the brain and cord is recorded, and in others they were found to be perfectly normal.

Dr Andrew Smith, of New York, says that the symptoms are—severe pains in the limbs, particularly in the bones of the lower extremity, gastralgia and vomiting, headache, vertigo, and sometimes coma, and paralysis, usually of the legs, but sometimes of the arms, with anæsthesia and retention of urine. The attack comes on quietly, after the diver has emerged into the upper air, and the symptoms do not in mild cases last more than a few hours, but may persist (as in the writer's case) for months. The prognosis is good on the whole, but death may occur almost suddenly in the most severe cases. The worst effects are seen in stout, plethoric men, and in those addicted to drink, and those who are new to the work are most liable to attack. The treatment is to relieve the severe pains by opium, and to give ergot, with tincture of ginger if there is gastralgia and vomiting. If a diver returns at once to the caisson the symptoms disappear, and the rational and efficient precaution to prevent these often severe and sometimes fatal attacks is for the men to pass through a chamber which allows of slow and gradual transition from the pressure of two or three atmospheres in the caisson, to that of the external air.

The writer has only once seen a case of this remarkable disease, in a patient in John Ward, No. 10, in 1887. He slowly recovered.

Hyperæmia of the cord.—All pathologists are agreed that distension of the blood-vessels within the spinal canal, if found in the dead body, affords no proof that they were over-full during life; it may be due either to the mode of death, or to gravitation afterwards.

There may, no doubt, be reasons for believing in the occurrence of morbid processes which cannot be recognised by the anatomist; but there are no such reasons for recognising hyperæmia or congestion of the cord as a permanent morbid condition. In the early days of pathology, writers assigned all diseases their anatomical lesions in an ascending order of severity. They found that almost every organ was liable to certain chronic changes which could be regarded as inflammatory, and any cases which seemed to stand lower in the scale were ascribed to hyperæmia or to anæmia, according as the one condition or the other seemed the more easily to be brought into harmony with current theories.

In 1866 Dr Radcliffe ('Reynolds' System,' ii, p. 619) had under his care a woman who had been found paralysed in all her limbs the morning after having had her menstrual flow checked by an alarm of fire. Among the other symptoms were tingling in the fingers and toes, some degree of general hyperæsthesia, and a dull burning aching in the limbs and along the spine, without special tenderness on pressure over the vertebræ, but with increased sensitiveness to a hot sponge. The bladder and rectum performed their functions naturally. There was no over-excitability of the reflex centres in the lower part of the cord. Within two or three weeks she began to improve, and at the end of five months she left the hospital convalescent.

It is easy to pick out cases of paraplegia presenting the same symptoms those of Dr Radcliffe's patient, and to label them accordingly; but no ound appears for giving to these cases the title of spinal congestion rather an to others in which the paraplegia is incomplete and terminates in recovery. Dr Radcliffe laid stress upon the sudden commencement as characteristic; while Erb affirms that a *slow* development of the symptoms distinguishes hyperæmia of the spinal cord. He also mentions, as one of the most important signs of this affection, its fluctuating and changeable course; but in some cases of myelitis inexplicable variations in the degree of motor paralysis and anæsthesia occur from day to day.

The absence of paraplegia in cases of mitral disease, in which the cord and viscera are certainly congested, is a strong reason against spinal congestion being recognised as a cause of paralysis.

Concussion of the cord.—In ordinary cases of fracture and dislocation of the spine, such as come under the care of the surgeon, the paralysis, which is commonly present from the first, is attributed either to crushing of the cord by a displaced vertebra or to effusion of blood.

A case recorded by Gull shows how a comparatively slight accident may be attended by fatal consequences in this way.

A man was carrying coals on his back down some cellar stairs when his foot slipped, so that he fell, and the sack of coals upon him. He died in thirty-four hours. Two of the cervical vertebræ were found torn asunder, but this was not the cause of the paraplegia from which he had suffered immediately after his fall, for there was no displacement of the bones, nor any injury to the posterior ligament of the spinal membranes. There were, however, several spots of ecchymosis in the posterior grey cornua of the cord, as well as in the posterior white columns and the posterior half of the left lateral column.

It is perhaps worthy of notice that even these lesions did not actually explain the paraplegia, for there is no proof that the motor tracts on both sides were injured. This, however, exactly illustrates the view which should be taken of capillary ecchymoses of the nervous centres, whether cerebral or spinal. It is not that they themselves produce paralytic or any other symptoms, but that their presence, showing that the violence was sufficient to tear asunder vessels, shows that it must have also been enough to lacerate the nervous elements themselves, which are more delicate.

These considerations tend to explain the fact that spinal injuries are sometimes followed by paraplegia, and may even terminate fatally without any morbid change whatever being discoverable even with the microscope. Leyden says that he met with an instance of this kind in which death occurred within five days, and in which he failed to detect any lesion, even after having hardened the tissues with chromic acid. Such cases are commonly described as cases of *spinal concussion*, and supposed to depend upon a molecular change in the nerve-fibres and cells of the cord due to the jarring force of the injury. Erichsen found that after a railway accident those persons who are sitting with their faces to the engine are less likely to suffer from spinal injuries because, being thrown forwards, they would break their fall with their hands, whereas those who sat with their backs to the engine would be driven against the carriage when its motion was suddenly arrested, so that the spinal cord would be violently shaken.

The cases of which one can most confidently speak as being examples of concussion of the cord are those in which a blow or a fall is instantly followed by paralysis, and in which recovery takes place so rapidly that the alternative diagnosis of hæmorrhage can be satisfactorily rejected. Such an instance, perhaps, is one related by Erb.

A man, aged fifty-five, fell twenty feet from a tree upon his heels and ischia. He was not unconscious, but was at once unable to walk, and had to be carried home. He experied severe pains in the sacrum and in the lower limbs, but these passed off after a time. ere was no loss of sensation, but the legs were said to be entirely motionless for a week. en he came under medical observation at the end of four weeks he could only make a steps at a time, and slowly and with hesitation. Reflex movements could be produced usual. The excitability of the nerves and muscles by electricity was much diminished. lvanic treatment, applied both to the spine and to the legs, was attended with great cess. He was soon able to walk well, and in three weeks he was discharged cured.

In most cases, however, when a railway accident is followed by ralysis this does not appear until after some time. The interval is ten of several days' duration, and Erichsen says that it may even last o or three months. During this time the patient is not, indeed, well; is suffering from other effects of the injury, but he frequently has no ea that his spine has been hurt. It is difficult to see how the diagnosis "concussion" is to be established as against that of myelitis when the mptoms are thus remote.

Acute Ascending Paralysis.*—Diffuse myelitis sometimes spreads pidly upwards along the cord, and destroys life in a few days. To such ses, which are not very infrequent, the name of acute ascending paralysis ight be fairly applied. It is, however, now usually given to a different fection, one in which the most minute scrutiny has hitherto failed to veal any lesion of the cord. Landry published ten cases of what is now garded as this form of paraplegia in the 'Gazette Hebdomadaire' for 59. Kussmaul's two cases of "Fatal Paraplegia without assignable use" (cited by Ross) were published in the same year.

It is said to be definitely characterised by its symptoms; for in a e of ascending acute myelitis there is marked anæsthesia, the bladder l rectum are totally paralysed, bedsores are formed at a very early iod, and the faradic excitability of the muscles soon becomes extinshed; whereas in the acute ascending paralysis of Landry none of these enomena are observed.

Onset, course, and symptoms.—Sometimes there are prodroma, conting of slight febrile disturbance, malaise, dragging and shooting pains in back and limbs, sensations of numbness and formication in the feet and the tips of the fingers, and, above all, a feeling of great muscular haustion and weakness. The patient may go on complaining in this way a day or two, or for a week, or even (in one recorded instance) longer. re often no such symptoms arise, the earliest indication that anything the matter being a loss of strength in the lower limbs, which rapidly sses into complete paraplegia. The feet first become motionless, then e legs, and afterwards the thighs. Soon the trunk is involved; aining becomes impossible during defæcation, and in coughing or sneezg; the intercostal muscles are paralysed in succession from below uprds. At the same time, or even earlier, the hands are affected; the tient is unable to write or to feed himself; his grasp rapidly becomes feebled; the loss of power extends to the arms and shoulders. Lastly, e muscles supplied by the upper cervical nerves, including the diaphragm, fail in their turn; the act of swallowing becomes impossible, and death by suffocation closes the scene. Towards the last it is sometimes noticed that the speech is embarrassed, that liquids regurgitate through the nose from paralysis of the palate, and that the masticating muscles and those of the face are weakened. The pupils are sometimes unequal, and transitory diplopia has twice been observed. There may also be very rapid action of the heart when the lesion has reached the upper part of the cervical cord.

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* *Synonym.*—Landry's paralysis. *Poliomyelitis anterior acutissima* was a name given wrong diagnosis of its nature, both clinical and pathological.

Neither Dr Fagge nor the present writer ever saw a case, and one or two cases cited by Ross appear to be all that have occurred in England during more than thirty years. account of Landry's paralysis in the text is founded on the statements by Erb, hhorst, Ross, and Gowers, and on Remak's article "Spinallähmung" in Eulenburg's al-Encyclopädie' (p. 651). See also Bailey and Ewing's paper in the 'New York Med. rn.,' *June 4th and 11th, 1896.*

In some cases the order in which different parts are paralysed is said to be reversed. The fatal illness of the great naturalist Cuvier was of this kind, and terminated in less than seven days. His first symptom was a sensation of discomfort at the epigastrium. Next morning he experienced a difficulty in deglutition, and in the evening he could swallow nothing, and had marked loss of power in the upper limbs. The paralysis gradually became absolute, and affected the lower limbs also. Such a course of events can only be explained on the supposition that the morbid change (of whatever nature) descends the cord, instead of ascending; and that it is limited to grey matter, leaving the white columns unaffected, which of course contain at each level fibres belonging to all parts of the body below. It is doubtful whether these cases can fairly be brought within the narrow definition of Landry's paralysis recognised by systematic authors; they only resemble the typical cases in there being no recognisable lesion, and in the sensibility of the paralysed parts remaining perfect.

The most characteristic symptoms of Landry's palsy seem to be the following. The affected limbs lie flaccid and free from spasm. At first reflex movements can be excited; after a few days both superficial and "deep reflexes" are lost; but the electrical contractility of the muscles persists to the last, nor do they waste. The functions of the bladder and rectum are unimpaired; there may for a time be retention of urine, but this quickly passes off, so that a catheter has seldom to be used. The patient may complain of slight feelings of numbness, or of formication, but the paralysed parts are still sensitive to touch and to painful impressions. No pains are experienced in the affected limbs, nor is there any tenderness of the spinal column. The patient may be so completely free from discomfort, and from all the ordinary signs of serious illness, that it may be difficult for those about him to realise the gravity of his state. In one of Landry's original cases M. Gubler thought for a day or two that the paralysis was feigned, nor was there any apprehension of danger until the patient was within a few hours of death.

Pathology.—Morbid anatomy has hitherto thrown no light wh[illegible]ater upon the nature of this disease. Westphal ('Arch. f. Psych.,' 18[illegible] examined the spinal cord microscopically in three typical cases with[illegible] lutely negative results; and the same conclusion followed autopsies b[illegible]y pian and Hayem. In other cases enlargement of spleen has been not[illegible]ce

Before, however, accepting any but an organic change as the or[illegible]gi the symptoms, it would be necessary to examine the whole of the [illegible] m tract, including the anterior roots of the nerves and their trunks.

Westphal supposes that the affection of Landry may be due [illegible] to operation of some hitherto unrecognised poison; others assume th[illegible]e p sence of a microbe. Nauwerck and Barth have published, in th[illegible] fif volume of Ziegler's 'Pathologische Beiträge,' a case of Landry's [illegible] acut ascending paralysis, in which they found after death the cord norm[illegible]l, bu neuritis of many of the nerve-roots and extensive destruction of the fibres in both sciatic nerves. Probably most of the recorded cases were affections of the nerve-trunks and not of the spinal cord: but others may be toxic in origin. A fatal case of paralysis at St Thomas's Hospital resembled Landry's cases: it was that of a man who had been inoculated for hydrophobia, and suggested to Dr Bristowe a comparison with the paralytic form of that disease in dogs and in rabbits.

Prognosis.—The mean duration of fatal acute ascending paralysis is said to be from eight to twelve days. It has been known to destroy life in two or three days, but sometimes it runs on for as many weeks, and may end in the patient's recovery. Landry spoke of eight out of his total of ten collected cases (only four of which he had seen himself) as having terminated favourably; but his paper contains no details to justify the diagnosis. Erb says that the disease may stop at any period of its course, even when respiration and deglutition are affected.

Ætiology.—With regard to its causes, nothing is known. It appears to occur mostly in young adults, and more frequently in men than in women, the proportion in Pellegrino-Levi's case being twelve to four. Some cases have been ascribed to exposure to cold; some have occurred during convalescence from enteric fever and acute diseases.

Rarity.—Landry's paralysis is of exceedingly infrequent occurrence in England, and not a single typical case has been recorded in Guy's Hospital. Wilks ('Lectures,' p. 274) relates seven cases of acute ascending paralysis, and in three of them the cord was found normal; but apart from the difficulty of accepting negative conclusions before modern histological methods were in use, the presence of anæsthesia, of vesical symptoms, and of pain in all these cases brings them under the clinical category of acute ascending diffuse myelitis. A case in a young man which ended favourably is recorded by Dr Ross, and another which proved fatal; but the latter he regards as extremely doubtful. Other authors, Bristowe, Buzzard, Gowers, Bastian, Bramwell, describe the disease from French or German accounts, rather than from their own experience. The early cases of Walford ('Brit. Med. Journ.,' November, 1854, Handfield Jones (ibid., October, 1866), and Harley ('Lancet,' October, 1868), in which last Lockhart Clarke found extensive lesions in the cord, cannot be admitted as genuine cases, and many of those published abroad are either myelitis or peripheral neuritis.

The fully described cases are certainly very few, even abroad, perhaps not thirty. The rapid course, the successive implication of legs, arms, and trunk, and finally of the bulbar nerves, including the portio dura, the absence of marked anæsthesia or pain, the absence of bedsores and rectal or vesical disturbance, the loss of knee-jerk, the absence of atrophy or spasm of the paralysed muscles and their retention of electrical contractility—make up a striking combination. The usually fatal course and negative anatomical result complete the type.

Probably no *treatment* is effectual, but the application to the spine of ice, of heat, of the actual cautery, and of other counter-irritants, have all been recommended.

Alcoholic paraplegia.—Under this name Wilks described a form of partial motor paralysis of the lower limbs not uncommon, particularly "in ladies who have given themselves up to brandy-drinking." Pains in the legs and anæsthesia are also present.

These symptoms, with the dragging gait from dropping of the toes, the integrity of the functions of the pelvic organs, and the favourable prognosis under proper treatment, make up a recognisable clinical "disease." As Buzzard suggested, symmetrical peripheral neuritis is its pathological cause (vide *supra*, p. 595). Gowers takes the same view, and regards as minor cases of the same malady those which have been described as Alcoholic Ataxia and Pseudo-tabes potatorum. This was in fact Wilks's own opinion, for he says that sometimes the symptoms are almost confined to the legs, and resemble in character those of locomotor ataxia.

In Dr Broadbent's remarkable case of alcoholic paralysis ('Med.-Chir. Trans.,' 1884) the cord was found normal, but the nerves were not examined. He compares it with Landry's paralysis. It was in some respects unique; but his other cases agree with Buzzard's cases of alcoholic neuritis in occurring in women, in being chiefly paraplegic, and in the muscles being tender on handling.

On the whole it seems probable that drink produces paraplegia occasionally by means of myelitis, but more frequently by peripheral neuritis. The latter not uncommon condition is recognised by the symptoms described in a previous chapter (p. 593), by the absence of pelvic symptoms, and by the frequent implication of the hands.

Syphilitic paraplegia.—That paralysis of the lower limbs may be one of the remote effects of syphilis has long been a well-known fact, but the changes which occur in such cases are not uniform. Hutchinson has recorded several cases of syphilitic paraplegia due to myelitis, and Buzzard believes that, in a person between twenty and forty years of age, a case of paraplegia, when not associated with Bright's disease or embolism, is (like hemiplegia under the same conditions) in nineteen cases out of twenty the result of syphilis. But primary acute softening of the cord is common; primary acute softening of the brain is rare, and probably Heubner is right when he asserts that the *lues venerea* affects the spinal cord far less often than the brain. Buzzard's limits of age appear to be too narrow, for in a case observed by Wilks the patient was a woman of fifty-three, and among the five or six other cases which have been examined in the *post-mortem* room of Guy's Hospital within the last few years, one occurred in a man aged fifty-seven, another in a woman aged forty-seven. Syphilitic paraplegia is most often due to myelitis from diseased arteries.

The frequency with which nodes are seen upon the inner surface of the skull, pushing the dura mater inwards, at one time led to the supposition that a similar affection of the bony walls of the spinal canal might occur. But this is not borne out by observation in the deadhouse. A gumma beginning in the interior of the cord is almost unknown, but it sometimes occurs in the membranes, and produces paraplegia by compression.

In certain cases supposed to be examples of syphilitic paraplegia, no obvious morbid change has been discovered in the cord, and they may have been due to syphilitic neuritis; or, again, syphilitic endarteritis may probably in the cord as in the brain sometimes lead not to myelitis, but to passive softening of the tissue.

A man, aged twenty, was in 1877 under treatment by the late Mr Davies Colley for syphilis, when he became paraplegic and was transferred to a medical ward and died two months later. The cord in the mid-dorsal region was flattened and soft for about an inch and a half of its length; the antero-lateral columns and the grey matter were especially

affected, the latter being of a rusty brown colour. To the naked eye there was no obvious change in the pia mater. But when a piece of it corresponding with the softened parts was placed on a microscopic slide and examined with a lens, the walls of the arteries were at once seen to be enormously thickened and degenerated. By reflected light they looked like solid, opaque white cylinders; by transmitted light they appeared black. We could not find any in which the affection was in an earlier stage, so that we might have compared it with Heubner's description of syphilitic arteritis in the brain.—C. H. F.

Reflex paraplegia.—It has often been suspected that a loss of function in the spinal cord, causing paralysis of the lower limbs, without any precedent anatomical lesion, may be an indirect result of certain visceral diseases.* Such cases would be comparable with two already mentioned in which paresis of one arm was caused by a carious tooth. Hammond relates the case of a girl who was brought to him on account of paraplegia which had suddenly developed. He administered several doses of santonine, followed by castor oil; many round-worms were passed, and the paralysis disappeared in the night.

In 1860 Dr Brown-Séquard published in the 'Lancet' an elaborate paper to prove that irritation of the afferent nerves of a diseased organ may set up a reflex spasm in the blood-vessels of the spinal cord, so as to render it anæmic and impair its functions. In the following year, however, Sir William Gull refuted this opinion in the 'Guy's Hospital Reports,' and no one now accepts it. Nor has a suggestion of Jaccoud's been adopted that reflex paralysis is due to "exhaustion" of that portion of the cord upon which fall the stimuli conveyed by the afferent nerves of an irritated part. The doctrine of "inhibition" would better meet the case; its experimental application to paraplegia seems to have been first made by Lewisson ('Reichert's Archiv,' 1869). The cases of paralysis caused by diseased teeth, and the paraplegia from worms in Hammond's case, seem to accord with Lewisson's experimental results, since they subsided as soon as the source of irritation was removed. So also Graves placed on record the case of a man who was admitted into the Richmond Hospital in 1835 with partial paraplegia of two weeks' duration, and a chronic stricture; in a few days after the introduction of a catheter a remarkable improvement took place, and within a month the power of the lower limbs was completely restored. Again, cases are recorded of the reduction of a displaced uterus being followed by a marvellously rapid recovery from paralysis; but we cannot exclude the supposition of hysteria from these cases.

The majority of cases of paralysis which are supposed to be of reflex origin run a protracted course, not only when the disease which is supposed to exert the inhibitory influence is itself permanent, but after it has departed. In such cases there must be more than reflex paraplegia; and, indeed, myelitis has sometimes been discovered after death. Of a particular group, in which paralysis appears to be secondary to some affection of the bladder or urethra, three instances were recorded by Gull in 1856 ('Med.-Chir. Trans.,' xxxix), and in each of them the spinal cord was found softened. Two similar cases came under the observation of Leyden; the paralysis seemed to be the result of a long-standing stricture, and there was inflammation of the upper portion of the lumbar enlargement with granule masses.[*]

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* In the Guy's Hospital Library copy of the 'Medico-Chirurgical Transactions' for 1833, containing Mr Stanley's paper on "Reflex Paralysis," there is a pencil-note by Dr Wilks, to the effect that the majority of his cases were wrongly interpreted, and were really examples of a primary inflammatory softening of the cord, attended with secondary cystiti and nephritis.

In the 17th chapter of his Essay on "Injuries and Diseases of Nerves" Mr Bowlby has collected a series of cases of "reflex paraplegia," and subjected them to the necessary criticism.

When chronic paraplegia develops itself in a person who has for years had a stricture, or who has recently had dysentery or some other intestinal affection, or who has a retroflexed or prolapsed uterus, one must not forget that this may be a mere coincidence. Myelitis is certainly present in most of the cases once supposed to be of reflex origin, and peripheral neuritis in others. If not so unreal a condition as congestion of the cord, reflex paraplegia must be at least an exceedingly rare malady.

Hysterical paraplegia.—It is a well-recognised fact that one of the symptoms of hysteria is paraplegia; the proof being that young women who have previously suffered, or are actually suffering, from other hysterical symptoms are apt to be affected with paralysis of the legs, which gets well after a time, or sometimes suddenly, under the influence of some mental or moral shock. To define as accurately as possible the characters of such an affection must evidently be of great importance in reference to prognosis and treatment; for the diagnosis cannot be taken for granted upon the mere fact that the patient has hysteria; while, on the other hand, affections really due to hysteria are occasionally met with in persons who would not be suspected of it.[†]

Mere malingering is not difficult to detect, but hysterical paraplegia is a very different thing. Apart from the probabilities afforded by the age and sex of the patient, the following points are of service:—There is usually retention but seldom or never incontinence of urine; obstinate constipation but never incontinence of fæces; and bedsores do not form. The motor palsy, though absolute at certain times, is from time to time only slight; sensory palsy, patches of anæsthesia, hyperæsthesia and analgesia are much more marked than in ordinary paraplegia, as much as in total transverse lesions with pelvic symptoms. Analgesia with integrity of sensations of touch and of temperature, and anæsthesia without motor palsy are almost decisive of hysteria. Electro-sensibility is often small or absent, but the muscles respond normally to galvanic or faradic stimuli. The paralysed limbs are cold but do not waste. Rigidity and contraction, most often flexion, are frequent and early symptoms, but they are completely removed under chloroform and during sleep. The knee-jerk is sometimes absent, more often exaggerated. Hysterical girls are well nourished in spite of their paralysis, and their legs are usually cold and pallid.

We ought to be extremely careful in diagnosing paraplegia as hysterical, since neurotic girls are not less liable than other people to myelitis, caries of the spine, and other causes of organic paraplegia.

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[*] Tiesler and Feinberg and Klemm succeeded in causing an inflammation of the lower part of the spinal cord in animals by setting up neuritis of the sciatic nerves. Roesingh, however, has repeated some of these experiments with negative results, so that the question can hardly be regarded as finally settled.

[†] There was a remarkable want of agreement among former writers as to the criteria of hysterical paraplegia. Radcliffe (and also Bastian) stated that the paralysis is usually incomplete; Wilks that completeness is, in a doubtful case, an argument for paraplegia being due to hysteria. Duchenne attached much importance to a diminution of electro-sensibility without loss of electro-contractility; but Reynolds has related two cases in which tactile sensibility, sensibility to electricity, and electro-contractility were all perfect.

The *treatment* of hysterical paraplegia is that of hysteria, and electricity should only be used, if at all, for its effect upon the mind.

NEURASTHENIA SPINALIS.—Patients frequently seek advice who suppose that they are suffering from spinal weakness. As a rule they are women or young men between eighteen and twenty-five. They complain of muscular fatigue and failing strength and energy. If they walk they feel aching pains or stiffness in their limbs, and when they have to stand their legs seem to give way under them. They feel tired, and their backs ache all day long, even before they get out of bed in the morning. "Spinal Neurasthenia," as a special name for these cases, was originally proposed by Beard and Rockwell in 1871, and was accepted by Erb, Ross, and other systematic writers. In some cases dragging pains in the limbs are complained of; and very often there is a peculiar pain in the back, seated apparently in the lumbar muscles, and increased by movements of the spine. Sometimes local tenderness of certain spinous processes is present, exactly as in rachialgia or *spinal irritation*. Often there is mental disturbance indicated by sleeplessness, anorexia, flatulence, timidity, and depression of spirits. These are supposed to be due to over-exertion of body or mind, especially during the hours that ought to be spent in sleep, or to some form of exhausting emotion, disappointment, anxiety, or what is called "worry." In youths and men under thirty, however, such complaints usually end in confession of solitary vice or of excess in venery; in fact, their condition is that long recognised as a form of sexual hypochondriasis, or, as it used to be called, tabes dorsalis.*

It is supposed by Erb that the state of the spinal centres in such cases is the same as in the healthy cord when fatigued, and that the difference is in a natural period of rest failing to restore it to vigour and activity. This view seems reasonable enough.

The *diagnosis* from myelitis, tabes, and other serious spinal diseases must be based mainly upon the absence of objective symptoms of a definite lesion of the cord, in contrast with the despondency with which the patient complains of his subjective sensations. Neurasthenia is distinguished from hysterical paraplegia by the absence of retention of urine and of paresis. In women, especially about the menopause, it is apt to be associated with alcoholic dyspepsia; in men with syphiliphobia, but very rarely indeed with syphilis.

Prognosis and treatment.—This miserable condition, as much mental as physical, is reached by slow and gradual steps, but may at last compel the sufferer to give up his occupation and to renounce all society. It is often obstinate, lasting for many months, or even years, and relapses sometimes occur. It never passes on into any organic affection, but may be the first stage of insanity. As a rule, however, the prognosis is good if the patient will take advice. The treatment should be directed entirely to the general health, and away from the spinal symptoms. We recommend cold sponging, exercise out of doors, abstinence from liquor and from sexual indulgence, and we rouse the patient's will to minister to himself. Early hours and adequate sleep are needful, and we prescribe steel or strychnia, with cod-liver oil and abundant unstimulating fattening food.

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* The cases cited in a work by Surgeon-Major Neale, published under this title in the year 1806, are many of them of this character. Similar symptoms were ascribed to a similar cause by the ancients and by the English dramatists of the seventeenth century, and the weakness and pain in the back, as well as the more or less marked loss of virility, gave the term tabes dorsalis a bad repute, which still clings to the very different disease now known under that title.

While there is no doubt that we meet with cases showing the symptoms above described, it appears to the writer very doubtful whether it is desirable to give them the recognition implied by such titles as neurasthenia or nervous debility. They are more appropriately regarded as neurotic or hypochondriacal. If no organic lesion is present, we should seek to trace such symptoms to their origin, either in abuse of the sexual functions, or in drink, or in insufficient sleep, or occasionally in mental or bodily strain continued beyond a patient's powers. The symptoms are often accompanied by that self-inspection and exaggerated view of the importance of one's own sensations which may lead to mental aberration. Women should shrink from the imputation of "nervous debility" as from that of hysteria; and schoolboys, young men, clergymen, artists, and literary men should be taught that "neurasthenia" suggests masturbation, or at best mental idleness, conceit, and selfishness. Bodily work, early hours, cold baths, substitution of porter for wine and spirits, and such moral treatment as will restore manly self-control are the best means of overcoming neurasthenia.

Rachialgia.—In 1828 Dr Thomas Brown, of Glasgow, drew attention to an affection attended with pain and tenderness in one or more of the vertebræ, and termed it *spinal irritation*. He was followed by Mr Teale, of Leeds (1829), by the brothers William and Daniel Griffin, of Limerick (1834), and by Stilling, of Cassel (1840), the last of whom devoted a volume of 540 pages to the subject. All these writers gave a wide scope to the definition. They detected tenderness on pressure over certain spinous processes in persons suffering from various neuralgic and other affections, and they maintained that in all such cases a morbid state of the spine was the fundamental disease. They enlarged their definition, so as to include transitory paralytic affections and a variety of other neuroses, and in this were no doubt influenced by the name they had adopted; for it would be difficult to exclude from "spinal irritation" almost any "functional" disorder of the lower neurons from tetanus to pleurodynia.

It is not surprising that these views met with much opposition. They were criticised in trenchant style, but very justly, by Romberg, and they have never met with general acceptance in this country. Stilling and Ollivier maintained that "spinal irritation" was due to the congestion of the cord, and more recently Hammond asserted that the essential condition was spinal anæmia. The boundary line between spinal irritation and other affections has been repeatedly shifted, and one is tempted to ignore it altogether.

There are, however, some cases which deserve the name rachialgia, defined as a neurosis marked by pain and tenderness in the back, but free from any symptoms of organic lesion of the vertebræ or of the cord. Rachialgia may be regarded as a form of neuralgia affecting the posterior branches of the spinal nerves, and perhaps the filaments distributed to the meninges of the cord. It is true that the law of Hilton and of Van der Kolk would explain how pain may be referred to the cutaneous nerves of the back, even though its starting-point is within the spinal cord; but after all, this question of its centric or excentric seat is not peculiar to rachialgia, but concerns every form of neuralgia.

Although spinal irritation is a neuralgia, it is usefully considered in this place, on account of the importance of the diagnosis between it and the more grave diseases of the vertebræ and of the cord—just as it is convenient to classify pleurodynia with affections of the chest, and gastralgia with those of the stomach. Considering the abuse of the term "spinal irritation," it is best to denote this condition exclusively by the word *Rachialgia*, which was long ago employed by Joseph Frank, and corresponds with those applied to other local neuralgic affections.

Symptoms.—The severity of the pain varies indefinitely in different cases. Sometimes there is no spontaneous pain at all; but by pressing upon the different spinous processes we may find that some of them are tender. Great stress was laid on cases of this kind by those who pushed the theory of "spinal irritation" furthest. They were in the habit of directing the treatment mainly to the supposed spinal affection, and applied leeches, blisters, or tartar-emetic ointment over the vertebræ. Romberg tells us that little good was effected by these measures, which we can well believe.

In other cases there is a dull aching sensation in the affected part of the spine, or even severe pain. Its development is usually gradual. At first it may be felt only after great fatigue or excitement; but, as time passes, slighter causes excite it and it is less ready to subside, until at last it becomes constant. It is almost always increased by muscular efforts and by movements of the vertebræ, so that the patient is unable to walk far, or to stand upright for more than a short time; even sitting at the piano, the writing desk, or the sewing machine may be too painful to be borne. Sometimes the best way of bringing out the increased sensitiveness of the affected nerves is to pass one's finger along the spine, so as to press upon the vertebræ in succession; sometimes it is more manifest when a sponge wrung out of hot water is drawn down the back; for to the patient this seems to scald at the tender spots, while he experiences no discomfort elsewhere.

There is often a close anatomical relation between the seat of rachialgia and that of any other neuralgia which happens to be present in the same case—the one corresponding with the posterior, the other with the anterior main branch of the same spinal nerve.

One of the cases related by Dr and Mr Griffin (at p. 19 of their work) is that of a girl who complained of headache and pains in all parts of her body. Her whole spinal column was acutely tender. Pressure upon the first or second cervical vertebra caused a pain which shot forwards from the occiput to the brow; a little further down it excited pain in the larynx; over the lowest cervical spine, at the spot where the trachea dips behind the sternum; and still lower, at the middle of the sternum, the ensiform cartilage, and the pubic region successively. This certainly was not due to any direct mechanical impression upon the cord; for when pressure was made behind the trochanter, pain was felt at the iliac crest, on the inside of the thigh, and even in the opposite hip; while pressure upon the thigh or knee set up pains in the shins and toes.

Another patient had great tenderness of all the dorsal and lumbar, but not of the cervical spinous processes. Pressure on the upper dorsal vertebræ caused pain at the middle of the sternum; from the third or fourth dorsal down to the sacrum it excited pain, not in the corresponding points, as usual, but at the ensiform cartilage. Pain at this spot was even brought on by pressure behind the trochanter, upon the muscles of the thigh, or over one knee-joint, and the patient felt the same pain if she chanced to tread on uneven ground, or if a pebble came beneath her feet in walking. To ascertain whether the seventh and eighth dorsal vertebræ were, as usual, more affected than other parts of the spine, firm pressure was made upon them. The result was that she suddenly tumbled forwards insensible, and would have struck her face against the floor had not some one caught her.

A third of their patients was a lady who complained of pain in the face, but who "had no conception that her spine was at all affected." When the second cervical vertebra was touched she sprang up, as if a needle had been driven through the cord, and then fell in a state approaching to insensibility. Out of this stupor she twice started up in the same way, and as often dropped back powerless, her countenance evincing the utmost terror and agitation. As soon as she could speak, she said that she had felt a numbness and sensation as of pins and needles in all parts above the ensiform cartilage. She would on no account permit her neck to be touched again.

A fourth instance was that of a young gentleman, who described himself as suffering from a chronic liver complaint. Pressure upon the spinal column was excessively disagreeable to him. When the finger rested on one of the dorsal vertebræ he grew pale and terrified, saying he felt a sudden thrill through every nerve in his frame. He had an unpleasant feeling about the part for the remainder of the day, and shuddered at the idea of allowing the pressure to be repeated. After a few weeks the experiment was tried again, and with precisely the same results.

A fifth case was that of a boy aged twelve, who fell forwards insensible, as if he had been shot, as soon as slight pressure was made upon the second lumbar spine.

Phenomena somewhat similar, though less marked in degree, are not uncommon. They undoubtedly lend support to the view that the seat of rachialgia is in the cord itself rather than in the nerves. Pressure on the tender vertebra will often bring out or increase the pain in any other part that happens to have its nerves in an irritable condition. Anstie met with a case in which pressure on one spot, over the lowest cervical vertebra, caused exquisite pain, a sensation of extreme nausea, and disappearance of the pulse at the right wrist, that on the left side remaining unaltered. The patient, a young lady, was also seen by Walshe and Reynolds. In the case of a child, brought to Dr Frederick Taylor at Guy's Hospital, it was repeatedly noticed that pressure upon the seventh cervical vertebra at once gave rise to a marked pallor of the left side of the face, which lasted a few minutes.

Ætiology.—Radcliffe believed that the cause of "spinal irritation" is often a strain of the back or a blow, which may have been forgotten by the patient. This supposition is confirmed by the fact that slight railway accidents so often give rise to it; the so-called "Railway spine" is, in fact, in the majority of cases an affection of this kind. Anstie's patient, just referred to, had received a very slight contusion in a collision. Her sister, who was severely injured at the same time, nursed her assiduously for three or four months, and then began to be seriously ill herself. Many think that no reliance can be placed on the statements of persons who have met with such accidents, so far as their subjective symptoms are concerned. But there are cases in which there is collateral proof of good faith.

Among the causes to which rachialgia has been attributed are over-fatigue, exhaustion by night-watching, onanism, anæmia, and starvation; but probably none of these are more than predisposing causes.

A general "nervous susceptibility" certainly plays an important part in the ætiology of the complaint. It is far more common in women than in men, and in persons between fifteen and thirty years of age than those at earlier or later periods of life. An inherited "neuropathic tendency" predisposes to it. Like other neuralgiæ, it often affects women who at the same time are obviously suffering from hysteria; and thus may be associated with any of the varied symptoms of that morbid state. At the same time there are cases of true rachialgia without any other hysterical symptom. The last well-marked case seen by the writer was that a lady of about forty, healthy and active in mind and body.

Diagnosis.—The most important thing is to distinguish this form neuralgia from pains which are the result of disease of the vertebræ, the ord, or its membranes. The pain of rachialgia is intermittent, it is forotten under distracting thoughts, and it is unaccompanied by other symtoms of organic disease of the spine. It is closely related to hysteria, nd to the so-called "nervous debility" above described; but it is more ompatible with good sense, less of a vice and more of a misfortune.

Prognosis.—Rachialgia may develop into marked hysteria in women, r into neurasthenia spinalis in men, but the usual course of the affection towards recovery. Relapses, however, are common, and often the subidence of pain in a particular spot in the back is followed by the appearnce of neuralgia elsewhere.

Treatment.—When we are satisfied that there is no organic disease of hich the pain is a symptom, it is a grave mistake for rest in the horizontal osture to be insisted on. Mr Teale says that he succeeded in curing everal persons belonging to the poorer classes while they were still pursung their work; and it is exceedingly desirable that the patient should have resh air and change of scene, and should be encouraged to take moderate xercise. An abundant supply of good food is of great importance. Radcliffe and Skey advised the use of alcohol; but Anstie would only allow it in great moderation, and only with the meals. This is undoubtedly the wiser course, or we may have not only rachialgia but a worse disease to deal with. If "spinal irritation" is only a combination of hysteria and neuralgia, it is food, not stimulants or narcotics, which is useful. The tincture of steel, arsenic, strychnia, and cod-liver oil are each of them valuable helps in treatment; but the main indication is to provide moderate exercise out of doors, fattening food, early hours, and occupation that will divert he patient's mind from self-consciousness.* Walking, bicycling, gardenng, and horse exercise are each of them useful when the patient is able to ake them, and after such moderate exertion she should lie down before aking the next meal.

Hemiparaplegia.—This is rather an experimental than a pathological ondition, but occasionally a patch of disease, or an accidental injury by sharp instrument, happens to affect one half of the cord, and thus to produce in a human patient what is the effect of division of half the ord in animals. There is loss of power in the half of the body which on the same side as the disease in the cord, and the upper limit of the aralysis varies with the distribution of the highest nerves coming off from e part of the cord below the seat of the disease. Such a condition ay conveniently be termed Hemiparaplegia, whether it is limited to the g or involves the corresponding arm as well. Brown-Séquard in 1860 iscovered this remarkable association of symptoms after transverse everance of one half of the spinal cord in animals. Anæsthesia is present, ot on the same side as the disease, but on the opposite side. That half f the body which retains its motor power loses its sensibility, while the aralysed half has its sensory functions unimpaired or exulted. The crossed næsthesia is due to the fact that the fibres belonging to each sensory nerve-root decussate in the substance of the spinal cord itself immediately above their entrance, or at least before they reach the level of the decussation of the motor tracts in the pyramids. This applies equally to the fibres which convey all sorts of impressions,—whether of touch, or pain, or heat and cold; but with the curious exception that the fibres belonging to the muscular sense run upwards without crossing. Thus there is loss of muscular sense, and with it of electro-muscular sensibility, on the paralysed and not on the anæsthetic side of the body. There is usually hyperæsthesia of the skin on the paralysed side,—the susceptibility to tactile and painful impressions and to change of temperature being all increased.

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* The application of blisters to the spine is recommended by some modern authors, and lder writers speak no less favourably of leeches and of cupping. These measures are now ut of fashion; but in one very severe and obstinate case, which arose out of the Thorpe ailway accident, and which the author saw with Mr. Erichsen and with Mr. Robinson of orwich, nothing gave so much relief as the repeated application of leeches at intervals of a few weeks, and especially at the catamenial periods. The patient was stout and florid. Other applications that have sometimes proved useful are turpentine liniment and unguentum veratriæ; or a bag of hot sand may be placed along the spine.—C. H. F.

No disease of the spinal cord can give rise to paralysis and anæsthesia limited to one and the same side of the body. If a patient is found to have one of his lower limbs, for example, affected in this way, we conclude that the lesion concerns the nerve-roots of that side which form the cauda equina—unless, indeed, it is seated above the decussation of the pyramids in the opposite half of the encephalon. In other words, it is either peripheral neuritis or a cerebral lesion.

On the other hand, it must not be supposed that every affection of one side of the spinal cord necessarily causes paralysis of the same side and anæsthesia of the opposite side of the body. This effect is observed only when the abolition of function in that half of the cord is complete or nearly so. All morbid changes in the nervous structures produce loss of motor power more constantly than loss of sensation, and unilateral lesions of the cord offer no exception to this rule. It is quite possible for such a lesion to give rise to complete paralysis of one lower limb, or of the arm and leg on one side of the body, without there being demonstrable anæsthesia of the limbs on the opposite side. The explanation is that the disease, while it is confined to one half of the cord, leaves a part of that half functionally active. The best marked cases are those which will be discussed in the next chapter as essential spinal paralysis in infants and in adults, where the anterior cornua are the seat of the lesion.

In acute cases the vaso-motor nerves are affected on that side which is the seat of the lesion. The temperature of the paralysed limb or limbs is at first higher than that of the corresponding parts by a difference amounting to 1° or 2° Fahrenheit or even more. After a time, however, it becomes normal, and sometimes falls to a still lower level.

The reflex excitability of the lower spinal centres in cases of hemiparaplegia seems not to be constant. On the paralysed side it has in some cases been increased, in others it has been diminished; on the anæsthetic side it has generally appeared to be normal, but occasionally it has been exalted. Erb says that the bladder and rectum have generally been paralysed—always when there has been a sudden traumatic lesion—with either complete retention, or complete incontinence of urine and involuntary passage of fæces. This lends support to the view that the paralysis of these parts in cases of ordinary paraplegia, due to a bilateral lesion limited to the upper part of the cord, depends on an inhibitory influence transmitted downwards from the diseased parts to the healthy centres below. An analogous fact, which has now been noticed in several cases, is that the muscles on the paralysed side in hemiparaplegia sometimes have their faradic contractility lowered in a marked degree; they may also undergo rapid wasting.

Hemiparaplegia has been observed in patients who have been stabbed in the back by a knife or dagger. It seems strange that a chance wound should make a section of one half of the cord with the accuracy of an incision performed on a rabbit in the laboratory; but the shape and size of the spaces between the arches of the vertebræ seem to prevent a cutting instrument from passing across the median line within the spinal canal. Now and then a fracture or a dislocation of the spine has been attended with hemiparaplegia. A tumour outside the cord, compressing one half of it, has more than once caused paraplegia of one leg. In still fewer other cases, this rare form of paralysis has been due to a lesion limited to the interior of the cord on one side—an effusion of blood, a patch of sclerosis, or of acute focal myelitis, a tumour, or a gumma.

Some of these various unilateral lesions are more apt than others to affect a considerable length of the cord, and involve the roots of several spinal nerves. This leads to the development in some cases of hemiparaplegia of a fresh set of symptoms due to interference with nerve-roots. Thus there is commonly an anæsthetic half-zone of greater or less depth passing round the paralysed half of the body from back to front, and dividing the hyperæsthetic part of the surface below from the normal part above; and the upper edge of this anæsthetic space is said sometimes to present a narrow hyperæsthetic border. The explanation is obvious: the nerves of the region deprived of sensation have had their roots destroyed by the disease; those of the region which is oversensitive have merely had their roots irritated. The other (or anæsthetic) half of the body sometimes also has a narrow hyperæsthetic half-zone, limiting the anæsthesia above, and due to irritation of fibres which have just decussated at the upper edge of the lesion in the cord. In some cases the patient experiences a disagreeable sense of constriction, or severe burning or shooting pains, round the trunk, at a level corresponding with that of the nerves whose roots are involved in the disease, and such sensations may be limited to one half of the body or affect both sides alike.

Hemiparaplegia rarely remains stationary. Most commonly it soon undergoes conversion into ordinary paraplegia, as the result of myelitis spreading through the whole thickness of the cord around the original lesion. This often ends fatally, but occasionally, according to Erb, it subsides and allows the symptoms of a unilateral lesion to reappear. These may then persist for years, and in certain cases have been known to end in recovery.

SCLEROSIS OF THE CORD.*—We have hitherto spoken of inflammation of the cord as an acute or subacute process attended with softening of the nervous tissue. We have now to consider a form of so-called myelitis which has been generally regarded as a chronic interstitial inflammation. There are none of the symptoms of inflammation, no fever, and often no pain, but there is evidence of exudation of leucocytes and of gradual increase of fibrous tissue, which, like a cicatrix, compresses and at last destroys the ganglion cells and nerve-fibres which form the parenchyma or active peculiar elements of the central nervous system. It is certain, however, that in some cases the first step in the process is atrophy of the parenchyma, to which the changes in the neuroglia are secondary. The same question of which is the primary process has been again and again raised not only over the various forms of spinal and cerebral sclerosis, but also over the closely analogous chronic interstitial inflammations of the liver, kidney, lungs, and other organs. In all of these, beside the acute and obviously inflammatory processes which affect the parenchyma and soften the organ we find chronic fibrous inflammations leading to hardening and shrinking, the condition called either cirrhosis or sclerosis. The weight of authority has hitherto been in favour of Virchow's original description of this condition as true, though exceedingly chronic, interstitial inflammation.

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* *Synonyms.*—Chronic interstitial myelitis—Grey induration—Fibrous degeneration of the cord—Degeneration of the lower neurons.

Of late, however, improved methods of histological investigation, and particularly Golgi's plan of staining, have led many pathologists to regard the atrophic and destructive process of ganglion cells and nerve-fibres to be primary,—to be, in fact, a degeneration of the neuron, to which changes in the neuroglia are only secondary.

Sclerosis—*i. e.* hardening, a convenient term which does not beg the question—always begins insidiously, continues gradually, and never lasts less than two or three years. Instead of being soft, as in acute myelitis, the substance of the cord is obviously tougher than natural. Ollivier long ago compared it to boiled white of egg; the knife meets with resistance in cutting through it, and the exposed surface is smooth and even. On close scrutiny it often looks gelatinous, and has a greyish or greyish-yellow tint, instead of the milk-white colour of a healthy cord. It is rather shrunken than increased in size. When held up to the light the diseased parts are translucent. They are untouched by osmic acid, and when stained with carmine they take the colour much better than the unaffected white columns.

With the aid of the microscope the neuroglia is found to be thickened and fibrillated, or even, in old cases, to be converted into a dense mass of connective tissue with delicate parallel, wavy fibres. Cells with a large number of radiating processes (Deiters' cells) are often seen; it is said that similar cells may be discovered in the normal neuroglia, but they are at any rate much more numerous in cases of chronic myelitis. The nerve-fibres are atrophied, they have lost their medullary sheaths, and their axons are swollen, hard, and glistening. The ganglion cells (when the grey substance of the cord is affected) are shrivelled and granular, or may be converted into homogeneous, bright-looking, angular bodies without processes, and having apparently no nuclei. Corpora amylacea are generally abundant, and granule-masses are also present, often in large numbers, but sometimes very few. The smaller blood-vessels are thickened.

This process of sclerosis is not peculiar to cases of chronic paraplegia, but forms the anatomical lesion of several other affections of the spinal cord, which will be described separately, because their limitation to special tracts gives them an independent clinical course (*systemic* or *columnar* diseases).

In other cases of chronic paraplegia the sclerosis extends diffusely over the segmental area of the cord so as to prevent the transmission of motor impulses from the brain to the parts below. Its longitudinal extent varies greatly. Sometimes it is confined to a single spot, and has been called "chronic transverse or segmental myelitis;" sometimes it advances slowly along the entire length of the cord (generally from below upwards) until it reaches the bulb. These are the cases known as "creeping palsy," in which the lower extremities and pelvis are first affected, then the abdominal and intercostal muscles, then the arms, and lastly the phrenic nerves.

Spastic Paraplegia.*—Of the forms of sclerosis which are confined to certain physiological tracts of the cord and produce a connected series of symptoms as the result, we will first take that which is clinically known as spastic paraplegia or paralytic rigidity of the limbs. It is not so much an independent disease as a series of symptoms which appear in chronic paraplegia from whatever cause. We have already mentioned the rigidity of the legs which sometimes comes on as the result of Syringomyelia, and we shall meet with similar affections of the muscles in cases of Hemiplegia of cerebral origin.

As a complication of chronic Paraplegia—for it never appears in cases of acute transverse myelitis—these symptoms are very common and characteristic, most frequently occurring in cases of compression of the cord from vertebral caries and in cases of chronic transverse and diffused myelitis.

In these cases of chronic paraplegia it is only when the lowest lumbar centres are destroyed that the legs are flaccid. In paraplegia from angular curvature and in hysterical paraplegia they are sometimes spasmodically flexed; more often they are forcibly extended.

The rigidity is caused by tonic contraction of the muscles ("contracture"), and is associated with increased reflex actions, both superficial and deep, and with the clonic contractions known as ankle-clonus. They are no doubt due to increased reflex irritability of the spinal centres below the lesion, and this again may be referred to the absence of the normal inhibitory influences from the cerebral centres. The difficulty is to explain why their appearance is delayed.

The anatomical change which is found in cases of spastic paraplegia is a sclerosis which descends from the seat of disease along the crossed pyramidal or motor tract in the lateral columns of the cord. Cases of secondary rigidity and spasm of muscles which have long lost voluntary power are probably of the same pathology, whether paraplegic, hemiplegic, or monoplegic in distribution.

Beside the cases of spastic paraplegia secondary to compression or other transverse lesions of the cord, we shall hereafter meet with others in which similar symptoms occur as complications of Progressive Muscular atrophy (Amyotrophic lateral sclerosis) and of Locomotor Ataxia (Spasmodic tabes).

There are, lastly, very rare cases in which rigidity is present from the first, and, indeed, constitutes the most marked symptom, so that it may seem to be the only obstacle to the patient standing or walking. Here the lesion is in all probability a *primary* symmetrical sclerosis of both lateral columns. Evidence on this point was afforded by a case of Dr Morgan's, of Manchester, in which Dr Dreschfeld found sclerosis of the crossed pyramidal tracts and no other lesion, spinal or cerebral ('Transactions of the International Medical Congress of 1881;' and 'British Medical Journal,' 1881, vol. i, p. 407). Spastic paraplegia, whether secondary or primary, is therefore anatomically a "systemic" or "columnar" disease, the sclerotic process keeping to a definite tract of fibres.

Spastic paraplegia is said to occur chiefly in persons between thirty and fifty years of age, and in men more often than in women; but it follows the incidence of the primary lesions of which it is most often a result. The causes of primary cases are unknown. Poisoning by syphilis and by lead have been suspected, and also by *Lathyrus cicera* in three cases reported from Italy.

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* *Synonyms.*—Spastic spinal paralysis (Erb)—Tabes dorsalis spasmodica, in part (Charcot)—Primary and secondary lateral sclerosis—Spasmodic paralysis.

Symptoms.—The lower limbs assume as a rule a position of rigid extension and adduction. The contraction varies greatly in degree, and at first it is only occasionally present, generally when the patient stands upright. One of the earliest symptoms of the complaint is a peculiar gait. In walking the foot is lifted with difficulty; the toes are scraped along the floor and catch against every inequality in the surface; the step is short and hesitating, and sometimes there is a peculiar hopping movement, the body being raised upon the toes at each step as in equino-varus. Very often the attempt to walk causes a tremor in the foot and leg, which may extend to the trunk also,—a true clonus. In some cases the back is arched, and the head thrown backwards; in others the body is bent forwards over the toes by similar tonic contraction of the flexor muscles of the trunk, so that there is danger of falling, especially in descending stairs. The gait at one stage may be rolling or waddling, as carefully described by the late Dr Ross. The legs feel weak and heavy and are easily tired. Sometimes, if the patient sits down, his legs are thrust forward, so that his feet do not touch the ground, as if his knees were ankylosed or he had wooden legs. In more extreme cases he is confined to bed, and is perfectly helpless. His knees are tightly pressed together by spasm of the adductors, and cannot be bent from spasm of the quadriceps. Yet in uncomplicated cases there is no impairment of sensibility, pain is absent, and the bladder and rectum perform their functions naturally. The galvanic and faradic contractility of the muscles is slightly lowered, but there is no reaction of degeneration. In cases, however, which are secondary to myelitis or compression these negative characters are often wanting.

The "tendon-reflexes" are increased. Not only is the knee-jerk exaggerated, but movements can be excited through the tendons of the tibialis anticus and posticus, the biceps femoris, and even through aponeurotic structures. Ankle-clonus is present. There is usually increase of the superficial reflex movements, which are excited by cutaneous irritation.

The plantar reflex is not only exaggerated but altered. Instead of the natural flexion of the foot and toes, which makes the dorsal aspect convex and the plantar concave, the result of tickling is to cause what normally is only seen in young children—dorsal flexure of the foot with extension and spreading of the toes. This "infantile" form of the plantar reflex is probably due to a similar condition in the healthy child and in the adult affected with spastic paraplegia: in the latter the fibres in the lateral columns which connect the upper with the lower neurons have lost their myelin, in the former it has not yet been developed.*

This extension (or "dorsiflexion") of the great toe and of the whole foot, with divarication of the toes, is therefore an important evidence of lateral sclerosis, and helps to distinguish it from the exaggerated tendon reflex seen in some cases of hysterical paraplegia. This remarkable symptom was first noted by Babinsky, and is sometimes described under his name.

The *course* of spastic paraplegia is progressive, but very slow, occupying a period of eight, ten, or fifteen years; and sometimes it remains stationary for a long time. It may never extend above the hips, or it may at length affect the lumbar and abdominal muscles or the upper limbs. The abdomen then feels hard, and is separated from the lower part of the chest by a furrow. The fingers are, from time to time, or permanently, clenched within the palm; there is an extension of the wrist- and elbow-joints with pronation of the forearm; and the arms are fixed rigidly by the side of the trunk. One leg is often affected before the other, and the contraction may then spread to the arm of the same side before it involves the opposite leg. In rare cases the upper limbs are the first to be attacked. Occasionally, and most often at the latest period of the disease, the lower limbs are rigidly flexed, instead of rigidly extended.

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* The medullation of the crossed pyramidal tract, as first observed by Flechsig, only takes place about five months after birth.

Dr Savage has observed cases of spastic paraplegia going on to the development of general paralysis of the insane. The writer had a well-marked case of this sequence in a man under treatment in Philip Ward during the greater part of the year 1889.

Cases of spastic paralysis affecting the lower limbs, which end in paraplegia and general paralysis of the insane, also have been described in America by Putnam and Dane as occurring chiefly in women, and marked anatomically by sclerosis of the posterior columns and cerebellar tract as well as the lateral columns.

In *diagnosis* we must bear in mind as probable antecedents, caries of the spine and transverse myelitis, tabes, progressive muscular atrophy, cervical pachymeningitis and syringomyelia. Hysterical paraplegia is often spastic, but may be distinguished by the above character of its knee-jerk, and by subsidence of the spastic symptoms under chloroform (cf. *supra*, p. 642). A more difficult condition to distinguish is Insular sclerosis (cf. *infra*). Charcot tells us that in one of the cases with which he used to illustrate his lecture on "spasmodic tabes" scattered patches of sclerosis were found at the autopsy as high as the crura cerebri.

Spastic paralysis in children.—Dr Gee recorded in the thirteenth and sixteenth volumes of the 'St Bartholomew's Hospital Reports' a series of cases which he called "spastic paraplegia." They all occurred in children, and the complaint was either congenital or began in early infancy. There were eight cases, six girls and two boys. The legs were always affected, and sometimes the arms also, and the symptoms corresponded with Erb's and Charcot's descriptions. Dr Gee laid stress on the fact that handling the limbs increased the rigidity. Chloroform relaxed it in all cases but one; in that instance the muscles of one calf were a little wasted, after the disease had lasted eleven years. The adduction of the thighs caused the legs to be crossed in walking ("scissors gait").*

Seeligmüller, Erb, and Gowers have also described spastic paraplegia in children. In some of these cases it was of the variety described by Charcot as "sclérose latérale amyotrophique," in which muscular wasting is also present, and the anterior cornua as well as the lateral columns are affected; in others the symptoms were referred to a cerebral rather than a spinal lesion, and during the last few years we have had two such cases in Guy's Hospital, one in a boy of three, the other in a girl of seven, both probably congenital.

Rigidity of the limbs with clonus is characteristic of the "birth palsies" in children, which are, as a rule, monoplegic or hemiplegic, but sometimes paraplegic.

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* See also Dr Gee's paper on "Hereditary Infantile Spastic Paraplegia," published in the twenty-fifth volume of the same 'Reports' (Jan., 1890), in which he describes spastic paraplegia with some muscular atrophy of the arms in a father and two children.

General prognosis and treatment.—Spastic paraplegia is always a very chronic, and probably an incurable disease. Erb has reported several cases as much relieved by galvanic currents applied to the spine; but he also speaks favourably of the "cold water cure," and of the gaseous saline baths (*Soolbaden*) of Rehme and Nauheim, and he has found the nitrate of silver sometimes useful as an internal medicine. Charcot recommended the bromides. Strychnia is likely to do harm rather than good, and conium and belladonna have been given without any useful results, but in some cases to be presently mentioned, physostigma has been of great service. The late Dr Ross believed the galvanic current to be by far the most trustworthy remedy, but Gowers has found that faradism is harmful, and that galvanism has no influence in lessening the spasm or improving the strength. As he well puts it, in primary lateral sclerosis the chance of recovery and the danger to life are both small.

Clinical cases of spastic paraplegia.—In patients suffering from chronic paraplegia, the spasmodic symptoms above described are constantly met with; but we have had very few cases at Guy's Hospital which could be regarded as primary spastic paraplegia.

In eight or nine instances contraction of the lower limbs was apparently the primary disease; but the legs and thighs were drawn up in a state of flexure instead of being stretched out. Any attempt to straighten the limbs has generally caused great suffering; but in one case—that of a man aged thirty-three, under Wilks's care—the suspension of a weight of fourteen pounds to the right foot led to a marked diminution of the pain; the limb was bent, so that the heel almost touched the buttock, and it could not be put straight even when chloroform had been inhaled. In certain instances the extract of Calabar bean appeared to remove the rigidity altogether.

The following are the cases referred to:—1. The first case in which I observed this satisfactory result was that of a boy, aged six, who was admitted on August 19th, 1874. He had been well until three weeks previously. He lay on his right side, with his legs drawn up, and unable to move them. They could be forcibly extended, but then became rigid. The expression of his face was indicative of pain, and he had complained of pain in the back of the head and over the spine. Sensation was unimpaired, but the fæces and urine were often passed into the bed. He could close his hands, but with a very feeble grasp, and sometimes they were noticed to be stiff. The diagnosis was chronic spinal meningitis, and hydrarg. c. creta, iodide of potassium, bromide of potassium, and other medicines were given without any result up to the 8th of October. At that time he began to take the extract of physostigma in doses of one sixth of a grain, gradually increased to half a grain, three times daily. Improvement quickly set in; the limbs became less rigid, and he regained the power of moving them. On November 21st cod-liver oil and steel wine were substituted for the Calabar bean. On January 9th, 1875, he left the hospital, able to walk pretty well without help.

2. In 1876 I was asked to see a boy, aged fourteen, who was under Mr Cooper Forster's care, with contraction of the thighs and legs, which had been coming on for six months. He appeared to suffer intensely from pains in the affected limbs, and screamed when they were touched. The legs and thighs were much wasted. Remembering the former case, I prescribed the Calabar bean in doses at first moderate, but quickly increased. The symptoms at once began to subside. He was after a time transferred to my charge, and left the hospital cured.

3. On October 31st, 1876, a woman, aged forty-one, was admitted under my care with a spinal affection which had come on suddenly two months before. She first complained of pains in the leg-muscles and then of stiffness in the joints. This was followed by great weakness and wasting of the lower limbs; and then they slowly began to contract. She lay in bed with her legs strongly flexed; when they were forcibly straightened it gave her great pain. Faradic contractility was found to be much diminished, and she had partial

loss of sensation; she could not pass her water. She took at first half a grain of extract of physostigma three times daily; after a week this dose was doubled, and four days later it was trebled. By this time she could move her legs to some extent. The medicine was continued for a month, and then some tincture of iron was prescribed. By December 23rd she could straighten her legs perfectly well. In the month of February, 1877, she became able to stand and to walk without help, and on March 1st she was discharged cured.

4. In 1878 a sailor, aged twenty-eight, came into the Clinical Ward under my care on May 29th. Fourteen months previously he had been on a river in South America loading timber, and had repeatedly got wet through and had allowed his clothes to dry on him; he said he was well used to getting wet with salt water, but not with fresh water. Four or five days after he left off this work he one morning on waking found that his left leg was stiff and painful in the ham. As the rigidity gradually got worse he went into hospital at New Orleans, and after a time it passed off. He returned to work, but two months later, the weather having been bad, he again began to suffer from stiffness and pain in the left leg. Presently the right one also was attacked. They both became weak, and for six weeks before admission he could not walk without a stick. At times he had short attacks of convulsive twitchings in the left leg. I found the muscles of the thigh decidedly rigid on both sides, especially the left. After leaving him for a few days without treatment, I prescribed the extract of Calabar bean in doses of a quarter of a grain, afterwards increased to one grain. Improvement was not at first very striking, but at the end of thirty-four days there was hardly any rigidity left.

5. The only case that I know of in which the extract of physostigma produced any unpleasant effects is one recorded in vol. xviii of the 'Guy's Hosp. Rep.' A boy, aged ten, had been suffering for some months with paraplegia before rigidity of the legs set in. A quarter of a grain of the extract was ordered, and after a week it was increased to half a grain three times daily. By mistake he took a grain in one dose, and an hour afterwards he was blue in the face but perfectly conscious, perspiring profusely, a clear froth coming from the mouth, the pupils of natural size, the hands cold, numb, and almost powerless. He had an emetic, and in three hours he was as well as before.—C. H. F.

These cases belong to a different clinical type from those described above, and have possibly a different pathology. Contraction of the legs in a *flexed* condition, apart from primary disease of the joints, may supervene in the last stage of chronic diffused myelitis, of compression-paraplegia, of infantile atrophic paralysis, and even of primary lateral sclerosis itself. It may also occur in hysterical paraplegia; but the "cadaveric" position with extended hips, knees, and ankles and adducted feet is characteristic of the clinical type of Charcot and Erb.

A well-marked case of the common secondary form of spastic paraplegia was during four years under the writer's care in Guy's Hospital, and ended in recovery.

The patient, a boy of twelve, when first admitted in January, 1885, suffered from caries of the cervical and of the lower dorsal vertebræ, which afterwards produced both lumbar and psoas abscesses. There was no anæsthesia or serious disturbance of the bladder or rectum, or bedsores, or pain. The muscles were not atrophied except from want of use. When first admitted there was tonic contraction of both arms and both legs, the former flexed, with *main en griffe*, the latter extended in the cadaveric position. Knee-jerks and plantar reflexes were exaggerated, and ankle-clonus was readily elicited. Faradic contractility was unaffected. Physostigma was prescribed and pushed without benefit; but after many months, as the result apparently of improvement of the vertebral disease by surgical treatment, the power of his arms gradually became almost normal, and contraction disappeared; he could move his legs well, although clonus and exaggerated reflexes remained, and his general condition greatly improved. Finally he was discharged in 1889, able to walk and use all his limbs, free from spasm, and only showing the angular curvature of his spine, and the scars of healed abscesses.

Of uncomplicated and possibly primary spastic paraplegia the writer has only seen two cases. Both patients happened to be clergymen. The one, a remarkably healthy, well-developed man of about forty, complained of gradually increasing difficulty in walking, owing to "stiffness" of the legs. He could not bend his knees, and found it almost impossible to ascend a broad and shallow flight of stairs. When lying down his legs were rigidly extended. Ankle-clonus was present and the knee-jerks were increased. There was no pain except during occasional cramps or spasms in the legs. The pelvic organs were unaffected. There was no true paraplegia, for the lower limbs could be moved readily until the check of stiffness came, and the skin was normally sensitive. The muscles were of good size. Physostigma was prescribed, and he thought there was some improvement after several months, but if any it was slight. He afterwards tried massage and other plans of treatment, but the local disease went slowly on until his death by an acute attack of pneumonia.

The other case occurred in an older man—one who had been an Alpine climber and enjoyed robust health. In his case also the loss of locomotive power only resulted from stiffness of the legs. The knees were bent, the body was bowed, as if with the "chronic rheumatism" of an aged peasant, so that it was almost impossible for him to walk or to stand upright. The pelvic organs were unaffected; knee-jerks were readily produced and clonus also. As in the previous patient, the upper limbs were free. In this case also physostigma was fully tried with only doubtful benefit, but under repeated courses of nitrate of silver decided improvement took place, whether in consequence or not must be doubtful. At present (1899) he enjoys good health, but the spastic paraplegia remains.

In a well-known paper, read before the Royal Medical and Chirurgical Society in 1839, Dr William Budd recorded the case of a young lady in whom the slightest disturbance of the bedclothes caused violent contractions of the right leg, which was paralysed; but there was a successive diminution in the vigour of the spasms on each renewal of the stimulus. These reflex movements were attended with pain like that of cramp. As a rule, however, in cases of complete paraplegia with subsequent lateral sclerosis, the patient has no consciousness either of the application of the stimulus or of its effect, unless he sees the jerking of his legs. The susceptibility of the lower neurons seems to be directly augmented; for the administration of strychnia excites spasms in paralysed limbs much earlier than in those which remain obedient to the will. Violent convulsions are sometimes excited, not only by tickling the sole of the foot, but also by micturition or defæcation. One of Dr Budd's patients was obliged, whenever he was placed upon the night-stool, to have his feet inserted into two loops of saddlers' webbing which were nailed to the floor, while two larger loops were adjusted over his knees; otherwise he was liable to be thrown forwards upon the ground.

Affections of the Spinal Membranes.—A few words must be added concerning lesions of the meninges of the cord which often produce paraplegia. They are not segmental, but indiscriminate or diffuse in their localisation.

Spinal meningeal hæmorrhage.—Except as the result of severe injuries, which come under the care of the surgeon, this is a very rare lesion. It is supposed to be caused by violent bodily efforts, by convulsions, or by purpura and other general conditions. If a diagnosis is to be made during life, it must be based upon the sudden development of the symptoms, and upon the coincidence of symptoms of irritation of the cord and its nerves with those of compression. Certain cases marked by severe pains in the back and limbs, rigidity of the spine, and partial paraplegia have been attributed to the same cause when recovery has slowly taken place. But it would seem that when meningeal hæmorrhage has been demonstrated in the deadhouse, the symptoms have not been equally characteristic.

Tumours of the spinal meninges have been already mentioned in their clinical place as causes of paraplegia by compression of the cord (p. 630).

Acute spinal meningitis.—Inflammation of the vertebral dura mater (spinal pachymeningitis) is a frequent sign of caries of the vertebræ, and forms part of the anatomy of paraplegia from compression.

Secondary inflammation of the spinal membranes may result from the invasion of a deep bedsore over the sacrum. In some cases the vertebral canal is actually opened by sloughing of the fibrous membrane which closes its lower end; in others, perhaps, the morbid process extends inwards along the posterior sacral foramina. Several instances of this kind have been observed at Guy's Hospital, and probably others have been overlooked. Pus has generally been found among the nerves of the cauda equina, and beneath the spinal arachnoid, up to the base of the brain. No distinctive symptoms seem to have been noted in these cases.

Other forms of acute spinal meningitis accompany myelitis and traumatic, tuberculous, epidemic, and other forms of cerebral meningitis, which will be fully described hereafter. Hæmorrhagic spinal pachymeningitis is also occasionally found in association with a similar affection of the cerebral dura mater. Primary acute inflammation of the spinal membranes does not appear to exist.

Chronic spinal meningitis.—Certain local forms of meningitis, occurring chiefly in association with syphilitic gummata, or with caries of the vertebræ, have been already mentioned; but it sometimes happens that the whole of the spinal membranes are found matted together and thickened round a cord which appears to be perfectly healthy. In 1878 a case of this kind occurred in an old woman of seventy, who had for about a year been suffering from slowly advancing paraplegia. She had no spasms or rigidity of the legs; she lay helpless in her bed, but the lower limbs still retained some power of sensation, so that she could distinguish heat from cold. She complained of pains in the legs, apparently not severe, and in the lower part of the abdomen. After death the visceral arachnoid, when it had been stripped off the inner surface of the dura mater, was found to be as thick as a sheet of writing-paper. In the lower part of the dorsal region the adhesion of the membranes to the cord was unusually close for about an inch and a half of its length. The thickening extended upwards around the pons, and to the under surface of the cerebellum, so that the cerebrospinal aperture was closed. Dr Frederick Taylor, under whose care the patient was during life, could detect no morbid change in the cord itself except that the fibrous septa entering it from the surface appeared thicker than usual.

A similar case was recorded by Gull in his well-known paper in the 'Guy's Hospital Reports' for 1856 (Case 7). In that instance rigid flexion of the legs was a marked symptom, and they were also affected with frequent spasms. But this was probably a case of compression or myelitis in the upper dorsal region, with secondary descending lateral sclerosis.

Erb describes chronic diffused spinal meningitis as not infrequent, and often recognised during life. It is characterised, he says, at first by pain and stiffness in the back, a troublesome "girdle sensation," a feeling of weight in the legs, with numbness, tingling, or dragging pains, and cutaneous hyperæsthesia. After a time partial paraplegia develops itself, which varies in degree from day to day. There is generally but little impairment of sensibility; and, according to Braun, the sphincters retain their functions. The change in the membranes is usually but slight; they are more or less thickened, opaque, and adherent, with excess of fluid round the cord.

This form of paraplegia has been ascribed to cold, and it has often been observed in soldiers after severe fatigue and exposure during a campaign. It may begin abruptly, and afterwards pass into a chronic form. If complicated with myelitis, the peripheral part of the cord beneath the pia mater sometimes undergoes sclerosis in an annular form (cf. p. 623).

Erb says that many such cases prove fatal by bedsores, cystitis, or exhaustion, while others recover more or less completely; and Braun regards the prognosis as favourable if the early stage be treated with local bleeding and cold applications. Afterwards, when paralytic symptoms alone remain, the patient is recommended a course of hot brine baths, particularly those of Rehme or Nauheim, which closely resemble the saline waters of Droitwich. Erb endorses Braun's statements, and declares that when there is no myelitis, baths at high temperatures (98°—108° Fahr.) are well borne, and that the reputation of hot springs for curing paraplegia in general is really derived from their success in this particular class of cases.

Charcot first described a form of primary inflammation of the spinal dura mater, producing pain and partial paralysis in one or the other arm—*Pachymeningitis cervicalis hypertrophica*. The origin of the ulnar nerve seems most often to suffer; and, after paralysis with pain a[illegible] atrophy of the arms, there follows secondary spastic paraplegia of the le[illegible] We shall meet this affection again in the next chapter (p. 674).

PARALYSIS ATTENDED WITH MUSCULAR WASTING

> "Weak shoulders, overcome with burdening grief,
> And pithless arms, like to a withered vine,
> That droops his sapless branches to the ground."
> K. HENRY VI, part i.

General characters of columnar diseases of the cord—Special characters of atrophic paralysis.

ACUTE ATROPHIC SPINAL PARALYSIS—*In infants—its course, sequelæ, anatomy, and pathology—its prognosis and treatment—In adults.*

CHRONIC ATROPHIC SPINAL PARALYSIS—*Progressive muscular atrophy—Symptoms—Pathology—Primary and secondary forms—Spastic atrophic paralysis—Pachymeningitis cervicalis—Diagnosis—Treatment.*

Bulbar paralysis—Myasthenia gravis.

Primary progressive muscular atrophy in children—Hereditary juvenile form.

PSEUDO-HYPERTROPHIC PARALYSIS—*Primary (myopathic) muscular atrophy.*

THE paraplegic affections discussed in the last chapter, when their anatomical seat is ascertained, are found to be due to a lesion of the cord which is "indiscriminate." Whether it affects the whole organ, as diffuse myelitis, or one or more segments only, as transverse myelitis and paraplegia from compression, or the surface, as meningo-myelitis, or the deepest parts, as peri-ependymal myelitis—in all these varieties of disease, the cord is affected indiscriminately throughout the diseased segments.

The only exception was spastic paraplegia, where the lesion is in the most typical cases limited to the crossed pyramidal tracts of the cord.

There are several other spinal diseases which agree with spastic paraplegia in this particular: they affect particular tracts of the cord, and as a rule do not transgress these anatomical limits. Moreover they are chronic in their course; they are much less dangerous than acute myelitis or paraplegia from compression; and the anatomical process which causes their symptoms is probably the same—either degeneration of the ganglion cells or their atrophy as the result of chronic interstitial myelitis, *i. e.* grey degeneration or sclerosis. They are called by German pathologists "system-diseases," an uncouth and obscure phrase in English. "*Longitudinal* (as opposed to *transverse*) chronic myelitis" would express their range. Perhaps the term "columnar" sclerosis may serve to denote the characteristic limitation of the lesion to the longitudinal columns or tracts of the cord.*

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* The recognition of the physiological division of the white substance of the cord into distinct tracts of ascending and descending fibres is due to Türck, of Vienna, who in 1851 and 1853 described them as regions of secondary degeneration after lesions of the brain and cord. Flechsig subsequently showed that the same distinction is observable on development, the fibres of the anterior column and anterior root-zone acquiring their myelin first, those of Goll's tract and the direct cerebellar tract next, and the crossed pyramidal tracts last, not indeed until some weeks after birth. (See Dr Bramwell's excellent plates, 19—21.)

The longitudinal, columnar, or systemic scleroses which have been as yet observed are the following:

1. Of the lateral columns (crossed pyramidal tract) rarely primary, often secondary; descending from a primary lesion of the motor tract in the brain or cord. The corresponding disease is that described in the last chapter as Spastic Paraplegia.

2. Of the antero-median column of Türck. This is always secondary and descending. It does not correspond to any known clinical group of symptoms.

3. Of the posterior columns of Burdach (*fasciculi cuneati*). This is usually primary, and corresponds with the clinical malady which will be described in the following chapter as Tabes.

4. Of the postero-median columns of Goll. This is almost always a secondary lesion, often combined with the last in tabes, sometimes appearing as an ascending degeneration from a transverse lesion. No constant symptoms are known to answer to it.

5. Of the direct cerebellar tract. This is always secondary and ascending. It also does not give rise to any ascertained malady.

Secondary sclerosis of the "mixed tract" outside the anterior root-zone antero-lateral column) has been observed by Gowers and by Tooth, as an ascending degeneration after fracture of the spine. Its pathological significance is as yet unknown ('St Barth. Hosp. Reports,' vol. xxi).

6. Of the large motor and trophic ganglion cells of the anterior cornua. This is sometimes an acute, sometimes a chronic process, and is often confined to a single segment or to some part of the anterior cornu in a single segment.

The cells which form the posterior vesicular column of Lockhart Clarke appear also to be subject to a destructive process which affects them alone.

In the present chapter we deal with clinical affections for the most part identified with disease of the anterior cornua. But they are also united by a common symptom, that of marked and rapid atrophy of the paralysed muscles, as well as negatively by the absence of anæsthesia, trophic changes in the skin, pelvic symptoms, ocular symptoms, or mental disturbance. They may be conveniently dealt with together as Atrophic Paralyses.

Pathologically these forms of spinal paralysis differ from the various kinds of paraplegia described in the last chapter, in that the paralysis is the result not of severance between the upper neuron of the brain and the lower neuron of the cord, but of destruction of the latter, without implication of the sensory tract. Hence the motor paralysis unaccompanied by anæsthesia, hence the paralysis of reflex as well as voluntary movement, and hence the trophic symptoms.

Some cases, however, of atrophic paralysis depend on lesions of the nerve-trunks, and have been described in a previous chapter (p. 593). Others begin in the muscles themselves, but these are so closely connected with those of spinal origin that at present it is most convenient to discuss them with those which own an undoubtedly central origin in the anterior cornua of the cord.

Muscular atrophy.—Wasting of the muscles may be the result of many causes which it is well to remember in distinction from that due to the direct effect of local lesions.

It is constantly observed to follow starvation, as from cancer of the œsophagus, and to accompany the general emaciation produced by diarrhœa, by specific fevers, phthisis, and pyrexia in general. Here muscular atrophy accompanies wasting of the fat, and to a less extent of the viscera; while the brain and cord, the bones, and the heart almost entirely escape. Anæmia, even when extreme, is not accompanied by muscular (or adipose) atrophy, as is seen in cases of chlorosis and of Addison's idiopathic (so-called pernicious) anæmia.

Atrophy of the muscles occurs from disuse in bedridden persons, in healthy men with a broken limb, and in the course of chronic affections of the joints, such as osteo-arthritis and gout. The hollowing of the interosseous spaces and of the ball of the thumb may be very striking in cases of this kind.

French writers describe under the name of *marasme essentiel*, an atrophic affection which they say occurs in hypochondriacal patients, who gradually assume the appearance of living skeletons. It corresponds with the condition described by Gull as *anorexia nervosa*, and with such remarkable cases of emaciation as that in a man who was cured by Weir Mitchell's treatment, published by Playfair and Brunton.

Muscular atrophy, again, results from the disuse of paralysed limbs, as in cases of hemiplegia.

These atrophies, however, are general in distribution, slow in progress, and usually moderate in degree. They are quite distinct from the rapid, excessive, and discriminating atrophies which are the subject of this chapter.

The following is the anatomical arrangement of the atrophic paralyses to be described. As Gull wrote in 1862, "Progressive," or perhaps better, "excessive" muscular atrophy may arise from primary lesion of the muscular elements, or from lesion of the trunks or branches of the nerves, or from morbid changes in the grey matter of the cord.

i. Having the primary seat in the cerebral cortex. Certain cases of birth-palsy from meningeal hæmorrhage in which atrophic as well as spastic symptoms develop.
ii. In the motor anterior cornua. Acute and chronic poliomyelitis, anterior cornual myelitis, atrophy of the motor cells of the cord.
iii. In the anterior roots—Pachymeningitis cervicalis.
iv. In the peripheral motor nerves—Multiple neuritis. Plumbic, alcoholic, and other toxic paralyses.
v. In the muscles—Myopathic atrophy.

The following are the clinical forms of atrophic spinal paralysis according to the order adopted in the present chapter:

1. Infantile paralysis, essential paralysis of children, acute atrophic spinal paralysis.
1*a*. The same condition in adults.
1*b*. A subacute form of the same (?).
2. Progressive muscular atrophy of Aran and Cruveilhier, chronic atrophic spinal paralysis.
2*a*. Bulbar palsy—the same condition affecting certain cranial nerves.
3. Pseudo-hypertrophic paralysis.
3*a*. Other forms of primary muscular atrophy.
4. Secondary or complex forms. Muscular atrophy enters into the series of symptoms observed in certain other nervous diseases beside those just enumerated.

Amyotrophic lateral sclerosis, Pachymeningitis cervicalis.
Syringomyelia and gliomatous growths affecting the cervical cord.

The following are the chief points of distinction from a clinical point of view between the various groups of atrophic paralysis which are at present recognised:

1. The age and the sex of the patient.
2. Hereditary or Family distribution.
3. Localisation.
    - α. Chiefly in the hands and shoulders—the face and the *triceps extensor brachii* exempt.
    - β. Trunk and limbs, particularly calves and buttocks, beginning in the face.
    - γ. Peronei.
    - δ. Upper limbs—*supinator longus* exempt.
    - ε. Lower limbs.
4. Complication with clonus, contractures, or other spastic symptoms preceding or following the atrophy or affecting the lower limbs while the upper are atrophied.
5. Presence of pain.
6. Electrical reactions.
7. Rapid, subacute, or chronic and ingravescent course.

ACUTE ATROPHIC PARALYSIS.[*]—In the last century this affection was described by Underwood in his treatise on the 'Diseases of Children' (1784). Jacob von Heine published a monograph on it in 1840, and placed (as did afterwards Duchenne) the cause of infantile paralysis in a lesion of the spinal cord. When, however, autopsies failed to reveal any morbid change, MM. Rilliet and Barthez proposed the name of "paralysie essentielle;" but since 1863 better histological methods have shown that the anterior cornua of the cord are diseased. Accordingly the affection is now known as "poliomyelitis[†] anterior acuta." The old name "infantile paralysis" is not wide enough, for it is now ascertained that the same disease occasionally occurs in adults.

Onset and course.—A child who is about to suffer from acute atrophic paralysis is sometimes apparently well until the loss of power appears. But more often he falls ill with febrile disturbance or drowsiness. Sometimes the earliest symptom is an epileptiform convulsion, or spasmodic twitchings of the face or limbs, grinding of the teeth, and rolling of the eyes. Pyrexia, when present, usually lasts a day or two; but it may pass off in a few hours, or continue for a week or more. Convulsions may be repeated during twenty-four or forty-eight hours.

When the child seems to be getting better, and is being washed or dressed—or perhaps when it first attempts to stand,—the mother or nurse finds that one or more of the limbs is powerless. He has gone to sleep at night with full use of his arms and legs, and in the morning a limb may hang flaccid and motionless. The affection may be a monoplegia, the paralysed limb being most often the right leg,* but sometimes an arm; or it may be a paraplegia, or a hemiplegia, or a crossed paralysis of one arm and the opposite leg. In rare cases there is loss of power of the trunk or of all four limbs, or they may be attacked in succession at intervals of a few hours.

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[*] *Synonyms.*—Acute amyotrophic spinal paralysis—Poliomyelitis anterior acutissima (Kussmaul)—Anterior cornual myelitis (Gowers)—Infantile paralysis—Wasting palsy of children.

[†] *I. e.* myelitis affecting the *grey* matter (πολιός). Charcot proposed Tephromyelitis (τεφρός, ash-coloured, cinereous); and Vulpian, Spodiomyelitis (σπόδιος, also ash-coloured, *like* the last, but an epithet only used in poetry).

Occasionally the initial fever is very slight; it may perhaps be absent, and the paralysis be the first symptom that the child is unwell. But, as might be anticipated, there is no thermometric evidence of this.

It is characteristic of acute atrophic paralysis that the affected parts are completely relaxed. No reflex movements can be elicited, whether by stimulation of the skin or of the tendons. On testing the muscles with faradic currents, one finds that their contractility becomes markedly diminished within the first four or five days, and is entirely extinguished at the end of a week or a fortnight; but that the susceptibility to galvanic currents is increased, with the other signs of the reaction of degeneration (p. 567). Rapid and early wasting of the muscles takes place, and the histological changes correspond exactly with those described as following lesions of the nerve-trunks. The surface of the paralysed limbs is cold, and they are pale or livid. Volkmann found the volume of their pulse to be diminished, and Charcot states that after death the main blood-vessels are remarkably reduced in size.

No impairment of sensation can be detected: the child cries lustily as soon as the poles of a galvanic battery are applied. The functions of the bladder and rectum are unaffected, and bedsores do not appear.

This form of paralysis shows no tendency to gradual increase. The limbs are often attacked one after another, but the loss of power in each, although not absolutely sudden, becomes complete within a few hours. Erb says that the whole development of the disease, in successive outbreaks, may be protracted over a week; but it generally occupies much less time.

Before long recovery begins to take place; sometimes within a few days, more often in the course of two or three weeks. Muscles which have begun to waste regain their size and strength. If the arms and the upper part of the trunk are affected they are more likely to recover quickly than if the legs are paralysed. In the course of a few months the disease may entirely disappear, especially when it is confined to a single limb. It would be arbitrary to separate such "temporary paralysis" cases from those in which there is permanent loss of power; they are both exceptional. As a rule the recovery is incomplete; it goes on for a month or eight weeks, so that the parents of the child are confident of complete recovery; but after this the progress becomes slower, and at the end of six or nine months it ceases entirely.

The condition of the patient at this time is very variable. One limb or two may be powerless and shrunken throughout, or the affection may have become limited to certain parts of an arm or of a leg. As a rule the distal parts are more apt to suffer permanently than those nearer the trunk. Volkmann, however, speaks of one patient as having a muscular forearm attached to a humerus like a stick; and Erb says that the deltoid sometimes suffers alone, or its acromial part may escape, while the rest wastes away. Certain muscles exhibit a peculiar independence, some by remaining paralysed when those near them get well, some by recovering while the others are undergoing atrophy. The following usually escape: the *supinator longus* of the forearm, and the *tensor fasciæ latæ* and *sartorius* of the thigh; while the *deltoid* and *serratus magnus*, the *quadriceps extensor, femoris, gastrocnemius,* and *peronæi* are apt to suffer; or the last-mentioned may suffer alone, and the extensors in front of the leg escape.[*] When a muscle wastes, the destruction of its fibres does not always lead to a corresponding diminution of its mass, for interstitial development of adipose tissue sometimes may entirely conceal it.

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* Gowers says that the limbs on the left side of the body are more often permanently affected than those on the right. The two statements are not formally contradictory. But any preponderance on one side or the other is small, and like similar slight preponderance in frequency of right or left hemiplegia or pneumonia or ovarian tumour, has no practical or scientific interest (cf. p. 665).

Apart from the muscles, the entire limbs affected with this disease do not grow at the natural rate. Hence a paralysed leg is sometimes shortened by an inch or an inch and a half, and occasionally by as much as eight inches. Even the corresponding half of the pelvis may remain undeveloped. The bones are not only shorter, but thinner and more spongy, and their processes smaller than on the healthy side.[†] This is not from disuse, for it occurs when the paralysis is so partial that the child halts very little and is on its legs all day.

But the most important remote effects of acute atrophic paralysis in children are the contractions and deformities which are so commonly observed. Thirty years ago Wilks maintained that club-foot, when not congenital, is the result of paralysis, and is not primarily a spasmodic affection; and this view, which was then opposed by "orthopædic" surgeons, is now generally accepted. The contraction is not the result of the unopposed action of the antagonists of the muscles paralysed; for Hüter and Volkmann have shown that when there is loss of power in several groups of muscles, it not infrequently happens that those on which the paralysis is most marked lie *within* the open angle formed by the displacement of the bones at a joint. For example, a *genu recurvatum* with the concavity directed forwards may appear when the extensor muscle of the knee is powerless, or a *talipes equino-varus* when no contractions can be obtained by galvanising the calf of the leg. Volkmann explains these facts by showing that they are in part due to the influence of gravitation; thus the weight of the foot causes the toe to point downward when the limb hangs in the air. But another important factor is the gradual strain on a joint in efforts made by the patient to employ the weight of the body as a propelling force. Volkmann has noticed that when a person whose thigh-muscles are paralysed learns to walk without crutches, he swings the trunk forwards so as to extend the knee as fully as possible. The result is that after a time the ligaments behind the joint yield, and the knee becomes bent the wrong way. A subordinate element in the causation of deformities is the contraction of the new connective tissue developed in the interstices of the wasted muscles, a result which has been compared to "sclerosis" of the cord and "cirrhosis" of the liver.

Distribution.—Putting together sixty-two cases collected by M. Duchenne, fils, thirty-two of the late Dr Charles West's patients, and sixteen of Dr Goodhart's, we find that of the total 110 patients, the right leg only was paralysed in thirty-six (a third), the left only in twelve, both legs in nineteen (paraplegia), one or other arm in seventeen (making sixty-five cases of monoplegia, or more than half); hemiplegia occurred in twelve (right five, left five, crossed two), facial palsy in seven (all observed by Dr West); while in five cases either three of the limbs, or all four, or the muscles of the trunk were paralysed.

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[*] On this curious distribution of paralysis, seen also in the chronic form of atrophic paralysis and in lead palsy, light is thrown by the interesting experimental evidence, obtained by Professors Ferrier and Yeo, on the motor roots of the nerves of the extremities in the monkey ('Proc. Royal Soc.,' March, 1881).

[†] Remembering Hilton's observations with regard to the arrest of growth in the feet of patients suffering from disuse of the hip or knee-joint, one might be disposed to refer all these conditions to disuse; but this is certainly not the case.—C. H. F.

The much more numerous cases collected by Dr Allen Starr, of New York ('Allbutt's System,' vol. vii, p. 191), although they include Duchenne's cases of twenty-five affecting the right to only seven affecting the left leg, yet show the fallacy of small numbers, for in 595 cases exactly the same number (123) affected the right and left leg, 170 affected both, 53 one or both arms, and 47 all four limbs.

Morbid anatomy.—After death the paralysed muscles are found much diminished in size; they have undergone granular degeneration, the fibres shrinking and the contents of the sarcolemma losing their striated appearance and becoming pigmented and amorphous. There is sometimes fatty overgrowth between the fibres, but more often atrophy of the adipose tissue: and there seems to be never true fatty degeneration of the muscle.

Cornil, in 1863, was the first to record an autopsy in which changes were found in the cord; but the body was that of a woman who had been paralysed when a child, nearly half a century before. Royer and Damaschino examined the cord in the case of a boy who died of scarlet fever within two months from the onset of the paralysis, and Dr Charlewood Turner described the minute anatomy of the cord in a child under three years old who died from an intercurrent attack of measles six weeks after the paralysis appeared in the legs ('Path. Trans.,' xxx, p. 202, 1879).* The results of this, and of a few other observations made within two years of the attack, may be summed up as follows:—To the naked eye there is generally no perceptible alteration. Sometimes in the cervical or lumbar enlargement, and on the right or left side, as the case may be, the anterior of the grey matter may be pink and softened, or reduced in size, and the corresponding motor nerve-roots may be grey horn, translucent and thin. The microscope always reveals an extensive area of disease, perhaps an inch or more in length, in the affected region. Granule-masses (Gluge's corpuscles) are generally present in abundance; there is nuclear overgrowth, the vessels are dilated, and there is abundant exudation of leucocytes—in other words, there is an inflammatory softening. But the most striking change is the disappearance of many large multipolar nerve-cells, while those that still remain are in various stages of atrophy. At the periphery of the affected area the exudation-cells and nuclei are often massed together in large numbers, so as to form a kind of capsule. Slight diffused lesions are discoverable in the grey matter of a large part of the cord, sometimes in the whole length of the dorsal region. In the anterior and lateral white columns there is little change to be seen, but the anterior nerve-roots display all the appearances of degenerative atrophy.

When many years have elapsed since the onset of the paralysis the appearances are more marked to the naked eye. The shrinking and atrophy of the anterior part of the cord is now evident on the cut surface, and when the affection is one-sided the symmetry of the two halves is lost. The anterior cornu is less deeply coloured by carmine, because the protoplasm and nuclei of the motor cells are gone, but the adjacent white substance is more so, because the myelin has also gone, and Deiters's cells and fibrous tissue stain better than normal nerve-fibres. Under the microscope the diseased area in the lumbar or the cervical enlargement is seen to consist almost entirely of a delicate connective tissue, containing an immense number of corpora amylacea, but now no granule-masses. No ganglion cells or nerve-fibres are at this stage to be seen; any remnants of cells are shrunken and pigmented. The anterior cornu often contains the small branched spider-cells of Deiters. The anterior and lateral columns may be obviously grey in tint, and translucent as compared with the posterior columns.

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* See also Dr Money's report of a case sixteen weeks after paralysis (*ibidem*, vol. xxxv, p. 46).

Pathology.—That a local affection of the spinal grey matter should set up pyrexia would be strange. There may be a question whether the myelitis is not at first diffused, and afterwards clears up and subsides throughout the greater part of the cord, while one particular region, to which it becomes limited, undergoes entire destruction. But more probably the febrile symptoms point to a primary acute infection which has its local lesion in the anterior cornua.

It has been much debated whether the starting-point of the lesion is in the multipolar cells or in the neuroglia of the anterior cornua. The same question applies to sclerosis of the cord generally as well as to the origin of cirrhosis of the liver and granular contraction of the kidney. The nutrition of the grey matter is always difficult, particularly, as Moxon showed, in the lumbar enlargement, from the great length and narrowness of the arteries which supply it; and an inflammatory exudation would compress them, produce coagulation, and by starvation and pressure destroy the large motor ganglion cells. But, as Charcot argued, the fact of its strict limitation to certain regions of the cord is strongly in favour of the former view; he also says that at some points the cells may be found diseased while the connective tissue remains all but normal. On the whole it is most likely that the process begins in a destructive change in the motor cells of the anterior cornua.

The cases in which the access of paralysis is not rapid, but sudden, and unaccompanied by febrile symptoms, can scarcely have the same pathology, and seem very likely due, as Gowers believes, to hæmorrhage.

Ætiology.—With regard to the causes of acute atrophic paralysis very little is as yet known. It is far more common in children between one and three years of age than in those who are older, but it is often seen in infants of from six to twelve months. Duchenne has recorded cases occurring twelve days and one month after birth, and the disorder may befall any age up to puberty and beyond, so that it is not separated by any absolute line of age from the atrophic spinal paralysis of adults.

The two sexes appear to be equally liable to infantile palsy. Whether teething plays any part in its ætiology is very doubtful. Wharton Sinkler, an American physician, met with it much more often in the summer months than during the rest of the year, the proportion being forty-seven cases to ten; and this is confirmed by Ross, Gowers, Starr, and other observers. In July and August Dr Sinkler recorded seventy-seven cases out of 149, and in the same months Dr W. H. Barlow, of Manchester, writing in 1878, forty-eight out of 111. The supposed general neuropathic family tendency seems not to be present; Duchenne never saw the disease in two children in the same family; and the few cases that have been published are probably mere coincidences. Those who are attacked are, as a rule, neither rachitic, nor scrofulous, nor syphilitic, and often robust and healthy. Sometimes infantile palsy is set up by a chill, at least Erb says that this has been demonstrated beyond dispute. Occasionally it arises during convalescence from some acute disease, such as scarlet fever, modified smallpox, or typhoid fever. The view of its pathology now most generally taken is that it depends on an acute infective disease.

The *diagnosis* is seldom difficult, if one keeps in mind the clinical history and features of the disease. One must remember that other forms of paralysis may occur in childhood; perhaps that which is most likely to cause a mistake is a peripheral paralysis of the brachial plexus from sleeping on the side, or from pressure from a band fastened tightly round the arm. A similar temporary paralysis of one arm may be caused by accidental stretching of the brachial plexus. In the case of an infant brought by a person ignorant of the history, it may be impossible to come to a positive conclusion at once.

Prognosis.—So far as is known, anterior cornual myelitis is never fatal, whether in a child or in an adult; but complete recovery is rare. When a child first comes under observation at an advanced stage, with the muscles already wasted, one is apt to think that the issue would have been better had treatment begun earlier; but even if it is placed under treatment from the commencement there is great difficulty in saying how far any improvement that occurs is due to the measures adopted.

Treatment.—In the early stage, next to keeping the palsied limbs warm and promoting their circulation, rest in the prone position, with cooling drinks and sponging, if fever is present, seem to be rational measures. Bromides or chloral may be given as sedatives. Only when signs of improvement appear should electricity be applied. Faradisation may be used if the muscles have not lost their reaction to induced currents, but interrupted galvanism is alone efficient in most cases. Even when several years have elapsed one must never hastily conclude that this treatment will be fruitless. It now and then happens that a few applications produce extraordinary effects; and often, by persevering for several weeks or even for months, one at length attains considerable success.

In one case of Dr Fagge's the internal administration of strychnia was, he felt sure, the means of restoring to a child the power of standing. Shampooing and friction with stimulating liniments may sometimes be of service. Dr Angel Money thought that massage well carried out is far more useful than electricity. Nourishing food and cod-liver oil are matters of course. For the avoidance of the different forms of contracture recourse must be had to mechanical apparatus. Volkmann says that drawing up of the limb may be prevented by fastening the feet every night on a splint with a flannel roller, and carrying a strip of plaster across to the leg. The later results fall under the surgeon, and are often wonderfully benefited by tenotomy and suitable apparatus.

Acute atrophic paralysis in an adult develops itself in most respects as in infants. Moritz Meyer, of Berlin, is said to have been the first who, in 1868, recorded two cases of this kind after measles. Duchenne, Erb, and many others have since written upon the subject. The disease seems never to set in with epileptiform convulsions; but there is more or less fever, with headache, drowsiness, and occasionally delirium. After a single night, or at the end of a few days, one or more of the limbs become paralysed, and sometimes there is a transitory failure of the bladder. There is not the slightest impairment of sensibility, the muscles are perfectly relaxed, and their susceptibility to reflex stimuli is lost. Their electrical reactions are like those of the same disease in children. The ætiology is equally unknown, but it is much more rare in women than in men. Recovery may be either complete or partial; in the latter case the affected parts become contracted and deformed, but not to the same extent as at an early period of life, and of course there cannot afterwards be any difference in the length of the limbs on the two sides of the body.

In the only marked case which has come under the writer's care, the patient, a healthy railway servant, aged about twenty-five, gradually but completely recovered from his atrophic paraplegia. Interrupted galvanism was used, and nux vomica taken.

PROGRESSIVE MUSCULAR ATROPHY.*—In 1850 a French physician, Aran described under the name of "*atrophie musculaire progressive*," an affection which he believed had been overlooked by previous writers. Duchenne however, had published a case in 1849, and instances had previously been recorded by Sir Charles Bell, Abercrombie, and Romberg. Three years later Cruveilhier, in 1853, published his classical case of the athlete Lecomte, whose arms wasted muscle by muscle, then his legs, and finally his intercostal muscles, until he died from want of power to breathe. The preparation from this case is preserved in the Musée Dupuytren. It was Cruveilhier who showed that the muscular atrophy is not primary, but secondary to wasting of the anterior roots of the spinal nerves; and Lockhart Clarke who, in 1867, showed that this again depends upon a primary degeneration of the anterior cornua of the corresponding segments of the cord. The late Sir Wm. Roberts published a monograph on this disease in 1858. Next to infantile paralysis it is the most common of the atrophic palsies described in this chapter, but even this is comparatively rare.

Distribution.—In the large majority of instances the upper limbs are first attacked. Aran found that this was the case in nine among eleven patients, and that in seven of them the right arm was that in which the disease began. The proportion cannot be fixed with accuracy, because it is doubtful whether all the cases in which the wasting is first observed in the legs really belong to the same category (cf. *infra*, p. 686); while Duchenne met with only two such cases out of 159, Hammond, of New York, saw eight in a total of twenty-five. Duchenne himself saw one patient in whom the disease began in the *sacro-lumbales*, and one in whom the *pectorales*, the *trapezii*, and *latissimi dorsi* were wasted before the arms became involved. The apparently capricious way in which the atrophy is sometimes distributed is shown by another of this writer's cases, in which, even at an advanced stage, no muscles of the upper limbs except the *supinatores longi* were attacked. Trousseau mentions a patient of Bretonneau's, an old lady, who retained power over none of her muscles save those of the right index finger; she could not speak, but with her finger she picked out of a heap of letters those which she required to form words and sentences, and in this way she made her will.

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* *Synonyms.*—Cruveilhier's paralysis—Atrophie musculaire progressive: type Duchenne Aran—Wasting palsy—Amyotrophie essentielle, or protopathique of Charcot (as distinguished from amyotrophic lateral sclerosis, p. 673)—Poliomyelitis anterior progressiv longissima—Chronic spinal (as distinguished from myopathic) muscular atrophy.

As a rule progressive muscular atrophy begins in the small muscles of the hand, and most often in the ball of the thumb; before passing to the forearm it usually spreads from the thenar to the hypothenar muscles, and also to the *lumbricales* and *interossei.* Sometimes it appears first in the *deltoid* and other scapular muscles. It is remarkable that when the disease attacks the *trapezius* it almost always leaves the upper part of the muscle uninjured; and that the *triceps* usually escapes, even when all the other muscles in the arm have wasted away. The levator scapulæ and the platysma are also seldom affected.

The patient's arm at last hangs helplessly by his side; he cannot raise it by any muscular effort, and can only swing it forward with a jerk. His hand often presents the "griffin" or "bird-claw" deformity (p. 569) from paralysis of the *interossei.* His power of prehension is greatly diminished, especially when the *opponens pollicis* is atrophied. Instead of being able to grasp an object between the fingers and the thumb, he has to fix it as well as he can within the hook-like concavity formed by the fingers alone.

Whatever may be the starting-point of the disease, it soon shows itself in the corresponding muscles of the other side. But the symmetry of its distribution is often incomplete; thus the right forearm and the left upper arm may be most affected. Having spread more or less generally over the upper limbs, it at length passes to the neck and to the trunk; but the lower limbs usually remain intact until a late period. Only in exceptional cases does the atrophy begin in the legs, and the face is seldom or never affected.

Symptoms.—The loss of substance in the muscles is in most cases visible through the integuments, for not only do the muscles waste, but also the adipose tissue, as it does in infantile palsy, and the skin itself becomes thin. The ball of the thumb is flat, and there is an obvious hollowing of the metacarpal spaces, especially of that which lies between the thumb and the forefinger; or the shoulder has lost its fulness and the bones form prominent angles; or the rounded curves of the forearm are replaced by shallow depressions. Sometimes, however, there is an actual new growth of adipose tissue in the interstices of the affected muscles, as again in some cases of infantile paralysis; and since at the same time a tough fibrous material is present, the part may feel as firm and fleshy as in the natural state. The application of a faradic current will clear up all doubt, if the disease is sufficiently advanced; or a minute portion of muscular tissue may be removed for microscopical examination by Middeldorpff's "harpoon," or by Duchenne's "emporte-pièce."

The weakness in the affected muscles is increased by fatigue, and to a remarkable extent by cold. Some patients tell us that when warm in bed after a night's rest, their hands felt as strong as ever; but during the day the hands and fingers often look pale and blue, and they are cold to the touch. Jaccoud and others have taken thermometric observations, from which it appears that the temperature is notably lower on the side on which the muscles are the more wasted; it is said that a difference can sometimes be detected even between the two axillæ. Sometimes there is an excessive secretion of sweat. All these symptoms are referred by Leyden to disturbance of the vaso-motor nerves. In several instances a contracted state of the pupils is recorded as having been noticed.

A symptom of importance is the occurrence of transient "fibrillary tremors," or slight quivering movements limited to particular fasciculi of the muscles involved in the disease. They may either be spontaneous, occurring when the part is perfectly at rest, or one may elicit them by giving the skin a tap with the finger. They are far from being pathognomonic, and may be seen in healthy persons if carefully looked for; those which follow taps upon the surface of the muscle are particularly well marked in patients who are emaciated by phthisis or any wasting disease. But in progressive muscular atrophy the spontaneous fibrillary contractions far exceed those which occur under other conditions, so as to produce perceptible movements of the limbs and to attract the notice of the patient, although they are attended with no pain. It is said, too, that they sometimes afford the earliest indication of the extension of the disease to a fresh group of muscles. Duchenne found fibrillary contractions present in about four fifths of the cases that came under his observation.

The cutaneous sensibility is intact;* there is usually no pain in the affected parts, nor are sensations of numbness experienced. If pains occur at all, it is in the early stages of the disease.

Course and event.—It is not in every case that progressive muscular atrophy spreads from one group of muscles to another. On the contrary, it may remain for many years limited to those muscles which are first attacked. But, as we have seen, it often extends from the limbs to the trunk; and then it may probably destroy life by interfering with the respiratory movements. If the intercostal muscles fail to contract, the upper two thirds of the chest cease to expand, and the act of expiration is proportionately shortened: if the diaphragm is atrophied, the epigastric and hypochondriac regions are drawn in during inspiration. In either case the patient has frequently to stop between his words to take breath, he has difficulty in crying out or singing, and the attempt to cough or sneeze causes him great distress. An attack of bronchitis is often fatal. Not infrequently the intercostal muscles and the diaphragm fail simultaneously; and then, as the disease advances, suffocation becomes inevitable. Sometimes, on the other hand, the disease proves fatal by spreading to the muscles of the throat and of the pharynx; the symptoms which are then observed will be presently described under the head of "Progressive Bulbar Paralysis." In not a few instances death occurs from phthisis or from an intercurrent attack of enteric fever or pneumonia. The duration of progressive muscular atrophy varies from two or three to as many as twenty years.

Ætiology.—The causes of progressive muscular atrophy are quite unknown. It occurs chiefly in young adults; seldom or never, at least in its typical form, in children (cf. *infra*, p. 685). It is rare under twenty and over fifty, but Gowers has met with it as early as fourteen and as late as seventy. It is far more common in men than in women. Friedreich found only thirty-three women among 209 patients, and Sir William Roberts only fifteen among ninety-nine.

Charcot and many other writers taught that the disease is frequently transmitted by inheritance; but the majority of the supposed examples are really cases of primary muscular atrophy, not of chronic anterior myelitis.

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* This, at least, is what Charcot says of that form of the disease which he terms "protopathic," and which alone is now under consideration. Other writers, while admitting that impairment is slight in comparison with the muscular wasting, have yet described partial anæsthesia as of rather frequent occurrence. But they probably included amyotrophic sclerosis and other affections which Charcot has since shown to be both clinically and pathologically distinct, and which will be mentioned further on in this chapter.

Over-use of the muscles, leading to exhaustion of the corresponding spinal centres, has been regarded as the cause of the disease when it occurs in a bank clerk, in a cobbler, in a saddler, or in a washerwoman. But here, again, confusion may have arisen from the admixture of cases of "writer's cramp" under the vague designation of "scrivener's palsy." Progressive muscular atrophy often occurs in persons who have in no way over-fatigued their muscles, and the relation between the supposed cause and its effect is probably that of mere coincidence. It is difficult to suppose that such common and vague antecedents as exposure to cold, overwork, and mental distress or anxiety can have any causal relation to such a definite lesion of the motor tract. A certain number of cases follow closely upon measles and other acute diseases, and others again after a longer interval upon syphilis.

Pathology and histology.—The true nature of this affection has only gradually been cleared up. Both Aran and Cruveilhier originally supposed that it was a primary atrophy of the muscles, but the latter altered his opinion when in 1853 he found, in the celebrated case of the rope-dancer Lecomte, that the anterior roots of the spinal nerves were grey and atrophied. The central origin of the disease in the destruction of the large motor ganglion cells of the anterior cornu of the cord was demonstrated by Lockhart Clarke in 1876; but in 1873, Friedreich, of Heidelberg, devoted a quarto volume to the support of the myopathic view, and proposed to term the affection "Polymyositis chronica progressiva." About the same time Charcot established beyond dispute not only that the most typical form of progressive muscular atrophy in one or both upper limbs is due to a lesion of the corresponding anterior grey horn or horns, but also that other clinical varieties of the complaint can be assigned to separate and distinct anatomical changes.

Duchenne asserted that in the disease under discussion there is *atrophy without paralysis.* Although he abandoned the myopathic theory of its origin, he continued to maintain that there was no failure of "nervous *motor* action." He thought that the multipolar cells of the anterior cornua did not lose their motor, but only their *trophic* function, and asserted that the loss of power was always proportionate to the degree of wasting, and that any muscular fibres remaining at a particular time would always obey the will, even though they might not succeed in overcoming resistance so as to effect a visible movement. In proof of this doctrine he appealed to the fact that faradisation of the affected muscles would cause contractions after they had undergone extreme wasting, so long as any fibres remained. But Erb subsequently investigated with great care the action of galvanic as well as of faradic currents, and he was always able to detect, in at least some of the affected muscles, a modified (or "middle") form of the reaction of degeneration: the fibres contracted slowly under a powerful galvanic current when they were no longer sensitive to the faradic current, and A.C.C. was greater than C.C.C. The facts that this change cannot be detected in other muscles, and that it is easily overlooked where present, he attributes to the partial and gradual character of the atrophy; the fibres which have undergone degeneration are concealed by the still healthy fibres which lie beside them.

We may therefore conclude that the most common and typical form of Cruveilhier's palsy is truly an atrophic spinal paralysis, like infantile palsy, but exceedingly chronic instead of exceedingly acute; and it picks out, not certain muscles of a limb, but certain fasciculi, or perhaps certain fibres of a muscle.

Histologically the morbid process in the paralysed muscles is far from being as simple as it was at one time supposed to be. By the earlier writers on the subject the condition was described as "fatty degeneration," the muscles having lost their transverse striation, and being full of drops and granules of oil. Robin, however, found that the granules were often dissolved by acetic acid, and therefore could not be all of a fatty nature; and it is now known that while many of the fibres undergo fatty degeneration like that to which the heart is subject, others become merely reduced in size and thickness, while retaining their striation; some pass into the condition known as "cloudy swelling," and others again undergo vitreous degeneration, such as may occur in enteric fever. Another part of the process consists in an increase in the number of the sarcolemma-nuclei; and at the same time the perimysium becomes the seat of a cell-growth, from which are developed the adipose and connective tissue that sometimes gives to the muscles a deceptive appearance of being still well nourished. The several fibres undergo destruction not simultaneously, but in succession. Thus there are all possible degrees of wasting, down to a point at which the microscope reveals not a vestige of the normal striation, nor even an empty sarcolemma-sheath. Muscles so affected, instead of red, are pale grey or yellowish, and at length become converted into mere bands of white fibrous tissue, or bundles of fat lying between their tendons of attachment.

The histological condition of the cord in progressive muscular atrophy is essentially the same as we have seen to exist in the acute atrophic paralysis of children and adults. The large motor ganglion-cells of the anterior cornua are found shrunken and often much pigmented; or they have disappeared, and Deiters' cells or corpora amylacea take their place, along with fibrous tissue made up of neuroglia, atrophied nerve-fibres, and obsolete blood-vessels. The arteries are thickened, and in extreme cases the whole anterior cornu appears obviously shrunken even to the naked eye. The pathological process differs from that of other forms of cornual myelitis chiefly by its exceeding chronicity.

Cases in which the anterior horns have been found unaffected must be separated pathologically, however difficult it may sometimes be during life, from those just described and referred to the primary or myopathic atrophy to be presently described (*infra*, pp. 685 *et seq.*).

Spastic paralysis with atrophy.—In a large number of cases, sclerosis of the crossed pyramidal tracts is also found, and these were separately described by Charcot: according to Gowers, this condition (of anterior cornual and lateral sclerosis combined) is more common than the uncomplicated cornual atrophy.

In the typical affection of Aran and Cruveilhier, as just described, which Charcot called *amyotrophie progressive protopathique*, the primary morbid change is atrophy of the multipolar cells of one of both of the anterior grey cornua. The resulting impairment of muscular power presents those characters of sharp and apparently capricious limitation which have already been detailed, and is accompanied neither by disturbances of sensation nor by spasmodic contractions. Charcot's account of this form of the disease was based upon six or seven autopsies, and it has beer since fully confirmed.

The spastic cases of progressive muscular atrophy were called by Char ıyotrophies *deutéropathiques,* because he believed that the change anterior cornua is secondary to a lesion of the lateral columns of rd, *sclérose latérale amyotrophique,* consisting in a chronic inflam- r process which begins in the lateral white columns (occupying them ıtrically on each side), and then spreads into the grey matter, ›ally in the cervical region. Türck, in 1856, appears to have been ıst to notice this morbid change occurring independently of any ı in the brain. Charcot taught that it is characterised clinically ı presence of paralysis with rigidity of the lower limbs, in addition atrophy of muscles in the upper limbs. The spasm in the legs is t transitory, but afterwards permanent; they generally assume a n of flexion, and remain but little wasted. In the arms and hands uscles undergo excessive atrophy, not one by one, as in primary ssive muscular atrophy, but several at once; the elbow assumes a n of semiflexion and pronation, the wrist is bent, and the fingers are into the palm of the hand. Charcot further maintained that the l change in the muscles presents inflammatory or irritative characters ore marked degree than in the "protopathic" form; the connective between the fibres undergoes a more decided overgrowth, and the with which it is infiltrated are more numerous. Another peculiarity the course of the disease is more rapid; all four limbs become quickly ›d; the patient is confined to his bed in a few months, and does not ›re than from one to three years, the usual cause of death being an ion of the morbid process to the medulla oblongata, with the sym- of *bulbar paralysis.* This form of progressive muscular atrophy is y Charcot to be always incurable; those affected by it have been wenty-six to fifty years old; exposure to damp and cold has some- ›een assigned as its cause. At the Salpêtrière five cases had occurred ch autopsies were made when Charcot wrote.

ere is no doubt of the accuracy of these clinical observations of ›t, and lateral sclerosis affecting the crossed pyramidal tracts in the umbar region is often discovered after death in cases of progressive lar atrophy in addition to the characteristic change in the anterior . But this lateral sclerosis with its spastic symptoms is probably not y, but secondary to the poliomyelitis of the cervical enlargement produces the atrophic palsy of the arms—the view taken by Leyden. ı believes that lateral sclerosis is frequently, perhaps constantly pre- n spinal cases of progressive muscular atrophy, and that spastic ›ms are frequently present in cases where anterior cornual myelitis d after death, not secondary to the cornual lesion, as Leyden believes, Charcot supposed, its cause, but part of the same process. He there- ›uld draw no sharp line between "Cruveilhier's palsy" and Charcot's ›ophic lateral sclerosis, either clinically or pathologically.* Erb and ›rst recognise the accuracy of Charcot's striking account of the latter ›n, and place it provisionally with other forms of spastic paralysis. been mentioned as one of the forms of secondary spasmodic paraplegia l), but its true clinical place is certainly here, in close union, if not ›ration, with progressive muscular atrophy.

ɛ also a paper by Dr Ferrier in the 'Lancet' for 1881, p. 822, and one by Frieden- f Copenhagen, and Roth, of Moscow, in the 'Transactions of the International Congress' for 1884, vol. iv, pp. 100, 105.

Hypertrophic cervical pachymeningitis.—Atrophic paralysis of the arms due to pressure of thickened membranes upon the origin of the brachial plexus, with secondary spastic paralysis and atrophy, was described by Charcot as another form of *amyotrophie deutéripathique*. As its name implies, it is a chronic thickening of the dura mater, which presents a number of concentric layers, and may fill up the whole vertebral canal. Both dura and pia adhere firmly to the cord, so as to compress and flatten it, and also press upon those nerve-roots which come off at the level of the lesion,—generally those of the brachial plexus on each side. These cases, like those just mentioned of amyotrophic lateral sclerosis, are marked by progressive wasting of the upper limbs, and by rigidity of the lower. They are only distinguished clinically by the circumstance that the ulnar and median nerves are especially involved (the musculo-spiral nerve escaping), so that the wrist assumes a position of extension instead of being flexed. Charcot has observed that the skin often becomes anæsthetic, not only in the arms, but in the upper part of the trunk; and that when the lower limbs become rigid they do not seem to waste. But what is most distinctive of cervical pachymeningitis is an early stage lasting two or three months, and accompanied by severe pains in the neck and back of the head, by a sort of rigidity of the cervical muscles, by sensations of numbness and tingling in the upper limbs, and sometimes by bullous eruptions. These symptoms point to irritation of the posterior nerve-roots, and gradual interference with the motor and trophic influences from the cells of the anterior cornua upon the muscles and the skin. This affection seems to stand to other cases of atrophic spinal paralysis with spasms in the same relation as "compression paraplegia" to the common forms of myelitis.

Perhaps the most important point is that it is not always incurable. A woman under Charcot's care, after an illness of five or six years, during which she was for a long time perfectly helpless and confined to her bed, ultimately became able to walk, and could also to some extent make use of her hands. The lesion does not spread upwards, so that the symptoms are seldom complicated by the supervention of bulbar paralysis; but there is descending sclerosis of the lateral tracts leading to spastic paralysis of the legs. In the last stages there may also be loss of power over the bladder and rectum, and bedsores may form, pelvic symptoms not observed in other forms of wasting palsy, and belonging to the latter stages of spastic paraplegia.

As Erb remarks, it is sometimes impossible to distinguish between a meningeal new growth and cervical pachymeningitis.

The writer once had what he supposed to be a case of this form of paralysis in a young man who lay for nearly three years in Philip Ward. His symptoms were at first those of acute cervical meningitis, ascribed to local injury; these were followed by a period of paralysis with wasting of the arms, and then of spastic paraplegia with pelvic symptoms. He died in 1890, and *post mortem* no inflammation of the membranes was found, but multiple gliomata of the cord, without syringomyelia.

A woman was admitted to Guy's Hospital in 1878 with atrophic paralysis of certain muscles in the left hand; and she also was liable to exceedingly violent paroxysms of pain in the right shoulder and arm, attended with sudden redness and swelling of the tender parts, evidently due to disturbance of the vaso-motor nerves. She had traces of iritis, and I inclined to the diagnosis of a syphilitic gumma, growing from the membranes and pressing on the cord in the cervical region. She improved under iodide of potassium.—C. H. F.

Subacute atrophic spinal paralysis.—A form of paralysis was first described by Duchenne in 1849 and 1853 under the name of *paralysie générale spinale antérieure subaiguë.* He thought that it was due to an atrophy of the cells of the anterior grey cornua; and Erb called the disease *poliomyelitis anterior subacuta et chronica.* Perhaps the most suitable designation is "chronic diffused atrophic paralysis," which distinguishes it from the "acute atrophic paralysis" of children already described, and also from the limited forms of paralysis seen in "progressive muscular atrophy" of Aran and Cruveilhier's type.

The disorder generally begins in the lower limbs. The patient first experiences a sense of weakness in one or both of his legs, especially in going upstairs, or if he attempts to walk far. After a few days, or sometimes weeks, this develops into paralysis, so that he is obliged to keep his bed. Before long the upper limbs are affected, especially the fingers and wrists. The muscles of the trunk also become powerless, so that he cannot sit up, and has difficulty in coughing or sneezing, and in defæcation.

The paralysed muscles are flaccid. No reflex movements can be excited in them by irritation of the skin or of the tendons. They rapidly waste, so that with a measuring tape one can follow the loss of substance in the calves or in the thighs from week to week, until the bones seem to be covered only by the integuments. When electrical tests are applied typical reaction of degeneration is found to be present. Duchenne long ago discovered that response to faradic currents is extinguished, but the muscles react well to interrupted galvanism, and A.C.C. is more marked than C.C.C.

Power over the bladder and rectum is retained, and there is no tendency to the formation of bedsores. There is no anæsthesia; pains in the back and slight paræsthesia in the limbs are sometimes present, but on testing the cutaneous sensibility one finds it either perfect, or only very slightly blunted. At first there may be a little febrile disturbance, with headache and digestive disorder, but these quickly pass off and the patient afterwards feels well, and eats, drinks, and sleeps as usual.

The further progress of the disease varies in different cases. Generally it remains for a certain time stationary; that is, it ceases to spread upwards, or to extend to fresh sets of muscles, although those already affected go on wasting. But at last, after several weeks, a gradual improvement is observed; the arms regain movement, and afterwards the legs. All this takes place very slowly, and months pass before the patient can feed himself, or write, or walk. Sometimes he ultimately becomes as muscular, strong, and active as before, but some particular muscles may remain wasted for the rest of his life; according to Erb this is especially apt to be the case with the peronei. As recovery goes on, the normal electric contractility of the muscles gradually reappears.

On the other hand, there are cases in which no change for the better occurs, but the paralysis extends upwards to affect the face and the tongue, and to interfere with deglutition and respiration. In this way the disease may terminate fatally from one to four years after it began. It does not appear that there are any special indications to direct a prognosis at an early period of the case.

In two autopsies recorded, one by MM Cornil and Lépine, the other by Dr Webber,* the multipolar nerve-cells in the anterior grey cornua had undergone a more or less complete destruction; there were evidences of inflammation in the presence of compound granule-masses, the proliferation of the cells of the neuroglia, and accumulation of nuclei round the blood-vessels. In the antero-lateral columns there was slight degeneration of nerve-fibres. The motor nerve-roots and the muscles were in a state of extreme atrophy.

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* 'Trans. American Neurological Association,' vol. i, p. 45.

This form of progressive muscular atrophy is a rare affection, least so perhaps between the ages of thirty and fifty. It is separated from ascending myelitis by the absence of anæsthesia and of pelvic symptoms; from acute atrophic paralysis of adults by its more general distribution and by its better prognosis; from multiple peripheral neuritis by the absence of tenderness and also of anæsthesia. Lastly, the morbid anatomy of the disease separates it sharply from atrophic paralysis beginning in the peripheral nerves or in the muscles. It is no doubt a clinical variety of "Cruveilhier's palsy," but its distribution and course make it worth separate mention.

Diagnosis.—We have seen that the symptom of paralysis with atrophy of the muscles is common to many pathological lesions which must be distinguished at the bedside to the best of our ability. The following summary may help the student in the task, which is interesting but often difficult.

1. Atrophic paralysis due to *peripheral neuritis.*—There is no doubt that cases due to alcohol or lead have often been mistaken for Cruveilhier's palsy; but a careful observer will generally recognise the limitation of the affection to the area of distribution of particular nerves. Moreover in peripheral neuritis there is almost always anæsthesia, and usually local tenderness. Again, the peculiarities of the reaction of degeneration are well marked; whereas in progressive muscular atrophy they are imperfect (p. 671). The prognosis is often good.

2. *Acute and chronic atrophic spinal paralysis*, including essential spinal paralysis in infants and adults, and the subacute cases described above (p. 675), is marked by the facts that paralysis precedes atrophy, instead of going with it, and that instead of the reaction of degeneration occurring only in the "middle" or qualitative form, or only towards the close of the disease, it is present in its typical form. Partial recovery is the rule.

3. Hypertrophic cervical pachymeningitis causing atrophy of the arms and spastic paralysis of the legs (p. 674), is accompanied by pain and anæsthesia, which are absent in those of progressive muscular atrophy.

4. Syringomyelia with glioma of the cord in the cervical region may be recognised by pain in the arms and by the disassociation of sensations (p. 631). The prognosis is unfavourable.

5. Progressive muscular atrophy of primary muscular origin is distinguished by its occurring in children and in families, by its affecting the face, and also by the complete absence of spastic symptoms and of the reaction of degeneration. The prognosis is grave (*infra*, p. 684).

6. Lastly remain the group of cases which include those of Aran and Cruveilhier, and those called by Charcot amyotrophic lateral sclerosis. They agree clinically in their very slow and painless course and their preference for certain muscles of the hands and shoulders, and anatomically in depending on primary lesion of motor and trophic cells of the anterior [illegible] The progress of the disease is gradual but fatal. Its tendency is

slowly but surely to spread from one muscle to another until those of respiration are involved. Only occasionally is its course arrested, without apparent cause, and the atrophied muscles remain like those of a withered limb after infantile palsy.

Treatment.—Experience has shown that drugs are almost valueless in chronic anterior poliomyelitis, whether accompanied or not by lateral sclerosis. Gowers thinks that arsenic and strychnia are occasionally of service, but neither these drugs nor nitrate of silver, phosphorus, or iodide of potassium can restore the wasted muscles, or prevent further extension of the disease.

There is much difference of opinion as to the value of electricity in progressive muscular atrophy. Faradisation of the affected parts was strongly recommended by Duchenne. He advised the application of currents of moderate intensity, with not too frequent intermissions, and for a few minutes only at a time, so as not to fatigue the fibres remaining undestroyed; he particularly urged the importance of including in the treatment important muscles, such as the diaphragm, the intercostals, and the deltoids, when they are first threatened by the disease, and before they are wasted. In the case of a man named Bonnard, who had lost many muscles of his trunk, and who was beginning to suffer from dyspnœa, so that he could scarcely walk a few steps without stopping to take breath, faradisation of the phrenic nerves, repeated three or four times a week, was of great service, enabling him to walk considerable distances and to go upstairs without fatigue. A similar treatment applied to certain muscles of the arms, which were wasted, restored their functions, so that at the end of six months he was again able to support his family by his exertions; and he went on for some years without the disease advancing further.

Remak advocated the use of the galvanic current in the neck. At present interrupted galvanism is usually applied to the affected muscles. Ross believed that all drugs are useless, and that galvanism is the most efficient remedy for progressive muscular atrophy. But Gowers finds that "the most sedulous and skilful use of electricity, voltaic or faradic, fails, as a rule, to produce any effect on the course of the disease." He is equally incredulous of the asserted benefits of *massage,* which are "usually inappreciable," and of *balnéothérapie.* Even when the disease follows syphilis he finds mercury useless. "The disease is one in which it is not easy to do good and not difficult to do harm."

Early in the disease the affected parts should be kept warm and rested as much as possible. In one of Dr Fagge's cases the forearms were wrapped in cotton wool and placed in splints, and after a week the patient was found to have much more power in his hands.

BULBAR PARALYSIS[*] is the name given to chronic progressive atrophic paralysis when it affects certain cranial nerves. In 1860 Duchenne gave a clinical description of a form of progressive paralysis of the tongue, palate, and lips which had previously received but slight recognition, although Trousseau, as far back as 1841, had noted the peculiar symptoms presented by the well-marked case of the Prince de la Moskowa. The disease is not infrequent in England, and is often termed labio-glosso-pharyngeal paralysis, after Trousseau and Duchenne; but a shorter and better name is that of "bulbar paralysis." As Kussmaul has remarked, it might be called "progressive paralysis of the bulbar nuclei;" for the morbid change does not involve that part of the bulb (*i. e.* medulla oblongata) which transmits the motor and sensory strands for the limbs and the body generally, but is confined to the grey centres for certain of the cranial nerves on the floor of the fourth ventricle.

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[*] *Synonyms.*—Labio-glosso-pharyngeal paralysis of Duchenne—Glosso-labio-laryngo-pharyngeal paralysis—Progressive muscular atrophy of the tongue, palate, and lips—Atrophic bulbar paralysis (Leyden)—Progressive bulbar paralysis (Wachsmuth).

Course and symptoms.—As a rule bulbar paralysis begins insidiously. The earliest symptoms are commonly subjective,—a feeling of pressure at the back of the neck and head, a little giddiness, a sense of constriction round the throat or chest, slight discomfort in talking, as if the tongue were heavy and its movements laboured, or a tired feeling after speaking. Krishaber has stated that in two cases he discovered a loss of reflex irritability in the pharynx and larynx some months before any signs of paralysis made their appearance. Sometimes the palate is affected before the tongue, or the disease begins in the lips; in the former case the speech acquires a "nasal" quality, and swallowing seems to require an unusual effort; in the latter the expression of the face about the mouth becomes altered, or there may be difficulty in the utterance of certain letters, as though (to use a phrase of Duchenne's) the lips were half paralysed by cold.

Sometimes, however, the commencement of the disease appears to be sudden. Kussmaul, in one of the clinical lectures published by Volkmann, relates that a patient of his, a Catholic priest, found one day, while preaching, that his mouth was distorted and that he had a difficulty in speaking. He was able to finish his sermon, but from that time he felt a "heaviness of the tongue." For a week previously he had suffered from pains in the back, but he had had no giddiness. Dysphagia soon set in, and within six months the case became one of confirmed atrophic, bulbar, and spinal paralysis, with loss of power in the arms and wasting of the small muscles of the hands. It was ascertained at the autopsy that there was no hæmorrhage into the bulb.

In a woman whose case is recorded by Leyden the first symptom was a sudden attack of dyspnœa, lasting five minutes; a few days afterwards she noticed a difficulty in moving the tongue when she spoke or ate.

In fully developed cases the tongue is generally the part in which the loss of power is most obvious. It lies flaccid in the floor of the mouth: the patient can neither bend it laterally, nor raise it against the palate, nor hollow its centre; and he may be unable to protrude it beyond the teeth. Scarcely less marked is the paralysis of the lips. The mouth remains open, with its angles drawn wide apart; the naso-labial furrows are deepened, the lower lip hangs away from the gum. The patient cannot whistle or blow out a candle, or kiss. What is most remarkable is that although the facial muscles supplied by the lower branches of the *portio dura* are thus affected on both sides in every case, those to which the upper branches of the same nerve are distributed as constantly escape. The orbicularis palpebrarum and the occipito-frontalis act as well as ever; and the countenance thus acquires a curiously contrasted expression: the eyes are full of life, while the mouth is fixed, sad, and gloomy.

The atrophy of the paralysed muscles is best seen in the case of the tongue, which is shrunken as well as motionless, but though less easy to detect, wasting is always present in the lips and muscles of the larynx. On the other hand, the paralysis of the palate is not indicated by any obvious change in its form. The uvula hangs in its ordinary place; and Duchenne says that he always found that touching the fauces caused the usual reflex movements. With the laryngoscope the vocal cords may in advanced cases be seen to be more or less completely paralysed.

The defect of speech varies with the part first paralysed. According to Kussmaul, if the lips suffer first, O and U are the vowels which the patient experiences most difficulty in uttering; if the tongue, I is sooner lost. A is always retained longer than any of the others.* Among consonants, loss of power in the tongue renders the patient first unable to utter TH, R and SH; next S, L, K, G, T; afterwards D and N. Paralysis of the lips prevents the formation of P and F, then of B and M, ultimately of V. Paralysis of the palate gives to the speech a nasal twang, and it specially prevents the formation of the lip-sounds B and P, because it allows so much of the air to escape through the nose; the proof of this being, as Duchenne pointed out, that closing the nostrils may enable these letters to be sounded. So far the affection of speech is one which merely concerns articulation, and may be called "anarthria," in distinction from the "aphasia" that depends upon lesions in the left side of the brain, and the "aphonia" that is caused by loss of power in the larynx. But in bulbar paralysis, after a time the vocal cords also lose their functions; and the voice becomes extinguished.

Another set of movements which are interfered with in progressive bulbar paralysis are those concerned in the prehension of food, and its transmission into the œsophagus. During mastication it collects in the cheek, not only when the buccinator is paralysed, but because the tongue cannot properly dislodge it. Very often the patient helps himself by supporting the floor of the mouth or the cheeks, or pushing the half-chewed food into the proper position. The tongue cannot roll up the food into morsels for swallowing, nor carry them into the pharynx. Loose fragments are constantly dropping out of the open mouth into the plate or upon the patient's clothes, or collect about the roof of the tongue, or in the grooves by the side of the epiglottis. The attempts to swallow them succeed very imperfectly—some pass up into the pharynx or into the nose, others enter the larynx and set up a choking cough, or accumulate in the fauces so as to interfere with the passage of air. It depends upon circumstances whether the patient finds most difficulty in dealing with solids or with liquids. If the principal defect is a weakness in the tongue and in the muscles of mastication, he requires to have all his food reduced to a semi-fluid state; but when there is paralysis of the parts concerned in closing the larynx during deglutition, he can often dispose of solid masses better than of liquids, which he cannot keep from trickling down into the air-passages. The inability to swallow is the cause of the saliva running out of the mouth; such patients keep a handkerchief constantly held below the chin, and as Wilks remarks, this often at the first glance enables one to guess what is the matter with them. The saliva is sometimes viscid, sometimes watery; and is so abundant that in one instance Schultz is said to have estimated that there were six or eight times as much of it as usual. Kussmaul, however, found no such increase in a case in which he determined its quantity.

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* These vowels must be pronounced in the Italian way: U=OO (in *fool*), I=E (in *feel*), A=Ah (in *father*). The two English sounds represented by the letters Th are among the first to disappear in cases in which the tongue is affected. Kussmaul's statements correspond closely with what might have been anticipated from theoretical considerations, of which an admirable account will be found in a paper by Dr Bristowe in the first volume of the 'St Thomas's Hospital Reports.'

Sometimes the masticatory muscles may be at length involved in the paralysis; the patient is then unable to move the jaw from side to side, nor can he close the mouth firmly.

Generally the respiration is not obviously affected at an early stage of the disease. Afterwards dyspnœa often becomes a marked symptom, and there may be a constant craving for air, while the diaphragm and thoracic muscles are still vigorous. There is generally inability to sneeze, to cough, to hawk up phlegm, and to blow the nose; or the patient complains that he is no longer able to smoke. Towards the last paroxysms of tachycardia may occur, which probably depend on paralysis of the vagi.

The urine has often been searched for sugar, but it appears not to be present.

The higher cerebral functions remain undisturbed, the patient sleeps well, and his intelligence and memory are perfect. The movements of the body and limbs are generally free and active, except towards the last, when there is emaciation from want of food and extreme weakness.

Pathology.—Although, as just stated, the great motor paths through the pons and bulb escape, it frequently happens that there is associated with bulbar paralysis an affection of the upper (or more rarely of the lower) limbs, which agrees in all respects with Progressive Muscular Atrophy of Aran and Cruveilhier. Sometimes one, sometimes the other of these two local forms of the same disease is the first to develop itself. Their essential characters are identical, while the differences are comparable to those of atrophic paralysis affecting the arms and the same affecting the legs. The bulb is but the *medulla spinalis oblongata*, and the grey matter of the floor of the fourth ventricle is formed by the anterior cornua laid open and separated, so that the seat of each disease is serially homologous.

The ganglion cells in bulbar paralysis are in the condition described above (p. 672), the nerves in that described in a previous chapter (p. 571), and the muscles are degenerated as described at p. 672. In many cases the fleshy substance of the tongue, and even that of the lips and palate, is obviously pale, of a yellow or greyish-red colour; and it is more or less completely converted into a mass of fatty connective tissue. In the earlier stages the tongue is often not obviously reduced in size; but in advanced cases it is soft, small, wrinkled on the surface, and incessantly agitated by a fibrillary tremor. The lips also become thin and sharp-edged, their muscular substance quivers, and the skin over them is marked with minute furrows.

Faradic contractility is lowered or extinguished; and with galvanic currents, Erb, Kussmaul, and later observers find the reaction of degeneration in a perfectly characteristic form.

It is remarkable that spastic paralysis of the arms or of the legs does not exist or is extremely rare in cases of bulbar palsy, which belongs, therefore, to Cruveilhier's type of progressive muscular atrophy, not to Charcot's and Gowers'. Nevertheless even here there are not wanting occasional resemblances, for Dr C. E. Beevor has discovered a reflex movement of the mandible on percussion of the lower teeth, which he terms "jaw-reflex," together with "jaw-clonus," but only in the rare cases in which other spastic phenomena complicate atrophic bulbar paralysis ('Brain,' 1886).

Histology.—In general no marked change is seen by the naked eye in the fresh bulb: sometimes a little want of symmetry in the two halves of the floor of the fourth ventricle has been detected, or a slight shrinking; or this part has appeared discoloured, or its consistency has been greater or less than natural. One thing, however, is very obvious to the naked eye,—an extreme degree of atrophy of the nerve-roots arising from this part of the cerebro-spinal axis. It is especially conspicuous in the hypoglossal and the facial nerves; but it is visible also in the glosso-pharyngeal nerve on each side, and sometimes in the vagus, and in the motor portion of the fifth. The roots in question are grey and transparent, and much reduced in size.

In stained sections of the hardened bulb the multipolar cells in certain nuclei are uniformly found to have undergone degenerative changes. Their colour is of a dark yellowish brown; they are often shrunken; their axons are indistinct or may have disappeared. They seem to be reduced in number; and in one case Charcot and Joffroy came to the conclusion that the hypoglossal nucleus contained only one tenth or one twelfth part of the cells which would have been present in it in a healthy bulb. French histologists appear not to have recognised any marked change in the neuroglia, but in Germany Leyden in 1870, and Maier and other observers since, have found it increased in quantity, and containing Deiters's cells; appearances like those which characterise chronic myelitis going on to sclerosis. The parts in which these changes are most constantly seen are the nuclei of the vagus, the hypoglossal, and the facial nerves. As regards the last-mentioned nerve, indeed, the symptoms of bulbar paralysis point to the conclusion that only a part of its nucleus is affected; and this accords well with Lockhart Clarke's statement that its roots arise in two separate masses of grey matter. The nucleus of the glosso-pharyngeal nerve escaped the morbid process in a case recorded by Duchenne and Joffroy. The sensory nucleus of the fifth and that of the auditory nerve have been constantly found intact. The olivary bodies have sometimes presented degenerative changes, but more often they have been unaffected.

Ætiology.—With regard to the causes of bulbar paralysis nothing is certainly known.* Its incidence is decidedly later in life than that of the corresponding spinal paralysis of Cruveilhier. It scarcely ever occurs in persons under thirty, and appears absolutely to increase in frequency as age advances up to the seventieth year. It is more common in men than in women—thirty-four cases in fifty-three collected by Kussmaul.

The few cases that have been recorded in young persons have presented peculiar features, and their pathology is still undetermined.

Diagnosis.—The early recognition of the disease requires caution; one might easily make light of the early symptoms, and so justly forfeit the confidence of the patient. Even when they are fully developed one must avoid a hasty conclusion, for the lips and the tongue may be paralysed by various other affections.

Wilks long ago pointed out that a condition precisely like that of progressive bulbar paralysis may be suddenly developed as the result of a circumscribed effusion of blood into the lower part of the pons. He related in the 'Guy's Hospital Reports' (for 1877) two cases, in one of which an old brownish cyst was found at that spot after death. Such cases are recognised as the hæmorrhagic variety of bulbar palsy.

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* Erb speaks confidently of its being sometimes caused by cold, and Kussmaul relates a case in which the patient traced it to a cold caught while he was at work in the fields; he was attacked with headache and pain in the neck, and a week later he was conscious of difficulty in swallowing and in speaking. Among other conditions which have been supposed to give rise to it are excessive smoking, over-exertion in playing wind-instruments, syphilis, and falls producing symptoms of concussion. In one of Trousseau's cases it began during convalescence from a febrile attack.

Other observers have recorded cases due to syphilis, which have been cured by iodide of potassium; and tumours growing near the bulb may cause similar symptoms. Like most other nervous diseases, bulbar paralysis has been simulated by hysteria.

Prognosis.—It seems probable that the very few recorded instances in which recovery has taken place from "bulbar paralysis" have been essentially different from those described by Duchenne and Kussmaul.

Genuine cases which depend upon a progressive change in the bulbar nuclei appear always to end fatally. This was Trousseau's verdict, and all later experience confirms it. Their duration is generally from one to three years; but it may be as long as five. Sometimes the patient is suddenly choked by a mass of food which blocks the entrance into the larynx; of this an instance occurred at Guy's Hospital in 1865. Sometimes death is brought about by an attack of syncope, sometimes by a paroxysm of dyspnœa. Kussmaul remarks that such seizures often follow exertion, but they may also occur at night when the patient is in bed. In other cases exhaustion and emaciation gradually lead to a fatal termination; or pulmonary phthisis may develop itself; or acute pneumonia, from food being drawn into the air-passages during the act of deglutition.

Treatment.—This can only be palliative. Kussmaul thinks the nitrate of silver more likely to be useful than any other drug. He has seen temporary benefit from faradisation of the palate and tongue; and he mentions two cases in which striking results were for a time attained by interrupted galvanic currents passed through the neck and spine. One of his patients, the priest mentioned above (p. 678), who had been unable to put his tongue out between his teeth, could, after being galvanised, protrude it beyond his lips; his speech became more distinct and his deglutition easier; but after four or five weeks the improvement ceased. Schulze's method of inducing the act of deglutition by galvanism seems to be applicable to progressive bulbar paralysis. It consists in fixing the positive pole upon the nape of the neck, and then rapidly moving the negative pole downwards over the side of the larynx. This may be repeated at short intervals during four or five minutes. In the first instance a current from six to eight cells should be tried; but to produce the desired result a large number of elements are often required.

When the patient cannot swallow food, or seems likely to be choked in the attempt, a tube must be passed into the stomach. If the irritation thus excited is too great, nutrient enemata afford the only means of sustaining life for a time, unless recourse is had to gastrostomy. In one case at Guy's Hospital the trachea was opened when danger was apprehended from frequent attacks of dyspnœa; and the result was that they ceased, although of course the other symptoms remained.

Acute bulbar paralysis—myelitis bulbi acuta focalis.—The remarkable malady just described is, we have seen, an example of atrophy of the ganglion cells of the motor tract, comparable with the similar anatomical condition which causes progressive muscular atrophy. The latter affects the anterior cornua of the medulla spinalis, the former the corresponding grey nuclei in the medulla oblongata.

A few cases have been recorded of an acute paralysis of the muscles supplied from the bulb, which similarly corresponds to acute atrophic spinal paralysis of children from a sudden anterior cornual lesion. Three examples were observed by Leyden. In one case a patch of softening with numerous capillary extravasations of blood was plainly visible in the pyramids and in the olivary bodies at the autopsy; in the others no lesion was discovered until the parts had been hardened, when the microscope revealed marked inflammatory changes. Each patient had been taken ill rather suddenly, and had died in from four to ten days. The symptoms varied considerably, since they depended upon the exact seat of the morbid process in a part of the nervous system where many important centres are gathered into a narrow space. Chief among them were dysphagia, headache, giddiness, vomiting, or severe hiccough, more or less impairment of speech, irregular, rapid, or interrupted breathing, a quick, feeble, and intermittent pulse, partial or complete paralysis of the tongue, formication and pains in the limbs. There was no failure of consciousness, but collapse set in, and death followed from paralysis of the respiratory muscles, with dyspnœa and lividity.

The *diagnosis* of the seat of the disease seems not to be difficult; but it is not easy to exclude the possibility of embolism of the basilar artery or of the vertebral arteries or their branches, or of a minute spot of hæmorrhage, or even of a rapidly-developed compression of the medulla oblongata by some disease in its neighbourhood.

Hæmorrhage, embolism, tubercular and other tumours of the bulb, have been recorded, with symptoms resembling those of focal myelitis affecting the same region. Lastly, in certain cases of cerebral hæmorrhage affecting first one and then the other hemisphere, symptoms very like those of bulbar paralysis may be produced. Such cases are distinguished as sudden ("apoplectic") bulbar palsy. Possibly they need not invariably prove fatal, since recovery from myelitis and hæmatomyelia is not unknown. A case which seems to have been of this kind occurred in Guy's Hospital in 1874, in a man aged forty-five. It lasted about ten days. After death the central part of the pons appeared softened; but when hardened in chromic acid, Dr Fagge could not make out decided morbid changes.

Myasthenia gravis.—Cases clinically like bulbar paralysis, but without any local lesion being found after death, have been recorded by Wilks ('Guy's Hosp. Rep.,' 1877), by Strümpel, by Dr L. E. Shaw ('Brain,' 1890), by Jolly ('Neurolog. Centralblatt,' 1894), and by several other physicians at home and abroad. Dr Buzzard has lately published two cases in the 'British Medical Journal' for March 3rd, 1900, under the title proposed by Jolly, *Myasthenia gravis pseudoparalytica.*

Although several of the cases begin with bulbar symptoms, the condition is far more wide-spread that bulbar paralysis, and causes extreme weakness of muscles in any, and sometimes in every part of the body; ptosis and ophthalmoplegia are not infrequently present. Moreover there is no muscular atrophy or fibrillary tremor. The muscles generally are weak and readily fatigued; and though they respond to faradism, they soon lose their contractility until a short period of repose has followed. In the same way, though to a less marked degree, they answer to a galvanic make and break: and in the same way they are soon exhausted by voluntary movements.

This myasthenic reaction, as Jolly named it, with the other symptoms above stated, the absence of muscular atrophy and the absence of lesions after death, widely separate the most characteristic of these cases from bulbar paralysis. Some recorded appear to belong to another group, and it is quite uncertain whether the cause of the symptoms is toxic, and whether its seat is in the upper or lower neurons, in the motor nerves, or in the muscles themselves. One fact of great practical importance is that these cases, as a rule, end fatally, and that after apparent improvement or even recovery has taken place.

No useful treatment of this singular condition is known; but it is known that attempts to excite contraction of the affected muscles are injurious, and that feeding by a tube is dangerous.

Here the account of atrophic paralysis as a disease of the spinal cord ends, so far as our present knowledge extends. But there are many forms of muscular atrophy in which neither the cord nor the motor nerves are implicated, which cause what is to the patient paralysis—weakness, and loss of movement—and yet are primary affections of the muscles themselves. It is, however, historically important not to separate them from the cases in which atrophy of the muscles is the result of peripheral neuritis or spinal disease, and, clinically, it is still sometimes no easy matter to decide to which group a case belongs.

PRIMARY MUSCULAR DYSTROPHY—*Idiopathic muscular atrophy—Progressive myopathy.*—Under this general head the remaining forms of "wasting palsy" may be collected. But they fall under several more or less decidedly separated clinical groups.

(1) *Progressive muscular atrophy of children.**—Progressive muscular atrophy is occasionally seen in children, but with certain peculiarities in its symptoms and course which correspond to a fundamental difference in pathology. It begins in the lips, which cannot be brought together; and if the child smiles, the angles of the mouth are drawn far apart, and the cheeks are flattened by the action of the buccinator muscles. The articulation of the labials and of the vowel *o* is impaired. It is almost always between the fifth and the seventh year that this affection first appears; but at that time it often attracts little notice from the parents, although the reality of the morbid change is at once made apparent if the orbicularis oris and the other muscles are tested by faradic currents, for they are found to have lost their contractility. About the eleventh or twelfth year the muscles of the shoulders and arms begin to waste, and then medical advice is sought. Later still the muscles of the trunk and those of the lower limbs are attacked in their turn, but the hands escape.

One fact which shows that the progressive muscular atrophy of childhood is distinct from the wasting paralysis of adults is that it very often occurs in two or more brothers and sisters.†

This form of disease is rare; but Duchenne, whose first cases were recorded in 1855, had observed no fewer than twenty in 1872. Landouzy and Déjérine published several in 1874, and one, with an autopsy, in 1885.

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* *Synonym.*—Primary muscular atrophy in children. Duchenne's early myopathy.

† It is curious that the father of the first two children in whom Duchenne recognised its peculiar characters was afterwards, at the age of forty-eight, attacked with typical "Cruveilhier's paralysis," affecting first the shoulders and arms, but ultimately the lower limbs; moreover his father had died of a similar complaint.

Barsickow's series of cases of hereditary muscular atrophy, published in 1872, are of a similar clinical type. Westphal, E. Remak, and other writers have published a few cases since, and several have been observed in this country.

The muscles of the face are not exempt, and girls as well as boys are affected. The cord and bulb have been found unaffected. If the seat of the disease is in the nerves, it is a form of peripheral neuritis affecting the facial and other motor nerves; but in most cases it appears to be of primary muscular origin.

A patient under the writer's care in 1890, a lad of ten, showed all the characters of "Landouzy's type" of muscular atrophy.

Erb's juvenile hereditary form.[*]—A clinically distinct "juvenile form" of progressive muscular atrophy has been described by Erb ('Deutsches Archiv f. klin. Med.,' 1884). It usually begins at puberty. The muscles of the shoulder are more affected than those of the arms, and the legs are not infrequently attacked, but the face and hands escape. These cases, like Duchenne's above noted, are marked by a hereditary, or at least a family, character. There is no fibrillary contraction, no reaction of degeneration even of the "middle" or imperfect kind, and *post mortem* the anterior cornua have been found intact by Friedreich. Occasionally the deltoid or other paralysed muscles hypertrophy—which connects this form with the pseudo-hypertrophic atrophy to be presently described.

Dr Tooth collected the previously published cases of hereditary atrophic paralysis, and added four new ones. Among 30 cases in all, he found that 8 occurred between one and five years of age, 10 between five and ten, and 18 between ten and twenty, while only 3 patients were older—twenty-five, thirty-seven, and forty-six; 28 were males and 16 females, so that the disproportion between the sexes was much less than in Cruveilhier's muscular atrophy. Grouped in families, there were 17 instances of more than one case in a family, and only 12 isolated cases.

In one group the lower extremities were first affected, and particularly the *peroneal* muscles. The extensors and gastrocnemii next shared in the paralysis, and the hands and forearms followed. Fibrillary tremors and marked reaction of degeneration were usually present ('St Barth. Hosp. Rep.,' 1889; and 'Lond. Med. Rec.' for October of the same year, p. 430).

It appears from the anatomical results of a few fatal cases, that the lesion is not central, nor a peripheral neuritis, but primary (myopathic) muscular atrophy.

Meryon's four remarkable cases of *hereditary muscular atrophy*, described most accurately as early as 1852 ('Med.-Chir. Trans.,' vol. xxxv, p. 73), probably belong to this same clinical group, though they have been often appropriated as examples of the following disease.

Pseudo-hypertrophic paralysis.[†]—In 1861, Duchenne recorded in the second edition of his 'Electrisation Localisée' the case of a boy whose legs were so weak that he could scarcely stand, although the muscles of his calves and hips looked like those of an infant Hercules. Seven years later the same fertile author wrote a detailed paper on the disease in question.

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[*] *Synonyms.*—Dystrophia musculorum progressiva juvenilis (Erb)—Myopathic atrophy of adolescents.

[†] *Synonyms.*—Myopathic atrophy with fatty overgrowth—Myosclerosis—Progressive muscular sclerosis—Lipomatosis musculorum luxurians.

In the meantime cases had been noticed by a few German observers. The writer saw one in 1863 in Oppolzer's wards at Vienna: and another, which came under Griesinger's notice in 1864, gave Billroth the opportunity of excising a piece of the deltoid muscle and of proving that there was no real but only an apparent hypertrophy, for it consisted almost entirely of adipose and fibrous tissue. Duchenne, in 1865, made similar observations in one of his cases with the aid of his "emporte-pièce." He therefore proposed to term the disease *paralysie myosclérosique*. Gowers' monograph, published in 1879, contained all the information obtained up to that date, and little has been added since.

Histology.—The substance of the enlarged muscles has a whitish-yellow colour, with only a faint reddish tint. There is still a linear arrangement of the fibres, and in extreme cases this alone distinguishes it from the subcutaneous adipose tissue. The fat sometimes extends into the tendons, so that during life they seem to have been encroached upon by the fleshy bellies of the muscles. Occasionally, on the other hand, fibrous tissue only and not fat has been found between the muscular fibres; and in the earlier stages of the disease, this new tissue has been found full of nuclei or spindle-cells. The muscular fibres seem to become greatly reduced in number by a process of simple atrophy, which at last leaves only the collapsed sarcolemma-sheaths. Those fibres which remain are not always completely unaltered, as described by Griesinger. Duchenne described their transverse striation as unusually faint. Other observers have seen some which were striated longitudinally, some translucent or vitreous, and some in a state of granular or fatty degeneration. Some fibres again are truly hypertrophied, so as to be two or three times as thick as normal, a fact first observed by Cohnheim.

Course.—The enlargement of the muscles is not present at all stages of pseudo-hypertrophic paralysis. There is an early period during which the only symptom is an impairment of power in the lower limbs. The child—for the affection almost always begins in childhood—is noticed to totter in walking, and to be apt to fall; he has difficulty in getting on to his feet, and is particularly awkward in going upstairs; when he tries to sit down he falls into the chair. In many cases the disease begins before the little patient has learned to walk, so that at first the parents think that the child is only backward, as many rachitic children are, but at length they see that there is something more seriously wrong.

When, at the end of a few months or a year from the beginning of the paralysis, the change in the muscles is discoverable, its extent and degree vary widely in different cases. The calves are often affected alone; next in liability to undergo enlargement are the masses of the *erector spinæ* in the loins, the *glutæi*, the *deltoids*, and the *infra spinati*. The *glutæus medius* usually escapes. The muscles of the thigh, the *latissimus dorsi*, and the lower part of the *pectoralis* and *serratus magnus* are more often reduced in size, so as to afford a strange contrast. When the femoral muscles are hypertrophied, it is usually the *vastus externus* or *rectus*, very seldom the muscles at the back or inner aspect of the thigh. But sometimes every muscle of the trunk and limbs is hypertrophied, until the patient looks like an athlete, though in reality as weak as the puniest child in the last stage of marasmus. Duchenne compares a sketch of the Farnese Hercules with portraits of a patient of his, a boy ten years old, and the child looks the more athletic of the two. The enlarged muscles are generally firm and elastic, and when they are brought into action they harden; so that it is difficult to believe that they consist of little but fibrous and adipose tissue.

When a child affected with pseudo-hypertrophic paralysis stands up, his attitude is characteristic. The abdomen is pushed forwards, and the hollow of the lumbar vertebræ is greatly exaggerated. The nates project backwards; but the shoulders are thrown further back still, so that a line dropped from the upper dorsal spines falls behind the sacrum. The legs are separated widely from one another. If an attempt is made to straighten the back, the child at once falls down. When he walks, he balances the body from side to side at every step, with a "waddling" gait. He cannot rise from the sitting posture without the use of his hands, and the way in which he uses them is characteristic. He places both hands upon his knees, and gradually pushes up his body into the erect posture by moving his hands from his knees to his groin. This manœuvre, which has been not inaptly called "climbing up the thighs," is well represented by sketches in Dr Gowers' work (p. 391), and by photographs in Dr Ross's first volume (p. 996).

The susceptibility of the affected muscles to faradic currents is sometimes normal, but in other cases it is considerably lowered. This appears to depend solely on the degree of atrophy of muscular fibres, for there is no reaction of degeneration. As with all the atrophic disorders of muscles described in this chapter, pelvic symptoms are absent, and the sphincters act naturally throughout the whole course of the disease. The knee-jerk gradually disappears, as in other atrophic forms of paralysis, not from severance of the muscle from its nutritive neuron, but from mere absence of the fibres that should contract. Fibrillary tremors, such as are seen in progressive muscular atrophy, are not generally present; but they have been observed in a few instances. The cutaneous sensibility is unimpaired. The legs and feet are often cold and damp and bluish after the atrophic stage is reached. The heels are usually drawn up to a greater or less extent by contraction of the *tendo Achillis*; and there may at last be a well-marked club-foot. Sometimes the knees are rigidly flexed. The intelligence may be perfect: but it is often defective, and in some of the cases recorded the patients have been idiots.

When the disease has developed itself to a certain point, it is described by Duchenne as remaining stationary for two or three years, or even longer. But at length a further advance takes place. If the legs only were affected, the arms are now involved; but the muscles which now lose their functions, do not even show an apparent hypertrophy, but are always reduced in size, while those muscles which were at first enlarged ultimately shrink, until they, too, are obviously atrophied. The patient, who has generally now reached adolescence, becomes altogether unable to stand, or even to sit up; he is a prisoner upon his couch. Ultimately he dies of exhaustion, or is carried off by phthisis, or some other intercurrent complaint. As Friedreich, however, remarks, most of the cases recorded have been lost sight of, so that it is not known how they ended.

Ætiology.—Nothing is known as to the cause of pseudo-hypertrophic paralysis. It often appears in succession in two, or three, or even four children of the same family, and at the same age in all of them. As might be expected, the parents themselves have always been free from it; but its hereditary origin has often been traceable by its having occurred in brothers or other relations of the father or mother. Transmission is almost always through mothers to sons. In this character of chiefly affecting males, but being chiefly transmitted through females, this curious disease resembles Hæmophilia.

Boys are far more subject to this malady than girls, the proportion, among seventy-seven cases collected by Friedreich, being sixty-four to thirteen. Of Duchenne's original fifteen cases only two were girls; of forty-one collected by Webber of Boston and quoted by Ross, only five; of thirty-three by Gowers, ten. In a remarkable series of cases recorded by Lutz it appeared in two successive generations in the female line only, and affected five girls. More often it attacks several sons of the same parents, and spares all the daughters.

Out of 75 cases in which the date of its commencement was ascertained, Friedreich found 45 in which it began under five years of age, 17 between six and ten, 8 between eleven and sixteen. In the remaining 5 cases the patients were adults; and 2 of them were aged forty and forty-one respectively when the disease began. There was in 1887 a patient in Guy's Hospital under Dr. Frederick Taylor, a youth of eighteen, in whom this disease had developed within only a few months.

Dr. Port, of the German Hospital, showed two cases at the Hunterian Society (1887-8), in brothers aged fourteen and nine years. Their four sisters were healthy and one brother; but three other brothers had died after showing symptoms of the same disease.

In eight cases at Guy's Hospital six were in male and two in female patients; two were under ten, and four between ten and twenty. Beside these the writer has only seen three more, all in boys and strikingly alike.

Diagnosis.—This is very easy in well-marked cases. One morbid state, which must be thought of in connection with it, is that which Brodie and Hilton both believed to depend on the hip-joints being congenitally placed too far backwards; the drawings given in Hilton's work show a compensatory curvature of the spine very like that which is seen in pseudo-hypertrophic paralysis.

Dr Leech's "harpoon" enables one to examine the muscular tissue during life, and is said to be more useful than Duchenne's "emporte-pièce."

The striking apparent hypertrophy of the muscles is not an essential feature of the disease. We have seen that it is absent from the earlier and the latest stages; and possibly Duchenne and Friedreich were right in including under the present head the cases described by Dr Meryon in 1852 under the name of "granular and fatty degeneration of the voluntary muscles," which occurred at about the same age in several boys belonging to two families (p. 687). Charcot and Roberts, however, regarded the cases in question as examples of progressive muscular atrophy; and Friedreich himself relates as instances of the latter disease several cases which seem to belong to the same class as Meryon's (cf. 685). They occurred in three families residing in or near Heidelberg; and what is very curious is that Hemptenbacher, a pupil of Friedreich's, succeeded in tracing all three families to a single pair of ancestors a century and a half back. A similar malady is said by Eichhorst to have appeared in six successive generations in a family at Königsberg.

Pathology.—In accordance with Charcot, most writers at the present time hold that this disease is primarily an affection of the muscles themselves, and thus differs absolutely from progressive muscular atrophy,

een traced to a lesion in the grey matter of the cord. In pro-
scular atrophy, and even in the atrophic paralysis of children,
muscles occasionally have their bulk made up to the natural
an interstitial development of adipose and fibrous tissues; so
he other hand, an apparent overgrowth of the muscles is not an
ture of pseudo-hypertrophic paralysis, this affection comes very
athology to the other muscular atrophies.

Pierret and Charcot studied the histology of the cord in a
Bergeron's, and detected no morbid change, afterwards Lock-
and Gowers recorded a case in which they discovered disintegra-
ous parts, in some of which the nerve-cells were in a state of
r Fagge was strongly inclined to the opinion that pseudo-hyper-
alysis will ultimately be found to be a spinal affection, and
st edition of this work was published Ross and Bramwell have
changes in the spinal cord. In a second case Ross found the
erves unaffected; and in the second edition of his treatise, he
he theory of a spinal origin, and came to the conclusion that
rtrophic paralysis is a primary and idiopathic atrophy of the
owers also holds that "the essential lesion is the growth of
issue by which the muscular fibres are damaged, whether fatty
nd or not."*

rest allies of this curious disease are the hereditary juvenile
y myopathic forms of progressive muscular atrophy.

gnosis of this disease is at present unfavourable, and no treat-
to check its progress. Duchenne's trial of faradism and Bene-
b's of galvanism show that both are useless.

multiple muscular atrophy is a condition closely allied to the
out differing in the fact that there is no stage of apparent
. Cases will be found in Poore's edition of Duchenne's great
n Gowers' treatise. They occur in families, and sometimes re-
everal generations. Thus Barsickow, in a thesis on the subject,
Gowers, found twenty-four cases in five generations. The
not much, if at all, more frequent in men than in women, and
ears, not in childhood, but in early adult life. It often begins
les of the face, giving what has been called the "myopathic
or rather want of expression. When fully developed, the con-
uch like that of advanced progressive muscular atrophy, and
ne of the cases might take their place as examples of Duchenne's
type referred to above (p. 686).

uscular hypertrophy.—Apart from the physiological hyper-
ealth, a few cases have been described—by Auerbach, Berger,
ich—in which the muscles of the limbs, particularly the arms,
increased in size without increase or with diminution of con-
er. When a minute portion is removed it is found that the
bres are increased in diameter, without interstitial fibrous or
owth. Though it is nearly twenty years since the first case
l, the pathology of this rare affection is still unknown.

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possible that the apparently inconstant changes in the cord may be the
the cause of the muscular atrophy, like the atrophy of motor nerve-cells
Dickinson and other observers as the result of amputation? ('Journ. of
. 88; see also Dr Dreschfield's case, *ibid.*, 1879, p. 424, and Mr Bowlby's
Diseases of Nerves,' p. 27, with reference to Vulpian's experimental results.)

TABES

> Ἡ νωτιὰς φθίσις ἀπὸ τοῦ μυελοῦ γίνεται.
> HIPPOCRATES: De morbis, lib. ii, cap. 19*

History and nomenclature—Ataxic symptoms—Anæsthesia—Lightning pains—Visceral pains—Paraplegic symptoms—Loss of knee-jerk—Disorders of the eyes—Atrophy of joints—Perforating ulcer—Other complications—Course and event—Anatomical seat and histology of tabes—Pathology and relation between the ataxy, dysæsthesia, and other symptoms—Ætiology—Diagnosis—Prognosis and treatment.

Ataxic paraplegia with spastic symptoms.

Hereditary tabes in the young, or Friedreich's disease: its symptoms and anatomy.

Cerebellar ataxia occurring in families.

Synonyms.—Tabes dorsalis *v.* dorsualis—Spinal tabes—Locomotor ataxia—Ataxie locomotrice progressive (Duchenne)—Asynergie locomotrice (Trousseau)—Posterior spinal sclerosis—Chronic posterior leucomyelitis.—*Fr.* Ataxie locomotrice.—*Germ.* Tabes dorsalis, Hinterstrangsclerose.

History and nomenclature.—The old term "tabes dorsalis" was first accurately defined by Romberg, who, in 1851, described the characters which are now known by that name. Duchenne, in 1858, re-described the symptoms and proposed the term "ataxie locomotrice progressive;" but this denotes a symptom and not a disease, and a symptom which, though common, is not constant in tabes. Romberg did not appreciate that the apparent loss of power was really a loss of co-ordination: but, with this important exception, he describes the symptoms of tabes with accuracy and insight: diminution of the sense of touch and muscular sense, the need to watch the movements of the legs with the eyes, the tottering in the dark, the weakness of the bladder, pains shooting through the legs, girdle-pain and colic-pains, amblyopia, myosis, impotence, late implication of the arms, and final complete paralysis of legs and bladder. He also described the atrophied posterior roots of the nerves, and atrophy of the cord confined to the lower part of the posterior column (ii, p. 395).

Duchenne honestly says that "M. Romberg a le mieux observé et décrit la plupart des symptômes propres à l'ataxie locomotrice progressive," and he himself admitted that he was ignorant of the constant anatomical lesions in the new disease.

A series of cases was described by Gull in 1856-7 as "cerebral paraplegia;" but earlier still Todd, in 1847, had pointed out the distinction between paralysis and loss of the power of co-ordinating movements (*ataxia*). He recognised the difficulty of walking and the tottering uncertain gait which result from ataxia. Moreover, he went beyond Romberg and Duchenne in correctly assigning the seat of co-ordination to the posterior columns of the cord. "Some support is obtained for this view of the function of the posterior columns from the phenomena of disease. In many cases, in which the principal symptom has been a gradually increasing difficulty of walking, the posterior columns have been the seat of disease. Two kinds of paralysis of motion may be noticed in the lower extremities, the one consisting simply in the impairment or loss of voluntary motion, the other distinguished by a diminution or total loss of the power of co-ordinating movements. In the latter form, while considerable voluntary power remains, the patient finds great difficulty in walking, and his gait is so tottering and uncertain that his centre of gravity is easily displaced. These cases are generally of the most chronic kind, and many of them go on from day to day without any increase of the disease or improvement of their condition. In two examples of this variety of paralysis I ventured to predict disease of the posterior columns, the diagnosis being founded upon the views of their functions which I now advocate; and this was found to exist on a *post-mortem* inspection; and in looking through the accounts of recorded cases in which the posterior columns were the seat of lesion, all seem to have commenced by evincing more or less disturbance of the locomotive powers, sensation being affected only when the morbid change of structure extended to, and more or less involved the posterior roots of the spinal nerves" ('Cyclop. of Anat. and Phys.,' vol. iii, p. 721).

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* Tabes dorsalis arises from the spinal marrow.

The original meaning of the term Tabes dorsalis was purely clinical, and had reference to the weakness and pains in the back, the malnutrition and other disorders which follow excessive sexual indulgence. In fact it nearly answered to what is often understood by Neurasthenia spinalis (p. 643). *Tabes dorsalis* from this cause is regarded by Cullen in his nosology as a species of atrophy, only distinguished by the absence of fever from that of phthisis, of mesenteric tabes, and of chronic suppuration. Celsus has no dorsal species of the genus Tabes.

Now, however, the word *tabes* is not used, any more than its Greek equivalent *phthisis*, to denote generally wasting, atrophy, or marasmus. "Tabes mesenterica" is tuberculous enteritis and peritonitis, with secondary swelling of the mesenteric lymph-glands. It is so different from Tabes dorsalis in its clinical features and associations, that confusion can scarcely arise between them. There is therefore no reason against the term Tabes (or, when needful for distinction from Tabes mesenterica, Tabes dorsalis) being used for the remarkable disease described by Romberg and Duchenne. It has every character of a good name. It is short, classical, unmeaning, distinctive, and capable of forming an adjective. This adjective is ready made: *tabidus*—not *tabeticus*.

Clinical characters.—Tabes is always insidious in its origin, in its progress gradual, and though certain in its event, yet of most uncertain duration. Ataxic symptoms occasionally only appear after months or years marked by pains, by loss of knee-jerk, or by contracted pupils; but they are almost constant and very characteristic.

Motor symptoms.—The patient begins to feel a want of control over his legs. He has power to move them, but cannot direct the force or combine the action of several muscles. He has unlearnt the act of walking, and when he tries to practise it, he lifts his foot high, throws it outwards, and brings it down with a stamp. At first it is only in starting that he experiences a difficulty. Wilks relates how a gentleman whom he knew, if he stopped to look in a shop-window, had to ask someone near to give him a push before he could set off again; once fairly started he did pretty well, but found himself unable to turn sharply round without stumbling. Another patient, when he had once walked some distance along a road, wanted to go back, but could only do so by guiding himself up against a bank. A third, an out-patient, one day apologised for being late at the hospital by saying that his friends had sent him off in a wrong direction, and that he went on until he fortunately met an acquaintance who turned him round.

Ataxic patients do not roll and stagger like a drunken man, but as Wilks says, the gait rather resembles that of a man walking on a ledge who is anxiously balancing himself. The distinction is a real one, but many persons in the early stage of the disease have been looked on as drunkards.

The attempt to run, to hop, or to ascend stairs, makes the patient's ataxia more manifest. He is unable to stand on one foot, or with the two feet close together. If told to walk in a straight line—as along one particular board in a floor—he finds that his utmost efforts cannot keep him to it. The use of a stick, or leaning upon the arm of a friend, has a remarkable effect in steadying his movements. In more advanced cases, as soon as he attempts to stand, his legs are jerked in all directions, even though he may be held up on each side by an attendant. When lying on a couch he cannot carry the foot straight towards an object so as to touch it, nor raise the leg up in the air with an even movement. Yet he can exert considerable power in moving his legs against pressure, as in lifting a weight upon his knees.

The disorder may last for years without extending beyond the lower limbs, but in many cases it at last affects the arms, and occasionally ataxic symptoms begin there. The patient can then no longer perform actions requiring delicate manipulation, such as writing, or playing the piano; he cannot fasten his necktie or button his clothes. If he is told to bring the two forefingers into contact from a distance, they miss one another; he cannot touch his own nose or rub his own eyes with certainty; he blunders in taking hold of anything held up before him; and if he attempts to write in the air his fingers are moved zigzag in all directions.

Beside co-ordination of muscles, the muscular sense is also lost. If a weight is hung by a strap to the foot, the patient cannot guess its absolute or even its comparative weight.

A symptom on which French writers lay great stress, as indicative of locomotor ataxy, is the failure to execute movements as well with the eyes shut as when they are open. Many a patient who can stand steadily so long as he is looking down at his feet, totters and falls when he is made to close his eyes. It may be the first indication that anything is amiss: thus, in a case recorded by the late Dr Bazire, a man believed himself to be quite well until he noticed that he could not wash his face in the morning unless he could lean against a wall; for as soon as he shut his eyes, he lost his balance and staggered.*

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* A person who has ataxy cannot walk so well when the eyes are closed as when they are open, even when something is held in front of his chest so as to prevent his [illegible]

This symptom of inability to execute movements accurately with the eyes closed is invariably present when there is much anæsthesia, although it may sometimes be observed when there is none. A patient suffering from any spinal disease may be expected to totter in attempting to stand with his eyes shut, if he has imperfect sensation in his legs and feet, but the want of stability is exaggerated by defect of co-ordinating power. That is to say, as Leyden rightly taught, ataxy is the direct result of disturbance of the tactile and also of the muscular sense. Hence, the test of walking with the eyes shut, though practically useful in the diagnosis of tabes from peripheral affections of the limbs, is not pathognomonic.

Sensory symptoms.—Feeling is more or less impaired in cases of tabes. Subjective feelings of numbness, or of formication, are very often complained of. The patient says that when he stands, his feet seem to be covered with thick woollen stockings, or to be treading upon a water-bed, or upon india-rubber. The cutaneous sensibility, when carefully tested, may be found diminished in the feet, the legs, or even the thighs; but there is rarely anything like complete anæsthesia. Ability to feel pain is often absent (*analgesia*), when tactile impressions are readily perceived, and patients generally retain the power of appreciating differences of temperature long after common sensation is diminished.

That curious symptom, the retarded transmission of impressions, is almost peculiar to locomotor ataxy. It especially concerns the sense of pain, but tactile impressions and those of heat and cold may also be delayed. If there is no algesia, the prick of a needle may first be only felt as a touch, and then, after an interval, a sensation of pain may follow. Erb says that the interval may be some minutes; Cruveilhier, who seems to have been the first to draw attention to the symptom in question, noted it at twenty or thirty seconds. Another point is that the pain produced by a slight injury may last longer than under normal circumstances and gradually culminate. The patient is often unable to count a series of similar impressions if they succeed one another at all quickly.

These paræsthesiæ are by no means limited to the lower limbs. Sensations of numbness are often felt in the parts of the hands supplied by the ulnar nerves, even at the commencement of the disease. Trousseau found in some cases that the mucous membrane of the mouth is anæsthetic, the patient not being able to feel the food between his lips, nor to appreciate its temperature.

Pains in the limbs.—Tabes is frequently attended with certain remarkable early symptoms, or *prodroma*, which belong to no other spinal affection. Chief among these are sudden pains in the lower limbs or elsewhere. These may either be of a stabbing character, as though a sharp instrument were thrust into the tissues at the spot, which is generally near a joint; or they may be like flashes of lightning or electric shocks shooting down along the course of a nerve. They last but an instant and return again and again during a period of from four to eight days, after which they may disappear altogether for a fortnight, or even for some months. Erb remarks that he has often been struck with the unanimity with which different patients have complained of the "lightning pains" on some particular day

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his feet. But, as Bazire remarked, this is no doubt due to the confidence inspired by looking about one, for nervousness has a marked tendency to aggravate the symptoms of tabes. A similar explanation may apply to the fact noticed by Benedikt and Friedreich, that some ataxic patients who are blind totter more when they are made to shut their eyes.

when there has been much wind, or a fall of snow or of rain. It is also said that they return at spring and autumn, and that they may be brought on by over-exertion, mental emotions, or sexual excitement. They are sometimes agonising, sometimes so slight that careful inquiries are needed to elicit the fact of their presence. They are often called "neuralgic" or "rheumatic." They frequently spread from the legs to the trunk, much more rarely to the arms. They may go on for five, ten, or fifteen years, before any other symptoms show themselves, and they often persist throughout the whole course of the disease. As to the frequency of their occurrence, Topinard found that they were wanting in only 22 cases out of 104, Erb in only 8 out of 60, Cyon that among 203 patients there were 138 in whom they were said to have been present, but no more than 8 in whom they were expressly stated to have been absent.

Visceral pains.—Other early symptoms mentioned by Charcot are pain in the bladder or in the urethra, with a constant desire to micturate, and a sudden pain in the rectum. Certain strange attacks of pain in the stomach, *crises gastriques*, which were first noticed by Delamare in 1866, consist of lightning pains, which start from the groins and pass up both sides of the abdomen towards the shoulders. They are generally accompanied by palpitation of the heart and a quick pulse, by vomiting (of a liquid at first clear but afterwards stained by bile or blood), and by giddiness and malaise. They commonly last for two or three days at a time, and then pass off entirely. They are distinct from the gastrodynia of dyspepsia, to which tabid patients are as liable as other people.

Muscular weakness.—In some cases of tabes the power of the muscles is undiminished. Duchenne invented the dynamometer for the purpose of demonstrating this fact. In the case of certain muscles, a rough estimate of their strength can easily be made. For example, one can measure that of the quadriceps extensor by getting an assistant to fix the lower part of the thigh, and then telling the patient to keep the knee straight, while one forcibly flexes it. Or one can test the force of the psoas and iliacus muscles by placing him in a chair and making him lift his knee, while at the same time one resists this movement by pressing with both hands upon the knee. Or, following Trousseau's plan, one may determine what weight the patient is able to bear upon his shoulders while standing with the support of a friend's arm or leaning against the wall. A young man under his care could support a hundred and sixty pounds in this way and another patient carried a doctor on his back who had supposed him to be paraplegic. But, while the theoretical importance of these observations is very great, as showing that locomotor ataxia may be altogether independent of paralysis, it is a mistake to suppose that all the patients who show a want of muscular co-ordination have their muscular power undiminished. On the contrary, they often experience great fatigue after standing or after walking a short distance; and sooner or later obvious loss of power in the ataxic limbs comes on.

Possibly loss of motor power may be due to a complication of posterior sclerosis, the special lesion of tabes, with chronic and more diffuse myelitis; and this would explain the occasional presence of "girdle-feeling" (*supra*, [illegible] 626). Trousseau speaks of some of his patients feeling as if the chest [illegible] the legs were compressed by an india-rubber cuirass or stocking. Or the sensation may be that of a belt round the waist, of a garter tied below the knee, or of a tight shoe on the foot.

Pelvic symptoms.—The bladder is apt to be irritable in tabes, and this is not infrequently an early symptom. Its muscular power may be diminished, so that the urine no longer flows in a good stream, or is from time to time passed into the bed. Erb notes fæcal incontinence as an occasional symptom, and attributes this to a loss of sensibility in the mucous membrane of the sphincter. The sexual functions are almost always impaired. At an early stage the genital organs are sometimes extremely irritable; and it is said that this may be accompanied with increased virile power; but much more frequently there is marked weakness, and as the disease advances the patient generally becomes impotent. Bedsores do not appear, or not until the last stage of the disease.

One of the most constant symptoms of tabes, first pointed out by Westphal, is *loss of the knee-jerk.* This begins early, and probably continues unchanged throughout the disease. The superficial reflexes are progressively diminished in proportion to the anæsthesia; but they are never absent as in atrophic paraplegia, and are sometimes exaggerated in the early stages. Of the visceral reflexes, that of the cremaster is impaired, along with the loss of sexual power.

The *electrical reaction* of the muscles is often perfect, but sometimes slightly augmented or impaired; they remain well nourished until the disease has reached an advanced stage, and never show the reaction of degeneration.

Ultimately, however, many tabid patients pass into a condition of complete paraplegia with atrophy of the muscles, bedsores, paralysis of the bladder, and cystitis.

In exceptional cases paraplegia is so marked a symptom that they must be described separately as Ataxic Paraplegia of Westphal. In most cases this is of the spastic kind, and depends on sclerosis of the lateral complicating that of the posterior columns, spasmodic tabes of Charcot (*v.* p. 705).

Ocular disorders.—Of the symptoms of tabes, which Gull called *cerebral* and Charcot *cephalic*, affections of the ocular muscles, of the iris, and of the optic nerve are the most important. The most constant is a remarkable disturbance of reflex action of the iris. The pupils are insensible to the stimulus of light, but contract with accommodation for near objects; the recognition of this important symptom of tabes is due to Dr Argyll Robertson, of Edinburgh.

Another common symptom is inequality of the pupils, one of them being constantly smaller than the other. Still more frequently both irides are symmetrically contracted to an extreme degree; this *myosis* is often among the earliest warnings.

One or other of the branches of the third nerve on one side, or the sixth nerve, may sometimes be paralysed, so as to cause diplopia, strabismus, or ptosis. The ocular paralysis may be transitory, or may last a few days, or may persist for weeks or months; it may return again and again; and may at last become permanent.

Impairment of vision is frequent. This usually begins as a progressive narrowing of the visual field, generally from without inwards, until the only part of the retina that remains sensitive to light may be a small patch to the inner side of the blind spot. It is accompanied by achromatopsy; the perception of green being generally the first to disappear, then that of red, and lastly that of yellow and blue. The defects of vision are commonly worse in bright light, so that the patient sees better after sunset.

The ophthalmoscope in many cases reveals atrophy of the disc, which in advanced stages is perfectly white, with sharply outlined edge, and very small arteries. The two retinæ generally suffer together; and the affection often goes on to amaurosis; but even when blindness is nearly complete, the pupils are often permanently contracted. This frequent complication of tabes was first accurately described by Dr Hughlings Jackson.

Erb estimates that, if slight and transitory phenomena are reckoned up with the rest, paresis of the ocular muscles is present in more than half of all the cases of ataxy; and that it is persistent in one third or one fifth. How often the optic discs undergo atrophy is uncertain; oculists are apt to rate it too high, physicians too low; Erb observed it only 8 times in about 70 cases. Topinard found visual disturbances in 49 of 102 cases; and Cyon, amblyopia or amaurosis in 60 of 203 cases. Optic neuritis sometimes occurs at a very early period; according to Charcot, it may precede all other symptoms of ataxy by ten years.

The contracted pupils and the peculiar reflex named after Dr A. Robertson are the most frequent of all ocular symptoms.

Among the less common symptoms is aphonia, sometimes permanent, from abductor paralysis of both vocal cords (Semon, 'Brain,' vol. xvi).*

Arthropathy.—Another most curious occasional complication of tabes—and often a very early one—is an affection of the joints, to which attention was drawn by Charcot (cf. *supra*). Its most common seat is the knee, next in frequency comes the shoulder, and then the elbow, the hip, the wrist. It is not traceable to any blow or other injury. It sets in suddenly with extreme swelling, due not only to effusion into the synovial cavity, but also to infiltration of the parts around. Yet there is neither pain, nor heat, nor redness. In the course of a few months the swelling may disappear, but more often the process goes on to destruction of the articular cartilages with erosion of the ends of the bones and partial dislocation. Charcot says that it never occurs at an advanced period of the disease, except in the upper limbs, when they are beginning to be affected in their turn. This remarkable morbid process differs from Osteo-arthritis in the two important particulars, that it is free from pain, and that it is atrophic not hypertrophic. Its progress, moreover, is rapid, not chronic.

Other trophic lesions occasionally seen in the course of tabes are: pigmentation or leucodermia in the course of the nerves, like that occasionally seen after neuralgia; vesicular eruptions like those of zona; painless ulceration about the fingers and toes which makes the nails fall out; painless loss of teeth, of which we once had a striking case in Philip Ward; and most remarkable of all, ulceration of the sole of the foot, which, still without notable pain, gradually bores deeper and deeper until it reaches the bone. This so-called perforating ulcer of the foot (*mal perforant*) is ill-named, for it does not perforate the foot as an ulcer does the stomach. It is a deep, painless sinus, usually beginning under a corn on the ball of the foot, and leading down to carious bone in the metatarsus.†

Still other complications or sequelæ are early loss of motor power, with spastic symptoms—ataxic paraplegia—described by some authors as a separate disease; secondly, muscular atrophy, either from extension of sclerosis to the anterior cornua, or according to Déjerine from peripheral neuritis; thirdly, cerebral symptoms, such as epileptiform fits, and occasionally hemiplegic attacks; and fourthly, general paralysis of the insane. Certain tabid symptoms, particularly loss of knee-jerk, are exceedingly common in asylums, often without locomotor ataxia. Cystitis, with other pelvic symptoms succeeding the slight affection of the vesical reflex, and complete optic atrophy with blindness succeeding the ordinary amblyopia, are rather ingravescence of normal symptoms than new complications.

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* See also Dr Hale White's case ('Lancet,' 1886, ii, 1128) and one by Dr G. E. Rennie of Sydney, of tabes with bulbar symptoms ('Brit. Med. Journ.,' 1900, i, 500), where he adds references to similar observations.

† This curious condition is met with in cases of diabetes and of leprosy, also no doubt as the result of peripheral neuritis.

Course and event.—Tabes is most insidious in its origin, most irregular, slow, and uncertain in its progress, and most intractable in its course. This is one of the greatest difficulties in tracing its natural history. Patients must be watched, not for a few weeks, but for months and years. They are common in our great hospitals, and by their frequent reappearance in fresh places, and under fresh observers, they give an exaggerated impression of their numbers; but autopsies on cases of tabes are rare.

The enumeration of the symptoms of tabes in the preceding pages has followed a physiological order; motor, sensory, and reflex symptoms, and then affections of the eyes and joints, with other trophic lesions.

In order of time, the earliest symptoms are usually the neuralgic pains; and these are accompanied before long by the characteristic condition of the pupil and loss of knee-jerk. Sometimes slight vesical symptoms are also among the very early symptoms. Ataxia usually follows, and optic neuritis later still. The pelvic symptoms only come on with those of decided paraplegia, and trophic lesions. After lightning pains, loss of knee-jerk, and myosis have lasted for several years there may still be no ataxic symptoms. After marked locomotor ataxia is developed, there may be long delay before the advent of the paraplegic, pelvic, or cerebral symptoms which belong to the latest stage. And after apparently steady, and, by comparison, rapid prorgess, the disease may suddenly be checked and remain stationary for months or years. Still the natural tendency is to ingravescence.

In order of frequency the symptoms of tabes may be thus arranged:—The knee-jerk is always (or almost always) lost, and "lightning pains" are nearly as constant. Next come ataxic symptoms (absent in not more than perhaps 3 or 4 per cent.) and myosis, or loss of accommodation to light. Much less frequent are the various visceral crises, and disturbances of the bladder; and least frequent are the disorganisation of the joints and other trophic lesions above mentioned.

Anatomy.—Todd's statement, that in cases of lack of co-ordination without paralysis a lesion is to be found in the posterior columns of the cord (p. 691), has been remarkably confirmed by modern methods of investigation. The lesion is sclerosis (chronic interstitial myelitis, or grey induration with atrophy of the nerve-fibres) of the posterior columns of Burdach. This change is first visible as a grey streak in the deeper part of the white wedge-shaped area presented by these columns (*fasciculi cuneati*) in transverse sections. The longitudinal distribution of the lesion is in the lumbar enlargement, and afterwards in the dorsal or cervical regions. Sometimes it reaches upwards as high as the restiform bodies. Its histological characters are those which have already been given of sclerosis generally (p. 650). In the more advanced stages most of the nerve-fibres have disappeared, their axis-cylinders being no longer discoverable; but even in the oldest cases a not inconsiderable number of them are found with their structure unaltered, scattered through the dense connective tissue, and can be readily recognised, either negatively by carmine staining, or positively by help of osmic acid. The morbid process extends over the whole of the column laterally, starting from the root-zones and including the posterior roots themselves, which become thin, grey, and atrophic. It also reaches the surface, and the pia mater is generally found opaque, thickened, and more or less adherent. The posterior cornua may be also found atrophied in advanced cases; and the sclerotic change is sometimes found to have spread to the posterior median columns (of Goll) or to the lateral columns; these, however, are probably, clinically as well as pathologically, mixed cases.

Pierret recorded the secondary ascending sclerosis just described, limited to the internal fasciculi of the posterior columns (the columns of Goll) in cases of tabes where the upper limbs have escaped; and believed that sclerosis in the external fasciculi of the cervical posterior columns (the *bandelettes externes*, or *fasciculi cuneati*, Burdach's columns) occurs only when there has been ataxia of the hands and arms, with the other characteristic symptoms of tabes.

We have already seen that Todd distinguished the symptoms of Locomotor Ataxy from Paraplegia, and on physiological grounds placed the disease in the posterior column of the cord. His hypothesis was confirmed in the case he examined *post mortem* (1847). Mr Stanley, of St Bartholomew's Hospital, had described the same anatomical condition in a case of tabes (probably for the first time) in the 'Med.-Chir. Trans.' for 1840.

Romberg described as follows the posterior sclerosis in a typical case which was seen by himself and by Froriep:

> "I was not a little surprised to find that the atrophy of the spinal cord (which, compared with the fresh cord of a man of the same age, fifty-two, was only two thirds of its size) was confined to the lower part of the posterior columns and the corresponding nerves. The medullary tissue (*i.e.* white substance) of the former had almost entirely disappeared, so that they were translucent and of a greyish-yellow colour. The posterior roots of the nerves were deprived of the nervous substance (*i.e.* myelin), and presented a watery appearance." He added that the lumbar and lower dorsal regions were alone affected, and that the anterior columns and nerve-roots were free.

There have been different opinions as to the mode of origin of this lesion, and particularly the old question has been discussed whether the nerve-fibres first undergo degeneration, and irritation of the neuroglia follows? or whether the original process is chronic inflammation of the neuroglia, leading afterwards to atrophy of the nerve-fibres, as cirrhosis of the liver leads to destruction of its secreting cells? At present the evidence is that the first step in the process is degeneration of the neurons, and that the inflammatory sclerosis is secondary.

But the most important question is whether the lesion itself is constant. Trousseau refers to a case of twelve years' duration, which had been seen by Duchenne and accepted as typical; but when Gubler and Luys and Duchenne examined the cord, they could detect no alteration there. Dr Alex. Hughes Bennett published in the 'Clinical Transactions,' vol. xviii, a case which presented the classical symptoms of tabes during life; but the posterior columns were found healthy. The lesion was sarcoma of the bulb and recent cerebral disease, but the posterior nerve-roots ɜ involved. He quoted two similar cases of "peripheral tabes" from ɜrine. The importance of such negative evidence is enhanced by the that the course of the disease is commonly so protracted as to give few ɔces of tracing it to its end in ordinary hospital practice.

Ihe sclerosis is not always obvious to the naked eye, nor until the cord been hardened, so that one can understand how formerly observers ared the cord healthy, when locomotor ataxy had been present during The negative results, however, obtained by competent histologists ı modern methods cannot be easily set aside, and there seems good on to believe that tabid symptoms may depend, not only on the sical lesion of the cord, but also in certain exceptional cases on primary phy of the posterior roots of the lumbar and sciatic plexus. Whether r cases which have been called "pseudo-tabes" are functional neuroses ch only simulate the true disease, or whether they have a peripheral in, are still open questions.

Pathology of tabes.—We have seen that inability to stand or walk dily with the eyes shut is an effect of anæsthesia of the lower limbs. imilar doctrine is held by some authors with regard to the failure of rdination generally. Basing his views upon the acknowledged fact that ling sensations contribute to the due execution of movements, Leyden ntained that ataxy is the consequence of interruption in the transsion of such sensations along the cord.

It must be admitted that strong arguments can be brought forward in ur of this doctrine. One is that the zone of the posterior roots of the e is more or less closely encroached on by the sclerosis. Another is frequency with which a more or less considerable degree of anæsthesia is ent, even at an early period. And if there are cases in which cutaneous ibility appears to be perfect, it may be replied that the sensibility of the cles and tendons and joints was not tested, and that this is no less ntial for the even and regular execution of muscular movements. The that it is impossible to elicit tendon-reflexes, even at the commencement he disease, is another argument in favour of a sensory lesion being nary.

Ihe old notion of a "centre of co-ordination" in the cerebellum or the) led to the theory that the posterior columns possess the special function onveying impulses from this co-ordinating centre above to the motor lei, in the cord. But in the first place impulses pass up, not down, the posor columns, and in the second, co-ordination is not a function for ch a special bit of grey and white matter is told off; it is the result omplicated and elaborate interaction of a thousand impulses and initions, arising in many different regions of the cerebro-spinal axis. The ex movements which are performed by the spinal cord independently of brain are never entirely without co-ordination, and are sometimes coinated in a very complex way, as in the case of decapitated reptiles and ls. One can hardly doubt that the machinery which is brought into ration under such circumstances is also made use of when similar movets are excited by the will.

Quite apart from cutaneous sensation, afferent impulses are certainly veyed to the brain through the posterior nerve-roots from the muscles the joints. This was clearly recognised by the late Sir Russell Reynolds arly as 1855, and formed part of Leyden's hypothesis. It remains the most probable theory of the ataxia of Tabes. It is, however, clear that other parts beside the sensory roots and posterior columns must be involved. Amaurosis from atrophy of the optic discs may precede all spinal symptoms by an interval of several years, so that Charcot believed that the majority of the female patients admitted into the Salpêtrière for simple atrophy of the discs become sooner or later the victims of atary. Complete paralysis of the ocular muscles, of the facial muscles, or of those concerned in mastication, have all been observed in tabid patients.

There can now be little doubt that the primary seat of tabes is in the nervous elements—*i. e.* it is a degeneration of the afferent neurons of the cord. The part specially affected in the posterior columns is the exogenous projection system derived from the T-shaped processes of the oval ganglion-cells on the posterior nerve-roots: the endogenous projection system derived from the ganglion-cells of the cord remains unaffected. The peripheral afferent nerve-trunks as well as their roots are affected, and also their peripheral end-organs—the muscle-spindles in the voluntary muscle-fibres.

The ganglion-cells of the retina, with the optic nerve-fibres, correspond to the ganglia of the posterior spinal nerve-roots (and the Casserian ganglion of the fifth nerve) with the afferent tract from the posterior cornua up to the internal capsule. It is degeneration of the afferent system also which interferes with the iris-reflex, as well as with the knee-jerk and genital reflex.

The upper projection system, the neurons of the cerebral cortex, remain unaffected, and with them the higher intellectual functions: when they are affected, we have the very different clinical picture presented by general paralysis of the insane.

The lower efferent projection system of the anterior cornua is unaffected, and thus voluntary motion is preserved, and there is no muscular atrophy.

The boring and flashing pains, no doubt, are caused by morbid changes in the posterior root-zones; but the exact pathology of Charcot's joint disease remains uncertain.

In the more advanced stages of tabes the degeneration may extend in almost any direction within the cord. Changes in the lateral columns (the crossed pyramidal tracts) were long ago noticed, and explain the spastic symptoms of ataxic paraplegia (p. 705). Charcot first described lesions the nerve-cells of the anterior cornua with atrophy in the correspondi muscles as an occasional complication of posterior sclerosis (cf. *supra*, p. 67 When there has been an absolute loss of power in the lower limbs, almo the whole thickness of the lumbar cord may be found wasted, tough, gre and translucent.

Ætiology.—Among tabid patients the preponderance of males ove females is very marked, but the proportion is variously stated by differen observers, as 8 to 1 (Erb), 7 to 2 (Cyon), 5 to 2 (Carré, Schulze). Eulenburg found in 149 cases, 128 men, including 2 youths under twenty and 3 men over fifty, *i. e.* 6 males to 1 female. Among 83 tabid patients in Guy's Hospital there were 75 men and only 8 women.

Tabes rarely occurs in persons under twenty years old, or begins in those who have passed the age of sixty. Most of the recorded cases in children belong to Friedreich's disease (v. *infra*, p. 706). The case of an old man of eighty is mentioned by Trousseau; but it is not stated how long he had been a sufferer.

Of 83 patients in Guy's Hospital one was under twenty, 3 between twenty and thirty, 27 between thirty and forty, 34 between forty and fifty, and 18 over fifty years of age. But in many of them, especially the older patients, the disease had begun several years before their admission into the hospital.

The disease is not hereditary, nor is it apt to affect more than one member of the same family.*

Tabes has sometimes been preceded by an attack of fever, acute rheumatism, pneumonia, or diphtheria (as in cases recorded by Jaccoud and Erb), and occasionally follows injuries of the spine. In some cases it has been ascribed to a severe chill, as from falling into water, or sleeping on damp ground; to over-fatigue, as in soldiers after forced marching; to prolonged mental anxiety or distress; or, lastly, to venereal excesses—the supposed cause of tabes dorsalis before its symptoms were defined or its anatomy known. The long war at the beginning of the present century seems to have given rise to numerous cases of ataxy among the French and German soldiers. The greater liability of men between the ages of thirty and fifty to these various morbid influences is perhaps a reason why male adults are most apt to suffer from tabes; or perhaps the liability of male adults of the military age to tabes may explain its comparative frequency among soldiers; or, again, the frequency of lues in young men, and in soldiers in particular, may account for both.

Tabes was first definitely connected with *syphilis* by Fournier, who believed that among 102 tabid patients 74 had suffered from that disorder. Erb strongly supported this view, and his statistics gave the proportion 61 in 99; Vulpian found 15 in 20, Buzzard 49 in 100, Gowers 29 in 50, and some writers made the proportion higher still. Thus Ross, among 20 cases of tabes, could only exclude syphilis as an antecedent in a single one. (See 'Trans. Internat. Med. Congr.,' 1881, pp. 32—42.) We must therefore regard lues as a strongly predisposing cause of tabes. Its effects on the afferent system are not those of syphilis either early or late, but it ranks with general paralysis as one of the group of para- or episyphilitic diseases.

Diagnosis.—The early detection of tabes is sometimes difficult. Premonitory pains may be dismissed as "neuralgic" or "rheumatic," but in most cases a careful investigation would elicit other symptoms: a contracted state of the pupils, or an absence of knee-jerk; or the patient may totter a little when told to walk, or to stand with his eyes shut and his feet close together.

When the complaint is fully developed, its diagnosis is generally easy, but pronounced paralysis with spastic symptoms, or with wasting of muscles, may sometimes cause one to overlook the previous existence of ataxy. As in these cases of ataxic paraplegia or tabes with muscular atrophy, so in a case of insular sclerosis, if the posterior columns are particularly attacked, the diagnosis is a double one. We have seen that tabes not infrequently occurs as a first stage of general paralysis of the insane, so as to puzzle those who happen to be less familiar with one than with the other of these two diseases.

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* Carré recorded tabes in a family of which the grandmother, the mother, her seven children, and eight others of her relations were all attacked. But such instances are very rare, and probably belong to an independent malady (p. 706).

The failure of co-ordination which accompanies disease of the cerebellum can scarcely be mistaken for the ataxia of tabes; for it is distinguished by the absence of other spinal symptoms, and by the presence of headache, giddiness, vomiting, optic neuritis, epileptiform attacks, and the various other signs of encephalic lesion. Moreover, the so-called cerebellar ataxy does not cause the peculiar mode of walking above described, but a reeling gait, like that of a drunken man (cf. p. 707).

Prognosis.—Notwithstanding what has been said above of the unrelenting progress of the disease, we must remember, first, that this progress is always slow, often exceedingly slow, and frequently interrupted for an indefinite period; and secondly, that it does not lead to death unless one of the complications above noticed supervene. Probably most tabid patients die of phthisis, because that is the commonest of diseases; some die of disease of the heart; many of pyelitis or consecutive Bright's disease, from vesical complications; others again from the bedsores of paraplegia; and many from common acute diseases which come on accidentally, as pneumonia or bronchitis. Great improvement often takes place under judicious general management, and although patients are not cured of the disease, they are relieved of its most distressing symptoms.

As a rule, the duration of tabes varies between six and twelve years, and it may last twenty or thirty years. Some writers have described an acute form of tabes, and one case was so diagnosed in a woman who died in Guy's Hospital six weeks after admission, and about ten months from the beginning of her illness. But in this instance there was softening of the anterior columns in the lumbar region, as well as sclerosis of the posterior columns.

Erb has recorded two cases, in which almost complete recovery took place. One patient afterwards held a Government appointment for several years; the other, who had been unable to get about without help, and had suffered from incontinence of urine with cystitis, regained the power of walking for three or four hours at a time, and of holding his water for five or six hours; he married and took the command of an ironclad frigate. The present writer knows of another, who was also a patient of Prof. Erb. But without an autopsy the diagnosis can always be doubted by a sceptic.

The course of the disease is not always steadily progressive; it is often quiescent for a long period, and generally it is better in the summer than in the winter. In some cases it scarcely seems to shorten the patient's life.

Treatment.—Tabes is one of the diseases for which no treatment hitherto known is effectual.

The patient should be protected from cold and damp by flannel underclothing, and should avoid bodily and mental strain. Dr Radcliffe thought it advisable that crutches should be used in the early stages, so as to save the lower limbs as much as possible. It has been thought that absolute and continued rest in bed might favour the subsidence of the disease. Weir Mitchell brings evidence in favour of this plan. Gowers recommends a sea voyage. Those who can afford it will find benefit from spending the winter and spring in the South of Europe, or still better in the Azores or the West Indies. Diet has no effect on the disease, and there is no reason to suppose that it is either caused or aggravated by the moderate (or even by the free) use of stimulants. *Abstineat tabidus a venere.*

Erb recommends galvanism applied as a continuous current to the spine. He uses a moderate number of cells, for three to six minutes at a time, once daily. Of sixty-six cases thus treated twenty-five received no benefit, forty-one were more or less improved. He also galvanises the peripheral nerves. In the case above mentioned this treatment was certainly followed by very remarkable benefit.

Locomotor ataxy is among the maladies for which persons resort to the various spas. Most writers are agreed that hot baths are injurious; many believe that cold baths are useless. Erb says that any temperature above 58·5° F. is undesirable, and that the patient should bathe only once in two or three days, and remain in the water not above fifteen or twenty minutes. Nauheim and Rehme are the places which seem at present to have the highest reputation. Peat baths are also recommended. But the figures given by Erb appear to show that the "cold water cure" with packing, friction, and douches is quite as useful; of nineteen patients treated in this way, sixteen are said to have been benefited.

Among the symptoms of tabes, the pains most need relief. Sinapisms, blisters, liniments of chloroform or belladonna, veratria ointment, may be of some use; but often one cannot avoid frequent injection of morphia. Of late years *phenazone* (antipyrin) has been used with much success, and the writer is disposed to believe that this is the most useful application of the drug. *Exalgine* (methyl-acetanilide), recommended by Dr Fraser of Edinburgh for facial neuralgia, is also of service ('Brit. Med. Journ.,' Feb. 15th, 1890). Cannabis indica is sometimes a most valuable anodyne, and so, according to Gowers, is ammonium chloride.

As a specific alterative the drug of most repute was once the nitrate of silver; it often fails entirely, but is still believed sometimes to do great good. Dr Fagge used to order a quarter of a grain three times a day. Belladonna and bromide of potassium seem to be useless, and strychnine to be injurious. Calabar bean, arsenic, and ergot in early cases, may perhaps check the progress of the disease, or relieve some of its symptoms.

When tabes follows syphilis, we should certainly give the patient a fair trial of antisyphilitic remedies. No doubt they often fail, and in the later stages of the disease probably do more harm than good, but two of the writer's cases of tabes which were treated by mercury were decidedly and greatly benefited.

One was that of a man about forty-five, who was admitted into Guy's Hospital with marked ataxy, loss of knee-jerk, and other symptoms; indeed, myosis was the only important one absent. He had the signs as well as the history of syphilis, acquired long before. Under a mercurial course he improved so much that he went out, walking well, free from pain, and in fact cured, though the knee-jerk had not returned. He stated while in the ward that he had some years before suffered from similar symptoms (ten years or so after primary syphilis), and had been cured by the same treatment. A second case is that of a patient who has been under observation for five or six years. He had syphilis years ago and still has occasional tertiary sores on one heel. The worst symptoms of tabes have disappeared under perchloride of mercury, but he still has minutely contracted pupils and loss of knee-jerk. Neither case is conclusive, but this method is well worth trying when we know that syphilis preceded the nervous symptoms.

Among more recent methods "extract of spinal cord" and "extract o testes" have been employed in tabes as in other incurable diseases.

A more rational plan of treatment is that carried out by Dr Fränkel a series of muscular exercises, by which the muscles and nerves are educated and the power of co-ordination preserved or even restored, so that patients have sometimes regained the lost power of locomotion.

Mechanical stretching of the great sciatic nerves has been used as a remedy for tabes. It was first performed by Langenbuch with apparent success; but when the patient died, the cord was found healthy. On the whole the good effects seem to be doubtful, and the drawbacks and dangers undoubted. See an interesting paper by Dr Cavafy, with eighteen cases, beside one of his own ('Brit. Med. Journ.,' Dec. 10th, 1881); also Mr Bowlby's record of cases (*loc. cit.*, pp. 360—368).

Another somewhat analogous plan of treatment was extensively tried a few years ago. It consists in suspension of the patient by the head, so that the weight of the body pulls upon the attachments of the cord to the spinal column by its membranes and nerve-roots. This method was introduced by Dr Motschukowsky, of Odessa, and has been tried on a large scale by Charcot and other physicians on the Continent, in England, and in America. Great care is needed in carrying it out, for in more than one case it has caused death, and in one of these the autopsy showed the absence of posterior sclerosis.

Many instances have been recorded in which the lightning pains of tabes have disappeared under this treatment, while faculty of locomotion and virile power have returned. But in many more these effects have been absent or transient; and the spontaneous variations in the symptoms of this disease during its slow and irregular course are so great, that improvement in symptoms, and particularly in subjective symptoms, must be accepted with great caution as proof of the efficacy of this or of any other plan of treatment. A patient on whom the writer carried out suspension said that he was greatly benefited; but the improvement was very doubtful to those who watched the case, and he had been equally gratified by the good effects of a few grains of salt, given as a control experiment while his symptoms were being recorded. In the absence of satisfactory evidence of the value of the method, interest attaches to experiments made on the dead body, which show that its effect is to straighten the vertebral curves, and to relax rather than stretch the spinal cord.* It may possibly have some effect on meningeal adhesions, possibly some on the circulation of the cord; but if so we cannot say beforehand whether either would be beneficial or the reverse. On the whole, it seems likely that the method of suspension will be finally abandoned as useless and dangerous.

Since the above paragraphs were written, in 1888, further experience has confirmed the view then expressed of the uselessness of the various specific measures of treatment of Tabes, with the exception, and that only in a minority of cases, of a mercurial course, when syphilis has preceded the disease. In a recent article by Eulenburg (in the 'Deutsche med. Wochenschrift' for Oct. 28th, 1897) he insists on the very chronic and unequal course of the disease as a perennial source of fallacy in estimating the efficacy of new methods of treatment. He is quite clear against all treatment by antiphlogistics and counter-irritants, and against the value of silver nitrate. He is wisely dubious as to the effects of electricity and hot and cold baths, sceptical as to mercurial treatment, and hostile to uspension; but believes that the subcutaneous injection of so-called 'spermin" (Brown-Séquard's fluid) is sometimes followed by improve-ent!

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* See Dr Cagney's paper, 'Med.-Chir. Trans.,' Jan., 1890.

ATAXIC SPASTIC PARAPLEGIA.—Cases in which locomotor ataxia is com-ined with spastic paraplegia have been described by Westphal, Charcot, nd other writers. The ataxic symptoms are not accompanied by the light-ing pains, or the girdle-feel, or the anæsthesia and paræsthesia of tabes; or do these cases present the ocular symptoms—ptosis, strabismus, optic euritis, nystagmus, or "the Argyll-Robertson pupil." The knee-jerk, in-tead of being lost, is markedly increased, and clonus is present. Lastly, here is weakness of the legs, and sometimes of the arms, ending in com-lete paraplegia with pelvic symptoms. The cause of these symptoms is uite unknown; and it is observable in such cases that there is no remark-ble connection with lues as in cases of Tabes. Gowers, however, admits hat there are cases intermediate between this ataxic paraplegia and rdinary Tabes.

We may consider the disease as a combination of some of the symptoms f tabes with those of lateral sclerosis, and in the cases which have been xamined after death there has been found extensive sclerosis of the lateral nd posterior columns without affection of the posterior nerve-roots and heir adjacent zones. The change, also, more often extends above the umbar enlargement than in tabes, and it is far from constant in its lateral xtent, so that it sometimes approaches the distribution of a diffuse rather han of a columnar ("systemic") or of a disseminated ("focal") disease.

The absence of sensory symptoms and the persistence of the knee-jerk epend, as Gowers remarks, on the exemption of the afferent lower neuron. f the knee-jerk is not lost early in the course of spinal sclerosis it is not st at all; and if it is lost early, spastic symptoms do not afterwards come n—*i. e.* it is lost by breaking the reflex arc in its afferent half, and cannot restored by a subsequent stimulation (or diminished inhibition) of the otor half.

Together with the intermediate cases mentioned above, this ataxic astic paraplegia forms a connecting link between typical Tabes and iedreich's hereditary tabes, which will next be noticed.

The clinical recognition of this variety of spinal sclerosis is not diffi-lt. Its prognosis is less grave than that of cases of ordinary tabes, ice its advance is slower and the occurrence of pelvic symptoms much nger postponed. The patients also are free from the distressing neu-gia and amblyopia of tabes. But no treatment is known which avails removing their symptoms.

FRIEDREICH'S DISEASE.*—Nicolaus Friedreich first described in 1863 irchow's Archiv,' vols. xxvi and xxvii), and more fully in 1876, an *editary* (or rather "*family*") form of tabes which presents several pecu-ar features. He recognised it in only three families; in each of them it tacked in succession several children of the same parents, in all nine tients, of whom seven were girls. It began much earlier than ordinary bes, at or near the age of puberty, between the thirteenth and the eighteenth years. Among eleven cases Rütimeyer found seven boys and four girls, the ages being all under puberty (1883). Since then further observations have shown that this form of tabes occasionally occurs in two generations, much more often in several brothers and sisters; that it begins in childhood as early as four, at puberty, or in early adult life as late as twenty-four; and that girls are almost as often affected by it as boys. Isolated cases also occur.

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* *Synonyms.*—Hereditary ataxy—Juvenile ataxia—Hereditary tabes in children—Here-itary ataxic paraplegia (Gowers)—Early tabes with nystagmus occurring in families.

The disease is attended with remarkably little disturbance of sensation; the prodromal pains in the limbs and the later girdle-sensation round the body are absent or slight, and the pupils contract normally.

A peculiar form of nystagmus, however, is sometimes present; it is characterised by occurring only when the patient endeavours to fix his eyes upon an object before him; the movements are always bilateral, and are comparatively slow, being repeated two or three times in a second.

Ataxia begins in the legs, but the upper limbs show a loss of co-ordination at an early period. There is occasionally curvature of the spine or some form of club-foot present as a complication.

In every instance there has been a remarkable disorder of speech. The utterance is at first, according to Calloud, slightly lisping; it then becomes stammering, but not as observed by the present writer slurred and almost unintelligible, as in Dementia paralytica; nor is it slow and accentuated as in multiple sclerosis. The skin and joints are unaffected. Knee-jerk is absent. The senses and the mental faculties are perfect.

The duration of these cases has been very long; in one instance it was more than thirty years.

Choreiform movements of the face and limbs have been observed sometimes as the first symptom, so that the disease has been mistaken for chorea. Clinically it resembles insular sclerosis or functional spasmodic disorders as much as it does ordinary tabes.

After death sclerosis of the posterior columns, including the root-zones and nerve-roots, has been found by Friedreich and subsequent observers; but the direct cerebellar tract and the internal posterior column of Goll have also been affected. In some cases of hereditary ataxia the cerebellum has been found wasted, but probably these should be entirely separated from those of Friedreich's disease. In two cases (Friedreich and Kahler and Pick) the entire cord was remarkably small.

I have at present (1880) under observation a typical example of Friedreich's ataxy. The patient, a medical man, was first noticed to be uncertain in his gait when about sixteen years old. He was one day walking across a plank in a house that was being built, when he tottered; afterwards he remembers that he had to be careful not to go near the edge of the path where it was raised above the road. But he came to the hospital as a student, passed his examinations, went into practice, and took a wife. At the age of about twenty-five he was obliged to give up his profession on account of increasing inability to walk or ride; he became unable to write, and his speech became laboured and thick. In 1878, when he was thirty, he could just walk from one room to another with assistance; there was even then but little impairment of sensation in his feet; when he moved his lips in speaking a large number of the facial muscles were thrown into action, so that the mouth became drawn outwards into a meaningless smile; the words were tolerably distinct, but some syllables were slurred over, and others pronounced too strongly. The nystagmus was very obvious when he was told to direct his eyes to either side. He is a member of a highly neurotic family, and one sister suffered from some form of paralysis, but whether her case is like his I do not know.—C. H. F.

We once had three brothers in Guy's Hospital suffering from this juvenile family type of ataxia with nystagmus. The case of one of them is reported in the forty-fourth volume of our 'Reports' (1887), and is identical with one of the five cases in the same family recorded by Dr Gowers in the fourteenth volume of the Clinical Society's 'Transactions.' One of them died under the late Dr Moxon's care. Sections of the cord were made by Dr Pitt, and are described and figured by him in the article referred to. They showed sclerosis of the posterior columns (both Burdach's and Goll's), with similar change in the crossed pyramidal and cerebellar tracts. The anterior columns, which also have been sometimes found affected, were free, and the grey substance normal. The lesion was most marked in the cervical and dorsal regions, but the cord in its whole length was remarkably small, as if from congenital atrophy.

Dr J. A. Ormerod published a critical digest of the recorded cases of this remarkable form of disease in 'Brain' for April, 1884; and Dr Bury in the ninth volume of the same journal. Dr Everett Smith (quoted by Gowers) has collected no less than fifty-seven cases in the 'Boston Medical and Surgical Journal' for Oct. 15th, 1885.

The progress of Friedreich's ataxia, like that of tabes, is very slow, but very sure. No treatment is of any service except for the temporary relief of symptoms.

Hereditary cerebellar ataxia.—In 1893 Marie described in the 'Semaine Médicale' a series of cases, some observed by himself, others already recorded by Fraser ('Glasgow Med. Journ.,' 1880), Noune ('Archiv f. Psych.,' 1891), and Sanger Brown, of Chicago (in the fifteenth volume of 'Brain,' 1892), and proposed to class them together under the above title. They resemble cases of Friedreich's ataxia in being hereditary—or rather in occurring in families, co-heral or collateral in distribution. The gait, however, is not the staggering gait of tabes, but the reeling gait of cerebellar disease, the knee-jerk is increased instead of being diminished, and there are no deformities, like the spinal curvature and club-foot above described.

In the few cases which have been examined after death, the cerebellum has been found atrophied, not hardened; and in none has sclerosis of the postero-lateral columns of the cord been present; but other lesions of both brain and cord have been observed.

The disease begins gradually in young adults of either sex. Beside the vertigo and spastic symptoms, there are frequently choreiform movements, dysphagia, and disturbance of accommodation of vision and of colour-perception, but neither Robertson's pupil nor optic neuritis.

The diagnosis from ordinary tabes and from Friedreich's ataxia depends upon the above characters, that from tumour of the cerebellum upon the absence of headache, vomiting, and optic neuritis.

Beyond the collateral distribution the ætiology is unknown, and the prognosis is that of slowly ingravescent progress towards a fatal issue. The reader is referred to an excellent account of this curious malady by Dr Ormerod (in 'Brain,' 1895), and to the articles by Grainger Stewart and by Ferrier in the seventh volume of 'Allbutt's System' (pp. 157 and 384).

INSULAR SCLEROSIS

"Tremor oritur a spiritibus animalibus per apertas valvulas in oppositos musculos influentibus, qui spiritus . . . istas non claudunt; ut ventus semiapertam januam non claudit."—JOH. DOLÆUS, 'Medica dogmatica,' 1691.

History—Symptoms—Anatomy—Ætiology—Diagnosis—Prognosis and treatment—Comparative frequency of diseases of the spinal cord in hospital practice.

Synonyms.—Multiple cerebro-spinal sclerosis—Disseminated sclerosis.—*Fr.* Sclérose en plaques (Vulpian)—Sclérose en îlots multiples et disseminés (Liouville).—*Germ.* Multiple inselförmige Sclerose (Leube)—Herdweise Sclerose.

In Cruveilhier's 'Atlas of Pathological Anatomy,' published between 1835 and 1842, there are figures taken from four patients which represent the spinal cord affected with "grey degeneration," in the form of patches scattered irregularly through its substance. In two of these cases he also gave the clinical features of the disease. About the same time Carswell figured the same lesions in his 'Illustrations of the Elementary Forms of Disease' (1838), and Marshall Hall described the characteristic symptoms in his 'Diseases of the Nervous System' (1841). Although from time to time similar cases were recorded, it was not until 1866 that a series of observations made at the Salpêtrière proved that the lesion in question is clinically marked by a remarkable group of symptoms, so that it can often be recognised during life. These researches were made by Charcot, Vulpian, and Bouchard. The subject was soon taken up in Germany, and in England the late Dr Moxon published a case in the 'Lancet,' 1873, and a paper containing eight cases in the 'Guy's Hospital Reports' for 1875.

Cruveilhier's drawings show that the bulb and the pons are affected as well as the cord, and the same morbid change has since been found in the hemispheres. Insular sclerosis might therefore with equal justice be placed among cerebral diseases; indeed, several of its symptoms are cerebral. Hence its most appropriate place seems at present to be betwee diseases of the cord and those of the brain.

The slow and hopeless course of the disease accounts perhaps for not having been earlier recognised in the wards of ordinary hospitals; ma cases were probably discoverable in workhouses and asylums. Dr Fag was unable to find a single case in the *post-mortem* records of Guy's H pital between 1854 and 1873; but in the ten years 1880 to 1889 inclusive had 12 cases, and since 1889 till now (1899) 36.

Symptoms.—That the clinical features of disseminated sclerosis sho vary in different cases is no more than we should expect from the irreg distribution of the patches. Charcot spoke of it as polymorphous in its clinical aspect. He with other French writers describes, in addition to the "cerebro-spinal" form of the disease, separate "spinal" and "cerebral" and "bulbar" forms. However, notwithstanding such variations, the symptoms present a degree of uniformity which is remarkable. This uniformity Dr Moxon regarded as "a constant average result of the numerous points of disease;" but the explanation leaves much unexplained.

We may refer disturbance of the special senses to sclerosis of their nerves, spasms to implication of the lateral columns, and mental symptoms to cerebral lesions; but neither in this disease nor in tabes could we have predicted the symptoms from the anatomical lesions.

Early prodromal symptoms are sometimes observed which, according to Dr Buzzard, closely simulate hysteria—loss of power or numbness in one limb, pins and needles, etc.

The first characteristic symptom is a peculiar *tremor*, which was formerly confounded with paralysis agitans. There are, in fact, records of two cases diagnosed as examples of that affection by Skoda and by Hasse respectively, in which patches of sclerosis in the nervous centres were discovered after death. In paralysis agitans, however, the trembling movement is less extensive, more regular, and more rhythmical than in insular sclerosis; it goes on at rest, and is not increased by voluntary movements. When a patient with insular sclerosis lifts his hand, the oscillations increase in amplitude as it is raised, until they become violent as well as disordered. If he is asked to carry a cup to his lips it is more and more shaken until at last it is dashed against the teeth, and its contents are spilt. In slight movements the tremor is not so marked; and the handwriting, although shaky, long remains legible.

There is no agitation of the muscles as long as they are at rest and supported. When the patient is lying in bed one could not tell that anything is the matter; but as soon as he is asked to sit up, the arms, and then the head and neck, begin to oscillate. When he is resting in a chair the arms are quiet, but the head may still continue to show a slight tremulous movement. If he attempts to get on his feet the whole trunk and all the limbs become violently shaken, so that in severe cases standing is out of the question. Mental emotion or excitement greatly aggravates the tremor. The tremors of insular sclerosis are earliest and most marked in the arms, next in the head and neck, next in the legs, and lastly in the trunk. The facial muscles are seldom or never affected with these tremors. The tremors are really slow—not above 6 or 8 in the second; but as Dr Pitt remarks, they seem to be quicker from the amplitude of the movements of the hand.

Secondly, *nystagmus* is commonly present. It, however, differs somewhat from the ceaseless oscillation of the eyeballs with which ophthalmic surgeons are familiar. As Dr Moxon pointed out, it is absent so long as the eyes are at rest, showing itself only when they are directed to an object. In this particular it agrees with the "intention tremors," as the German writers call the movements described in the last paragraph. It also agrees with the nystagmus observed in Friedreich's hereditary ataxy (see p. 707); so that both Friedreich and Erb thought that the anatomical lesion might prove the same.

A third characteristic symptom of disseminated sclerosis is a peculiar *affection of the speech.* The pronunciation is slow and monotonous, as if the utterance of each syllable was an effort. With this drawling, interrupted, or *staccato* speech, the separate syllables are, with few exceptions, distinct. Charcot found the letters L, P, and G most apt to be slurred over; Dr Pitt thinks the difficulty, if any, is with G, P, R, and D. German writers describe the articulation as "scanning," each syllable being pronounced separately. In advanced stages speech may become quite unintelligible. The voice is pitched in a monotone, and so is remarkably wanting in the cadence which gives expression. Leube found with the laryngoscope that the vocal cords, although capable of closing the glottis, were yet able to relax, and liable to changes of tension ('Deutsches Archiv,' 1871).

In addition to these more distinctive symptoms, there are others which belong to disseminated sclerosis in common with other diseases of the nervous system.

The gait is often from an early stage *spastic* in character. The cutaneous reflexes are usually normal, but sometimes they are increased, knee-jerk is exaggerated, and clonus often present. The extension-plantar reflex, first noticed by Babinsky in 1898, and described above (p. 652), is present. In the later stages there is a tendency for the lower limbs to become rigidly extended and adducted—a condition at first transitory, but afterwards permanent. Moxon mentions that one of his patients was obliged to sit in an elbow-chair, so that when her legs stiffened she could hold on to the arms and save herself from sliding down on to the floor. Such early contractions are painless, but at a later period a painful flexion of the limbs is not infrequent. In ordinary cases there is no reaction of degeneration, and the muscles react to both currents until they finally undergo atrophy.

Subjective sensations of *numbness* and other paræsthesiæ are not uncommon, although they were absent in Moxon's eight cases; anæsthesia, or impairment of tactile sensibility, can seldom be made out, but was present in a case of Dr Pitt's.*

Symptoms of locomotor *ataxia* are now and then present in disseminated sclerosis; this is supposed to depend upon the existence of patches in the posterior columns of the cord.

After a long time *paraplegia* usually develops itself, but the functions of the pelvic organs are scarcely affected. Constipation is frequent, but there is no incontinence of fæces, the sexual organs retain their powers, and there is no retention of urine until the latter stages of the disease.

Among other *cephalic* symptoms amblyopia often occurs, with narrowing of the field of vision and achromatopsy, and transient *diplopia* from ocular paresis is not infrequent at an early stage of the disease. Charcot observed that the impairment of sight rarely goes on to complete amaurosis, even when the optic nerves have been found sclerosed in their whole thickness; and he referred this to the persistence of naked axis-cylinders in the midst of the diseased tissues. The optic discs are often found altered, and when there is total blindness they have been found in a state of white atrophy. Any degree of optic neuritis with disturbed vision may be an important indication of an early stage of insular sclerosis. The pupils are unaffected throughout the disease; but headache, giddiness, and sleeplessness are not infrequent.

As the disease slowly advances, the *memory* and *intelligence* become gradually impaired, and the patient sometimes bursts into uncontrollable laughter, or cries without cause. The mental condition most often observed is, however, rather the self-satisfaction of paralytic dementia, or the contented apathy of myxœdema, than either melancholy or irritability of temper. Patients with insular sclerosis always say they are better.

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* 'Guy's Hosp. Gazette,' May, 1900, pp. 209 and 259.

Charcot noticed a peculiar *facies;* the expression vague and uncertain; the lips drooping and half opened; the features wearing an air of dulness or stupidity. In certain cases the *délire des grandeurs* and other symptoms of general paralysis develop; in others the mental condition passes into melancholia, with refusal of food. Brissaud believes that the uncontrollable laughter which marks some cases of insular sclerosis points to patches in the anterior part of the thalamus.

A curious feature, which is observed in about one fifth of the cases of disseminated sclerosis, is the occurrence of attacks of stupor, followed by transitory *hemiplegia.* These apoplectiform seizures are attended with flushing of the face, a rapid pulse, and a temperature raised to 104° or even 106°. The coma lasts for a day or two, and then passes off into sleep. Sometimes, however, a seizure of this kind is directly fatal; one of Moxon's patients died so in about twenty-four hours. Such an attack, even when it is recovered from, seems always to lead to aggravation of the general symptoms of the disease.

Lastly, in some exceptional cases atrophic paralysis affects the upper or lower limbs, the face, or the trunk. At the same time their electrical excitability, which otherwise remains normal, is lowered; and the natural reaction is replaced by the reaction of degeneration. Charcot says that the characteristic symptoms of bulbar paralysis sometimes show themselves, and so dysphagia or paroxysmal dyspnœa may be the immediate cause of death.

It is obvious that many of the above symptoms of insular sclerosis are due to the disease invading the lateral or the posterior columns, the anterior cornua or the nuclei in the bulb and cerebrum, so as to produce the symptoms which belong to tabes, to spastic paralysis, or to atrophic, spinal, or bulbar palsy respectively. The final paraplegic period, again, with bedsores, retention of urine, and other pelvic symptoms, belongs to the last stage of all chronic disease of the cord.

Anatomy.—As a rule the morbid change is at once obvious when the nervous centres are examined in the *post-mortem* room. Bourneville, however, recorded one case in which they appeared to be healthy until the microscope was used. The patches of sclerosis are rounded or elliptical or irregular in form. In the cord they come to the surface, and are sometimes seen through the pia mater. In the brain they affect the white substance of the hemispheres, and scarcely ever penetrate into the cortex, but they are often visible on the surface at the base. In the basal ganglia, the pons, and the cord, they show no tendency to spare the grey matter, or to affect one column or cornu more than another. Hence Insular Sclerosis is not a "systemic" disease in its distribution, nor focal, nor transverse, but essentially indiscriminate or scattered.* The grey

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*** See an excellent plate in Dr Bramwell's work, which shows the distribution of the patches of sclerosis throughout the cord in a well-marked case, and the way in which they stain with carmine. This is an example of the larger sized islands. In Moxon's paper in the Guy's 'Reports' (1875) is a coloured drawing of the centrum ovale, with smaller sclerotic patches, and a microscopic section of the cord.**

patches are often scattered in large numbers throughout the white matter of the hemispheres, the pons and thalamus; but the cerebellum seldom contains many. A similar change is frequently present in the optic nerves, the olfactory bulbs, and the roots of the various cranial and spinal nerves. In the peripheral trunks it has only occasionally been recognised: Moxon found the brachial plexus in one case normal on each side.

The oldest patches feel hard, but recent ones are softer than the rest and of a reddish grey tint, pinkish when exposed to the air, but gradually become yellowish grey and less translucent. They may either project somewhat above the cut surface, or lie at the same level, or be slightly depressed, the first condition being probably the most recent and the last the oldest. They vary from a microscopic minuteness up to the size of a hazelnut or more. Histologically they present the characteristic appearances of chronic interstitial or grey degeneration. Charcot believed that naked axis-cylinders persist; and their persistence, after having long lost their sheath of myelin, is confirmed by several recent observers. The neuroglia is highly fibrillated and felted together.

The affected parts are found not to be so sharply defined at their edges as they appear to the naked eye; on the contrary, the sclerosis fades off very gradually into the healthy tissue. Moxon remarked that granule-cells are more numerous at the circumference of the patches (and even in the apparently normal brain-substance beyond) than towards their centres. Deiters's cells and corpora amylacea are present, as in other forms of sclerosis.

Pathology.—The nature of the change in the nervous centres is apparently the same as we have described in spastic paraplegia and in tabes; but the distribution is different. The lesion is not columnar or systemic in transverse distribution, nor is it confined longitudinally to the lumbar, dorsal, cervical, or bulbar part of the cord, but is present in all these regions, and also, unlike the other scleroses of the cord, in the pons and in the white substance of the brain.

The same question which has met us in other cases arises here. Does the morbid process begin in atrophy of the nervous element, or in interstitial inflammation of the neuroglia? In this case, unlike that of tabes, it is still generally held that it begins in the neuroglia, and probably in the lymphatic sheaths of the blood-vessels. Marie conjectures that the disease is toxic, and affects the nervous system through the blood supplied to it; while Nothnagel and Strümpell regard the patches as being really neoplastic, not inflammatory or degenerative,—in fact, gliomata.

Why such disseminated lesions should cause so uniform a set of symptoms is not clear; but the symptoms are not so uniform or so unique as was at first supposed. Nystagmus is present in other diseases, the mental symptoms are associated only with cerebral distribution of the sclerotic insulæ, and spastic symptoms only accompany implication of the lateral columns.

Charcot suggested that the peculiar tremors depend upon the persistence of naked axis-cylinders in the parts affected with the sclerosis, and conceives that the transmission of volitional impulses may become interrupted and, as it were, jerking. Ordenstein is disposed to think that the tremor depends in some way upon the presence of patches in the pons and still higher in the motor tracts, and Erb says that an analysis of twenty-two recent cases tends to support the notion of the connection of this symptom with abundant islets in the mesencephalon.

The natural allies of insular sclerosis are dementia paralytica and spastic paralysis.

Ætiology.—Disseminated sclerosis is now ascertained to be equally frequent in both sexes, although Charcot found twenty-five females to nine males among the first thirty-four cases which he collected. The age at which it most frequently develops itself is between twenty and thirty-five, but it has been several times observed at puberty, and instances of its occurrence in childhood have been recorded. It has seldom been known to begin in persons over forty years old. Now and then it has occurred in two children of the same parents.

Like other chronic affections of the cord, it has been ascribed to "chills," to falls and blows on the head, and to moral shock, mental excitement, or, as it is now called, "worry" and "overstrain."*

Charcot cited instances in which the disease began during convalescence from enteric fever, cholera, or smallpox, and it may or may not have been preceded by syphilis. It has also been ascribed to influenza and to plumbism. All that we can conclude is that these several antecedents do not *protect* a person from insular sclerosis.

Diagnosis.—This is not always easy, and in some cases impossible, especially in the earlier stages of the disease, when the characteristic triad of symptoms is absent. Tremors, precisely like those which are present in this disease, may occur not only in mercurial poisoning (when the history will in most cases remove all doubt), but also when the lesion is a "chronic cervical meningitis with cortical sclerosis," and even where it is a "primary or consecutive sclerosis of the lateral columns." The presence or absence of the other characteristic symptoms of these affections must guide the observer to a decision so far as they are concerned.

The diagnosis from paralysis agitans is not the most difficult, for that disease occurs in older patients, the tremors continue when the muscles are at rest, and affect the fingers more than the arms, and the trunk more than the head; their amplitude is smaller and more uniform, and their rate is generally quicker. Moreover, they are not originated or much increased by the patient's voluntary attempt to move. In paralysis agitans, again, the shake precedes the palsy, whereas slight loss of power in walking is often the earliest symptom of insular sclerosis, as in a typical case recorded in 1875 by Dr Goodhart, in a woman of thirty-eight, where characteristic lesions were found after death ('Path. Trans.,' xxvii, p. 17).

Diagnosis of insular sclerosis from tabes, from Friedreich's ataxia, from spastic paralysis and bulbar palsy, probably means only that in mixed forms of disease we must distinguish clinically the characters of one type which are complicated by some usually associated with another. Corresponding coincidences of posterior, lateral, anterior, cornual, or bulbar sclerosis with disseminated cerebro-spinal sclerosis have been found *post mortem*.

The usual tremors are occasionally absent, and not only in the later stages of the disease, as Charcot supposed, for Erb refers to several cases in which they were wanting throughout the whole course of the disease. He cites Westphal and Killian, who recorded instances to the same effect.

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* Bauwinkel records a case in which a man fell into water and allowed his clothes to dry upon him, three days before the first symptoms of insular sclerosis appeared.

Occasionally cases which during life presented the characteristic speech, nystagmus, tremors, and increased knee-jerk have after death failed to show insular sclerosis of the brain and cord. Some of these were probably cases of *dementia paralytica* (general paralysis of the insane);* others of cerebral tumour. But others of such cases recorded by Westphal and Strümpell were certainly without any lesion of brain or cord at present discoverable. They are, however, so rare as scarcely to disturb a diagnosis. It is of much greater practical importance to be on one's guard against mistaking *hysteria* for disseminated sclerosis in women. Moxon saw several patients about whom he had for a time been in doubt, but who completely recovered. See also Dr Buzzard's instructive address before the Neurological Society on the "Simulation of Hysteria by Organic Disease of the Nervous System" ('Lancet,' Jan. 23rd, 1890, and 'Brain,' Feb., 1890). Perhaps the most characteristic symptoms of insular sclerosis as against hysteria are Babinsky's extension-reflex and the presence of optic neuritis.

The writer had a remarkable case of acute paraplegia in a young woman under his care in Guy's Hospital in November, 1896. She had shortly before been admitted under Dr Taylor with symptoms of acute meningitis: pelvic symptoms speedily appeared, and then spastic characters developed. The case became chronic, but after many weeks, lateral nystagmus, *staccato* speech, and intention-tremors gradually appeared, and the case became clinically one of insular sclerosis. Finally the paraplegia and these later symptoms diminished, and she at last recovered.

Course, prognosis, and treatment.—This remarkable disease usually begins insidiously, with slight giddiness, affections of sight or hearing, or with weakness in the legs and difficulty in walking. As an exception it may commence more suddenly, perhaps with an epileptiform fit.

The course of insular sclerosis is irregularly but slowly progressive, although remissions in the symptoms may occur spontaneously or under treatment, and last for months. The average duration of the disease is said to be from five to ten years, the most rapidly fatal course about one year from the commencement, and the longest seventeen years. This, however, applies to cerebro-spinal cases; those in which spinal symptoms alone ar present may go on for twenty years.

If death does not occur in one of the ways already mentioned—he plegia, epilepsy, paralytic dementia, or paraplegia with bedsores and cyst —it is generally caused by some intercurrent disease, such as pneumo pleurisy, diarrhœa, œdema of the glottis, or, most frequent of all, phthi As a rule, cases of insular sclerosis are less protracted than those of tabes

Few remedies have been found of any service. The administration strychnia or of nitrate of silver has sometimes appeared to diminish tremor and the weakness of the lower limbs, but only for a time. S physicians have seen transitory benefit follow the cold water treatm galvanism, or the subcutaneous injection of arsenic. Many of the dr given probably do harm. The only rational treatment is to alleviate i vidual symptoms, and to make the patient as comfortable as he can made—particularly to keep him warm. The few cases like that menti above which ultimately recover, if the diagnosis is admitted to be cor do not seem to have been decided by any special line of treatment.

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* See Dr Bristowe's cases in his collected papers on diseases of the nervous syste

Before passing on to our next subject, Diseases of the Brain, it may be of interest to give the reader some notion of the relative frequency of the Diseases of the Spinal Cord which we have been describing, as seen in the wards of a large London hospital. For the first set of statistics the writer is indebted to his former house-physicians—Dr Halstead, now of Ramsgate, and Dr E. W. Goodall—for 1880—1889, and to these he has added the statistics of the hospital registrar for the following nine years (1890—1898).

Among 301 consecutive cases of diseases of the spinal cord there were 167 of *paraplegia*—105 men to 62 women.

These might be classified as follows, on the plan adopted in the chapter with that heading.

(*a*) Paraplegia due to intrinsic causes: 78 cases, 25 with symptoms of acute *myelitis* (probably in two or three cases hæmorrhage into the meninges or cord), and the remainder chronic cases with more or less marked characters of *spastic paraplegia.*

(*b*) Paraplegia due to extrinsic causes: from compression by *caries* of the spine, 22 cases; from *cancer* of the spine, 3. Paraplegia due to *tumours* of the cord or meninges: 4 cases—tubercle of the cord, glioma of brain and cord, and two meningeal tumours.

(*c*) Eighteen cases of paraplegia were diagnosed as due to *syphilis*: 12 were *alcoholic*, probably not all of them due to peripheral neuritis, and 30 were classed as *hysterical* or *functional*. One of the last, in a man under the writer's care, was a case of diver's paraplegia.

There were no instances recorded as Landry's paralysis, nor as hemiparaplegia. Cases of lead palsy and of alcoholic paralysis were numerous, but were not classified among other forms of paraplegia; and many of them were treated as out-patients.

There were 39 cases of *atrophic paralysis*, 25 male and 14 female. Of these, 3 were due to chronic cervical meningitis, and were thus pathologically distinct from the rest; only 15 were cases of infantile paralysis, but many more were treated in the out-patient room; there were 2 of spinal atrophic paralysis in adults, 11 of progressive muscular atrophy, 1 of bulbar palsy, and 8 of pseudo-hypertrophic paralysis in children.

There were 83 cases of tabes, including 2 of Friedreich's disease, of which 75 occurred in men. Lastly, there were 12 cases of Insular sclerosis —5 in men and 7 in women.

The statistics for the subsequent years (1890—1893 inclusive), also taken from the 'Clinical Reports' of Guy's Hospital, are as follows:

In these nine years there were 208 cases of Paraplegia (including about 30 regarded as functional or hysterical; 20 of Progressive muscular atrophy in adults, including two of bulbar palsy; 11 of pseudo-hypertrophic paralysis, beside 10 others of primary muscular atrophy without previous apparent hypertrophy. Lastly, there were 127 cases of Tabes, and 36 of Insular sclerosis.

DISEASES OF THE BRAIN

CEREBRAL DISORDERS DUE TO LOCAL LESIONS OF THE ARTERIES

> Ἀποπληξίη ὅλου τοῦ σκήνεως [*i.e.* σώματος] καὶ τῆς αἰσθήσεώς τε καὶ γνώμης καὶ κινήσεώς ἐστι παράλυσις. Ἢν δὲ κατάρχῃ κεφαλὴ, ἐπὶ μὲν τοῖσι δεξιοῖσι τὰ λαιὰ παραλύεται δεξιὰ δὲ ἐπ' ἀριστεροῖσι.—ARETÆUS, A.D. 70.*

> "Paralyticos plerumque altero latere captos spectamus. Hæc hemiplegia vocatur. Fertur, et sane plurimorum jam medicorum observationibus confirmatur, latus adversum ab eo in quo cerebri vitium est, sic resolvi."—GREGORY'S *Conspectus*, 1832.

Anatomical effects of Obstruction of the cerebral arteries by embolism, thrombosis, syphilitic endarteritis—Effects of Hæmorrhage—Renal disease, atheroma, miliary aneurysms, as causes of cerebral hæmorrhage.
HEMIPLEGIA—*Its characters and extent—The local lesion—Course and sequelæ.*
APHASIA—*Amnesia—Agraphia—Their characters, seat, and physiology.*
APOPLEXY—*Symptoms, onset, and course—Determination of locality—Its diagnosis from injury, narcotic poison, alcoholic intoxication, pyæmia, uræmia, epilepsy—Diagnosis of the anatomical causes of apoplexy.*
Treatment of apoplexy and hemiplegia—Softening of the brain.

THE Spinal Cord and the Brain are so closely related in their structure and histology, in their development, and in their functions, that their pathology might be expected to be also closely connected. This, however, is not the case. The acute softening we call myelitis has no counterpart in the brain, and the chronic degeneration of the neuron with sclerosis of the neuroglia which we have found to play so important a part in the pathology of the cord is only represented in that of the brain by two not very frequent diseases, Insular sclerosis and Paralytic dementia. In both parts of the central nervous system we find paralysis from pressure of the containing bony cavity; but while in the case of the cord disease causes this compression at least as often as accident, in the brain depressed fracture of the skull is more frequent than osseous disease as a cause of cerebral symptoms. The large size of the human cerebrum has led to an enormous development of its blood-vessels; so that the cerebral arteries are larger and more abundantly ramified than those of any other organ except the lung. Hence, we shall find that hæmorrhage, which is very rare in the cord, is the most frequent and important pathological event in the brain; and the same statement is certainly true of obstruction of the arteries by an embolus, and probably of the same obstruction by a local thrombus.

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* "Apoplexy is a paralysis of the whole frame, both of feeling and sense and motion: and if the head is affected on the right side, the paralysis is on the left, but on the right if the cephalic lesion is on the left."

In contrast with this clear and accurate statement of ancient Greek medicine is the doubtful statement made in the present century, that "it is said and is certainly confirmed by the observation of most physicians" that paralysis is on the opposite side to the lesion of the brain.

The frequency of cerebral tumours and cerebral abscess compared with the rarity of a new growth and the absence of secondary abscess in the cord, probably depends on the great size of the brain proper, and even of the little brain and the basal ganglia, when compared with the short and slender cord.

The higher functions of the cerebral hemispheres are often affected, and thus various mental symptoms belong to diseases of the brain which are entirely absent in those of the cord.

Lastly, the division of the hemispheres explains the fact that cerebral paralysis is almost always one-sided. Thus Hemiplegia is as characteristic of lesions of the brain as Paraplegia of those of the cord.

The upper projection-system of Meynert, the neurons of the cerebral cortex, with their axons passing down by the corona radiata, the internal and external capsules, the crura cerebri, the longitudinal fibres of the pons, the anterior pyramids, and restiform bodies, form the theatre of cerebral diseases, as the lower or peripheral projection-system with its neurons and axons in the cord, the nerve-roots and posterior ganglia, and the afferent and efferent nerves and nerve-endings form the theatre of the spinal diseases described in the preceding chapters.

The pathological, like the physiological, boundary between Brain and Cord is not the foramen magnum; for the Bulb is but the medulla spinalis oblongata—the continuation upwards of the cord—and is subject to at least one of the diseases of the cord, that already described as bulbar paralysis; while it is as rarely affected by hæmorrhage, abscess, or tumour as any other part of the cord. The Pons may, however, be the seat of tumours, and not infrequently of arterial lesions, which cause a fatal form of apoplexy. In fact, the pons is liable at once to the chronic morbid processes of the cord, and also to the characteristic lesions of the higher cerebral centres.

We will first give an account of the anatomical effects of diseases of the cerebral blood-vessels; and then proceed to discuss their clinical symptoms.

The arterial lesions fall into two groups: those in which there is an arrest of the flow of blood from occlusion of the vessels by embolism or thrombosis; and those in which an artery is ruptured and allows the blood to escape.

Obstruction of the cerebral arteries.—The obstruction may arise outside the skull. In a case under Gull, arteritis deformans of the arch of the aorta led to complete obliteration of the innominate and left carotid arteries, so that the left subclavian artery alone was left to carry on the circulation in the brain; the patient, a woman aged forty-one, died paralysed and insensible; and the brain was in a state of softening. A woman affected with carotid aneurysm was once admitted into Guy's Hospital, who had nearly a year before been attacked with hemiplegia and loss of speech; but after Mr Durham had ligatured the common carotid artery, she regained her speech to some extent. She died soon afterwards, and it was found that the aneurysmal sac extended upwards by the side of the internal carotid artery, and so pressed upon it that until the tension was lowered by ligature of the vessel below, the blood which reached the internal carotid from collateral sources had been unable to pass on to the brain. A much less rare event is for hemiplegia to set in *after* ligature or compression of the carotid artery. In most of these cases the paralysis is the direct result of the operation. But in another case hemiplegia did not occur until three days after the ligature. The writer remembers when he was a dresser, how Dr Stevenson, the eminent medical jurist, who was then house surgeon, ligatured the common carotid to stop hæmorrhage from a wound in the neck, and paralysis of the opposite side followed at once. Perhaps, in such cases, the communicating arteries which make up the circle of Willis may be smaller than usual.

Embolism.—In the great majority of cases, however, the cause of arrested cerebral circulation lies within the skull; and it is generally an embolon—a clot derived from some distant source, and washed into the vessel by the blood-stream. Most frequently the starting-point of the morbid process is in endocarditis of the mitral or aortic valves. At an autopsy made in 1877 a rough friable mass of calcareous deposit was exposed upon the surface of the mitral valve, and a fragment of the same material was found wedged in one of the Sylvian arteries. When there is stenosis of the mitral orifice, without roughness of its surface, the clot is almost always derived from the dilated left auricle, where thrombi form in the recesses between the fleshy columns of the appendix. In rare instances the primary lesion is in the aorta itself; we have had at Guy's Hospital two such cases: in one there was a patch of softening thrombus which adhered to the arterial wall; in the other there was septic ulceration.

In Dr Fagge's record of inspections during twenty-three years, he found forty-seven cases in which it was believed that embolism of one of the cerebral arteries had occurred; although in twenty-one no clot was discovered in any of the vessels at the autopsy, there was disease of the cardiac valves, and such changes in the brain as are known to result from embolism. All writers admit that anatomical proof of plugging of the arteries is wanting in some cases, especially when life has been prolonged; and suppose that the clot has in the meantime undergone absorption. Another possible explanation is that the spot at which the embolism was impacted may have been the carotid artery within the petrous bone or in the cavernous sinus, or the vertebral artery where it winds round the arch of the atlas—parts which generally escape examination.

The vessel into which the clot passes is almost always one of the Sylvian arteries. In twenty-three cases of cerebral embolism, Dr Fagge found that these vessels were blocked, against three in which the plug was in the vertebral artery. The fact that embola entering the carotid are almost always carried into the Sylvian branch is doubtless owing to its course being more in a direct line with the trunk than that of the two other branches.

It does not appear that there is any decided difference in the liability of the right or left Sylvian artery to embolism.*

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* It has been stated that the left Sylvian artery is much more often plugged than the right one, and an explanation for this supposed fact has been found in the difference of the angles at which the innominate artery and the left carotid artery leave the aorta. But this statement is not confirmed by experience at Guy's Hospital, for among twenty-one cases of embolism limited to the Sylvian artery of one side there were eleven in

Cerebral embolism affects adults of all ages, and is as common in women as in men (cf. *infra*, p. 729). In Dr Fagge's experience of 46 autopsies, 12 cases occurred between the ages of thirty-one and forty years, as compared with 10 between twenty-one and thirty, 9 between forty-one and fifty, 8 between fifty-one and sixty, 6 between eleven and twenty, and 1 above the age of sixty.

Dr Pitt collected 79 later cases of cerebral embolism, 44 in men, 35 in women. The ages varied between the youngest, thirteen, and the oldest, seventy-three, arranged in decades as follows: under twenty, 18; twenty-one to thirty, 15; thirty-one to forty, 15; forty-one to fifty, 10; fifty-one to sixty, 15; sixty-one to seventy, 6; and over seventy, 1. Of the 79 cases, 68 showed valvular lesions, most frequently mitral stenosis, then mitral incompetence and then left sigmoid disease, often complicating that of the mitral valves. In four cases an ante-mortem clot was discovered in the left auricle, and two in the left ventricle. Two were the result of ulcerative endocarditis of gonorrhœal origin.

In three fourths of the cases the obstructed artery was the middle cerebral, and the right was as often the blocked vessel as the left.

Thrombosis of the cerebral arteries—the formation of clots *in situ*, independently of any extraneous source—is far less frequent than embolism. It is generally secondary to disease of the arterial wall, either atheroma or syphilitic endarteritis. The vertebral arteries are more liable to be affected by thrombosis than the carotids. In one remarkable instance recorded by Dr Fagge, both the carotid arteries, the middle cerebrals, the anterior cerebrals, and the posterior cerebellar arteries were all obstructed by adherent clots in the case of a man of thirty-five. Thrombosis usually occurs at a later period of life than embolism.

Syphilitic disease of the cerebral arteries is the most important cause of local clotting. A monograph on the subject by Heubner, of Leipzig, appeared in 1874; there he states that a Danish writer, Steinburg, first attributed to an affection of the blood-vessels many of the cerebral symptoms of syphilitic infection; and that Wilks ascertained the change by adherent clots in the case of a man of thirty-five. Thrombosis usually occurs at a later period of life than embolism.

Heubner found that the process begins between the endothelium and the fenestrated membrane, where a number of cells accumulate, and encroach on the calibre of the vessel. Dr Fagge observed two cases at Guy's Hospital in which what seemed evidently syphilitic ulceration of the vertebral artery passed right through the walls of the vessel into the substance of the pons. The arterial affection is often associated with gummata in the pia mater at the base of the brain. The new tissue becomes vascular, and this perhaps saves it from undergoing retrograde changes; at any rate, it is constantly found greyish white and semi-translucent, and, as stated by Dr Allbutt, does not show any tendency to caseate. As the channel of the artery becomes narrowed the blood is apt to coagulate, and thus the local circulation is arrested. This process is known as *endarteritis obliterans*. Ultimately a process of cicatrisation may take place, the cells

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which the left one was affected, and ten in which the clot entered the right artery, a difference too slight to be noticed. The error (if error it be) has arisen from the statistical collection of miscellaneous cases from Journals and Transactions; the interest attaching to aphasia must have often led to the publication of cases of embolism on the left side, while others were left unrecorded.—C. H. F.

developing into connective tissue, and the vessel undergoing conversion into a fibrous cord.

Syphilitic endarteritis affects the carotid artery and its branches much more frequently than the vertebral and basilar and their branches—twelve times as often according to Heubner. In one of his cases the circulation at the base was interrupted at four distinct points; the left vertebral artery was obliterated, and so were also the basilar, the left middle cerebral, and the origin of the right anterior cerebral arteries. Several arteries may be diseased simultaneously, but syphilitic endarteritis is not so widely diffused as atheroma. Moreover, it affects smaller arteries and tends to narrow and occlude rather than to dilate and render them tortuous.

It is generally at an advanced stage of syphilis that the cerebral arteries become affected. Among the cases collected by Heubner the oldest patient was fifty-one years old, the youngest twenty-two; and the numbers were distributed over the three decennial periods between twenty and fifty years of age. Of fifty cases collected by Gowers, all occurred between the ages of twenty-one and forty-five, and there were three in men to one in women. The Sylvian branch of the internal carotid artery was affected in nineteen out of twenty cases.

Syphilitic endarteritis of the brain, like other remote effects of lues, often occurs with no history of the disease, and unaccompanied by eruptions, nodes, or its other obvious signs. At Guy's Hospital, since the first case recorded by Wilks in 1863, we have had numerous well-marked instances of the affection, besides others of a more doubtful character; except in a few with a history of syphilis, the evidence of their origin was the presence of gummata in the liver or testes, or of lardaceous disease.

Effects of obstruction of cerebral arteries.—The result which follows obstruction of a cerebral artery, whether by embolus or thrombus, is sometimes, as in the retina, the kidney, and the lungs, venous hyperæmia from reflux hæmorrhage, but more frequently it is softening.

The anatomical relations of the vessel which happens to be obstructed of course determine the locality and extent of the morbid change; and upon this, in turn, depends the nature of the symptoms. With regard to the Sylvian artery, we shall find that the whole clinical aspect of a case differs according as this vessel is plugged at its origin, or a little further on its course. It is therefore important to be acquainted with the exact distribution of the cerebral arteries, which was worked out by two independent observers, Duret in France, and Heubner in Germany, and their results have since been amply confirmed.

The small arteries that alone enter the tissue of the brain form two separate systems, which may be distinguished as that of the cortex and that of the central ganglia (the corpus striatum and thalamus). These systems are independent, with no anastomoses between them; the zone at which they meet within the cerebral substance is situated about an inch and a half beneath the convolutions. The "central" arteries arise directly from the trunks forming the circle of Willis; and they are "end-arteries," *i. e.* they do not anastomose among themselves. The "cortical" arteries spring from a network in the pia mater, where there is pretty free communication between the tertiary branches of the same trunk, and sometimes between the branches of different trunks.

Let us now apply these facts to explain the effects of obstruction of the Sylvian artery at different points. That artery, close to its origin, gives off a number of small "central" twigs, which supply the whole corpus striatum (except the inner end of the caudate nucleus), and also the anterior part of the thalamus. It then divides into four terminal branches, of which one is distributed to the third frontal convolution, while the other three pass to the second frontal, the ascending frontal and ascending parietal, the other convolutions of the parietal lobe, and those of the temporo-sphenoidal.

When the middle cerebral artery is obliterated beyond the point at which its "central" offshoots arise, the superficial parts of the brain are the only ones to suffer. And since the subdivisions of the vessel in the pia mater anastomose with those of the anterior and posterior arteries, it is not certain that in the supposed case any softening will result. There may be merely a temporary interference with the circulation in the area to which the Sylvian branches are distributed; or a limited part in the midst of this area may be permanently deprived of its blood-supply; or, lastly, almost the whole of the convolutions fed by the Sylvian artery may undergo destruction. Thus Charcot has recorded an instance in which an enormous superficial patch of softening involved the ascending frontal and the ascending parietal convolutions, as well as those of the insula, while the corpus striatum and thalamus remained intact. The differences in the result in different patients depend partly upon variations in the extent to which the vessels communicate with one another, partly upon whether the plugging takes place suddenly or gradually. Moreover, a single one of the four terminal branches, such as that to the third frontal convolution, may undergo obstruction apart from the rest.

On the other hand, when the seat of the lesion is at the spot where the Sylvian artery arises from the internal carotid, the "basal ganglia" suffer, and here softening is almost inevitable, because their arteries have no anastomoses; it is only when the closure of the vessel takes place very slowly, as the result of chronic disease of its coats, that collateral channels sometimes seem to develop themselves, so that the blood-supply is maintained. When the main channel of the Sylvian artery has been obliterated extensive softening or hæmorrhage in the corpus striatum and thalamus is likely to occur, while the convolutions may entirely escape.

The appearances presented after death by the affected parts of the brain vary considerably under different conditions. When the patient dies rapidly, the cerebral substance may look perfectly healthy, as Dr Fagge found to be the case in an autopsy on a man who was attacked with hemiplegia twenty-six hours after ligature of the internal carotid artery, and who lived only fifty-seven hours afterwards. Even when closure of an artery takes place gradually, the regions of the brain that are deprived of their normal blood-supply often suffer less than might be expected. Heubner remarks that in syphilitic affections of the cerebral arteries the nutrition of the cortex is seldom seriously interfered with, unless two adjacent runks out of the six that arise from the circle of Willis are completely bstructed. In the case already referred to, in which the circulation at he base of the brain was interrupted at four distinct points, there was no oftening at all, although one Sylvian artery was obliterated.

Softening of the brain.—This is a term formerly, and even now, nproperly applied to cases of dementia, whether following an apoplectic roke or not. True histological cerebral softening is, though not constant, he most frequent result of obstruction of an artery.

In cases of *embolism*, softening is, as a rule, the only change presented by the parts of the brain deprived of their blood-supply. Occasionally, however, there is rupture of the diseased artery above the block, and more often reflux hæmorrhage from the veins, as occurs in the lungs and spleen from embolus. Indeed, after ligature of the carotid artery, the corresponding cerebral hemisphere is sometimes found in a condition of vascular turgescence as in the early stage of an "infarctus."

The two conditions of hæmorrhage and softening may be present together. Thus, Wilks once saw as the result of embolism all the vessels on the affected side of the brain—including the veins as far as the longitudinal sinus—distended and filled with coagula, while the cerebral tissue was pulpy and dark red. In other cases of embolism at Guy's Hospital, this "red softening" is described, and in two other cases tough yellow masses were found which exactly resembled the wedge-shaped patches that are so common in the spleen and the kidney.

A more frequent, probably a later change after arterial obstruction, has been called "white softening." The brain-substance is diffluent, being replaced by a milky liquid, which occupies an ill-defined cavity, containing some loose shreds of connective tissue, and perhaps roofed in by the pia mater or by the ependyma of the lateral ventricle. Every intermediate degree of consistence may be seen up to a point at which one can hardly perceive any difference from the healthy substance around, until one allows a gentle stream of water to play over the diseased surface, when it soon becomes ragged and assumes a worm-eaten appearance.

The softened parts of the brain may be white, or yellow, or red. Distinctions between these as separate affections have no real existence, and have led to great confusion. "Red softening" derives its characteristic colour from a multitude of minute extravasations of blood. In other words it is softening with capillary hæmorrhage. The reason for its being seldom seen in cases of thrombosis or syphilitic endarteritis being that in these cases death does not take place soon enough.*

The differences in colour in different cases of softening of the brain depend on the presence or absence of hæmoglobin. The blood undergoes disintegration with the tissue-elements among which it lies, and under the microscope it is easily recognised in the form of yellow or red granules, or of hæmatoidin crystals; what was first red becomes brown and then yellow, or if there is no hæmorrhage it remains white.

In softened brain-substance, as in degenerated spinal cord, are also seen "compound granule-masses," or "corpuscles of Gluge," often in large numbers. These mulberry-like aggregations of minute granules of oil are probably degenerated exudation cells.†

Aneurysm from embolism.—The coats of the obstructed artery occasionally become softened, and yield immediately above the seat of obstruction, so that an *aneurysm* is formed. The embolus in these cases is derived from ulcerative (bacterial) endocarditis, and leads to septic softening of the wall of the artery. Repeated examples of this were observed by Dr Fagge; in another Dr Goodhart found an aneurysm on the Sylvian artery on each side. Death was in most cases due to rupture of the aneurysm, blood being extravasated into the pia mater, the corpus striatum, or the lateral ventricle. Cerebral hæmorrhage has been found in other cases of ulcerative endocarditis with vegetations on the mitral or aortic valves; probably an aneurysm, itself the result of embolism, was present in these cases likewise, although it escaped notice at the autopsy.

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* What most English pathologists call "white softening" is named "yellow softening" by Rindfleisch. He uses the term "white softening" for a partly post-mortem appearance in the central parts of the brain in cases of hydrocephalus, and in England we have been accustomed to describe as "yellow softening" the œdematous state of the cerebral substance that is often found in the neighbourhood of tumours.

† Huguénin admits no less than seven origins of these corpuscles—the nuclei of the neuroglia, the cells which make up the walls of the capillaries, those of the "adventitia" of the arteries, the nuclei of the smooth muscular fibres of the vessels, those of the perivascular lymph-spaces, the spindle-cells of the cortex, and probably even its ganglion-cells!

Cerebral hæmorrhage.—Although effusion of blood into the brain may be the result of embolism, of embolic aneurysm, of purpura, or of external injury, yet in the great majority of cases it is due to primary disease of the cerebral arteries and their consequent rupture.

Since the days of Morgagni it has been known that hæmorrhage is far more apt to occur in or near one of the corpora striata than in any other part of the brain; but Gendrin, Charcot, and other observers arrived at a more exact determination of its seat, and accounted for that being so generally the seat of rupture of an artery. On the outer side of each lenticular nucleus lies the external capsule. In recent brains the two structures seem to be continuous; but after a brain has been hardened in spirit it is found that they can be separated from one another with great ease, and apparently, although not really, without any nervous fibres being torn through. Thus the connection between them must be somewhat imperfect. Now the central branches of the Sylvian artery (p. 720) which supply the corpus striatum run upwards for some distance outside the lenticulus before penetrating its interior. These are more liable to laceration than those of the hemispheres generally, because they are less supported. Moreover they are much larger than the nutrient arteries of the cortex, they have no anastomosis with one another, and they arise directly from a large trunk, almost in a straight line from the heart. Indeed, as Watson long ago noticed, when injections are forced into the cerebral arteries of the dead body it is in the corpora striata that the vessels are most apt to give way, and the wave to be extravasated. Charcot called one large lenticulo-striate branch "the artery of cerebral hæmorrhage."*

The blood begins by making for itself a space between the lenticulus and the external capsule. At first it forms a thin layer, but as it increases in quantity it gradually becomes a rounded mass. It now flattens out the island of Reil and the claustrum, which lie below and to its outer side; it pushes inwards the corpus striatum, and also the thalamus if it reaches far enough backwards. When it accumulates slowly it may press upon the parts around, so as to form a smooth-walled cavity. But more frequently it tears up the white matter of the hemisphere and lenticulus. In some rare cases it reaches the superficial convolutions: more often it ruptures into the lateral ventricle. Coagulation quickly occurs, and at an autopsy we may find a mass of clot weighing four ounces or more, beside a red liquid, consisting of blood mixed with cerebro-spinal fluid in the lateral ventricles. Sometimes a clot occupies one ventricle, while the contents of the other are only bloodstained, with perhaps a little clot in the cornua. Sir Wm. Broadbent has noticed that the middle ventricular cornu close to the seat of the hæmorrhage is commonly empty, having been compressed by the extravasated blood before rupture into the cavity of the ventricle took place. Not infrequently the third ventricle, the aqueduct, and the fourth ventricle are all filled with moulded coagula exactly fitting their cavities: of this curious condition we have a drawing and preparation made by Mr Hilton in a case of traumatic cerebral hæmorrhage ('Cat. Mus. Guy's Hosp.'). The blood occasionally escapes along the subarachnoid space so as to reach the exterior of the bulb and pons, and the parts at the base as far as the opposite Sylvian fissure. The fissure on the side of the hæmorrhage is too closely compressed to receive any of the blood; indeed, the convolutions of the vertex of that hemisphere are often greatly flattened, so that as soon as the skull-cap is taken off it is plain which side is affected.

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* It is true that the rupture of any artery in the brain is always preceded by the occurrence of morbid changes in its walls; but while this diseased state is common to most of the cerebral vessels, the special liability to rupture of those in the external capsule may be explained by the considerations just offered.—C. H. F.

Seat of hæmorrhage.—Of 95 consecutive fatal cases of cerebral hæmorrhage observed by Dr Fagge—

In 70 the seat of the lesion was in or near the *basal ganglia*. In only 4 of these cases is it stated that the blood was confined to the substance of the hemisphere, not having found its way either to the surface or into the lateral ventricles. In 57 it is expressly recorded that laceration into the lateral ventricle had taken place; and in 22 of these the fourth ventricle also contained either a clot or bloodstained fluid. In 6 cases the superficial convolutions were reached. In 6 instances the part into which the blood was effused was behind the thalamus, somewhat beyond the limits of the region which is the usual seat of cerebral hæmorrhage. Twice the caudate nucleus was alone affected. Next to the external capsule in liability to hæmorrhage comes the internal capsule and the area supplied by the thalamo-striate (or opto-striate) artery, and next the grey substance of the corpus striatum and the claustrum. The right side of the brain was attacked in 36 of the 70 cases, the left side in 34.

In 12 of the remaining 25 cases the seat of the hæmorrhage was in the *pons*; in 1 it was in the right half of the *cerebellum*. Among the cases of "apoplexy of the pons" there were 5 in which the blood had escaped into the fourth ventricle; and 2 in which it had oozed out through the convex surface of the pons, so as to reach the base of the brain. In no less than 8 of the cases in which there was hæmorrhage in the neighbourhood of the corpus striatum, blood was also effused into the pons; in one instance three independent hæmorrhages were found in this part.

Lastly, there were 12 cases in which the extravasation was into the *membranes* on the surface of the brain. "Meningeal apoplexy" is often described as a distinct affection; and this is justified by the fact that it is sometimes connected with septicæmia, purpura, anæmia, or pneumonia, and is sometimes caused by blows or falls on the head, or by the increased venous pressure in the infant's skull caused by prolonged labour. But in the twelve cases here cited it was associated with exactly the same changes in the heart and kidneys which we shall see to be commonly found with ordinary cerebral hæmorrhage, and there is every reason to believe that the difference in seat was accidental. The quantity of blood was often large; the nerves at the base of the brain were buried in a thick clot, and the extravasation extended along the Sylvian fissures and in the meshes of the pia mater over the surface of the hemispheres, and also in some instances along the subarachnoid space to the spine, and into the fourth ventricle.

Among 912 cases of non-traumatic intra-cranial hæmorrhage, collected from various sources, the writer found that nearly 65 per cent. were in the *cerebrum*, and more than five sixths of these in the corpus striatum, thalamus, or adjacent parts; that more than 20 per cent. were *meningeal*, nearly 10 per cent. in the *pons*, and about 5 per cent. in the *cerebellum*—a proportion not widely different from that observed in the above ninety-five cases collected for the first edition of this book.

Histological effects of hæmorrhage.—When life has been prolonged for a few days after the occurrence of cerebral hæmorrhage alterations are observed in the tissue round the clot. At first it is reddened, or visibly ecchymosed; then it becomes œdematous and of a yellow colour, exactly as occurs around a tumour of the brain, and finally passes into a state of softening. Suppuration appears never to take place round a clot.

The later results of cerebral hæmorrhage when the patient survives the attack for months or years are not less remarkable. Sometimes all that is left is a soft tawny discoloured spot, containing abundant granule-masses and crystals of hæmatoidin, or a tough, flat, fibrous mass—a hæmorrhagic cicatrix. Sometimes there is a membranous cyst, with shreds of connective tissue and a clear or turbid fluid in its interior. The cavity may be the size of a walnut, or even as large as a bantam's egg. Such appearances have been plainly recognised thirteen or fourteen years after the apoplectic stroke, when the patient has died of some different disease. Possibly even hæmorrhage into the ventricle may not be invariably fatal.

Ætiology.—The immediate causes of cerebral hæmorrhage are undoubtedly disease of the arterial coats and increase of the blood-pressure in the cerebral arteries.

It is equally certain that in this country the most frequently found condition in cases of hæmorrhage into the brain is *chronic Bright's disease* with hypertrophy of the left ventricle. Among 116 cases* which came under observation consecutively in the deadhouse of Guy's Hospital some morbid condition of the kidneys was found in eighty-six; while in only fifteen out of the whole number is it reported that these organs were healthy. But, strange to say, Continental pathologists seem not to have found the kidneys diseased in a similar proportion of cases. Charcot and Bouchard say that interstitial or parenchymatous nephritis was present in only three among forty-nine cases of theirs in which it was looked for, there being, however, thirteen other cases in which the kidneys were "simply atrophied." Nothnagel, in 'Ziemssen's Cyclopædia,' appears to be equally ignorant of the close connection between renal affections and cerebral hæmorrhage, for he quotes, almost without comment, the statement of Frerichs that among 241 cases of Bright's disease there were only six in which effusion of blood into the brain occurred. This, indeed, is not so incompatible with the English observations as might at first sight appear. For the small red kidney described by Bright is far more common in England than on the Continent, and none of our cases have been examples of tubular (epithelial or parenchymatous) nephritis; and comparatively few of them seem to have presented mixed epithelial and interstitial changes.

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* All those instances were excluded in which there was reason to believe that embolism was the starting-point of the lesion, and also a few cases of meningeal hæmorrhage which accompanied purpura, or were due to an injury.

In the great majority the renal affection has been of the "granular" or cirrhotic kind, the cortex having shrivelled up into a mere relic. In a few instances one kidney had undergone destruction from a stone in its pelvis, or the two organs were each converted into a congeries of cysts. It is possible that in these cases the occurrence of cerebral hæmorrhage was merely an accidental coincidence.

All trustworthy observers are agreed that morbid changes in the arterial walls precede their rupture; but when the cerebral arteries are already diseased, there can be no doubt that the hypertrophied left ventricle and high tension of chronic Bright's disease make the hæmorrhage more likely, just as we shall see that it not infrequently follows a sudden and temporary increase of blood-pressure.

Sometimes the arteries which arise from the circle of Willis are found to be extremely *atheromatous*, or their walls are extensively calcified.

In the case of a man aged forty, who died in Guy's Hospital in 1869, and who had a hypertrophied heart and granular kidneys, there was a small *aneurysm* on the left middle cerebral artery which had given way and poured blood into the Sylvian fissure, while there was also a large clot outside the corpus striatum, with laceration into the lateral ventricle, apparently of independent origin.

In 1866 Bouchard discovered, in a case of cerebral hæmorrhage taken from the wards of M. Charcot, two small aneurysms upon one of the vessels; and afterwards published seventy-seven cases of what he termed *miliary aneurysms*. These are minute globular or fusiform swellings, from one fifth of a millimètre to one millimètre ($\frac{1}{125}$ to $\frac{1}{25}$ of an inch) in diameter, so as to be plainly visible to the naked eye. They are sometimes very soft, sometimes firm and elastic, sometimes hard like grains of sand. In colour they are purple, or reddish brown, or greyish, according to the thickness of their walls and the state of their contents, which may be either fluid or more or less decolourised clot. Sometimes only two or three can be discovered in the whole of the brain after careful search; sometimes as many as a hundred have been counted. They are most conspicuous upon the surface of the convolutions, and their favourite seat is the deep layer of the grey matter.*

Sir William Gull long ago taught that it was chiefly in young subjects that intra-cranial aneurysm should be suspected as a cause of cerebral hæmorrhage; and Dr Church, in tabulating a series of cases of this kind, confined his attention to individuals under twenty years of age. But of nine cases observed by Dr Fagge, while seven were between fourteen and twenty-six, one patient was thirty-four, and another fifty years old. We have already seen that cerebral embolism is also not so strictly limited to an early period of life as has been supposed.

Miliary aneurysms are not found with especial frequency in that region of the brain which we have seen to be so generally the seat of effusions of blood. Liouville demonstrated a similar affection in the arteries of other parts, and found miliary aneurysms beneath the mucous membrane of the œsophagus and upon the surface of the heart. Indeed, the lesion is but one of the effects of a wide-spread arterial change—a chronic "sclerous periarteritis,"—consisting in thickening of the arterial sheath and of the adventitia, with overgrowth of nuclei, while the muscular coat undergoes atrophy.*

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* As Charcot and Bouchard admit, when an extensive effusion of blood into the brain has occurred, it is difficult to find the miliary aneurysm the rupture of which led to the hæmorrhage. The best way is to place the affected hemisphere in a basin, and to change the water frequently by tilting, without shaking it. After a time the clots become loosened and float away, leaving a number of little bloody masses connected by vascular filaments with the brain tissue beneath. Most of these, when magnified, show only the open ends of vessels, torn across secondarily by the extravasated blood, and covered with little caps of fibrin that require to be carefully distinguished from actual aneurysms; but at length some are found which contain spherical ampullæ, continuous with small arteries and split on one side.—C. H. F.

Apart from the formation of aneurysms, large or small, there is no doubt that this diffused change in the arteries of the brain increases the risk of hæmorrhage. In twenty autopsies on cases of localised softening Dr Fagge found in a large majority granular kidneys and hypertrophied heart; while there was not a single instance in which the kidneys were healthy. In the ages of the patients, and the fact that most of them were males, these cases correspond exactly with those of cerebral hæmorrhage; and probably it was a mere accident that death occurred before any of the softened arteries gave way.

Sex and age.—Cerebral hæmorrhage does not occur with equal frequency in the two sexes or at different periods of life. Men are much more liable to it than women. Of 113 consecutive fatal cases at Guy's Hospital, 82 occurred in men, and only 31 in women.

Among 110 of these cases, in the reports of which the ages of the patients are stated, there were 6 between twenty-one and thirty, 18 between thirty-one and forty, 37 between forty-one and fifty, 29 between fifty-one and sixty, 17 between sixty-one and seventy, and 3 above the age of seventy. These figures do not quite correspond with the statements of most writers, who make the frequency of apoplexy increase up to the age of seventy or even eighty years, not only relatively to the numbers of persons living at different ages, but also absolutely. It may be that men at a middle period of life are more likely than those who are older to be brought to the hospital when attacked with apoplexy, because their relatives are not so apt to suppose that the case is hopeless.

Gintrac's 658 cases (excluding meningeal apoplexy), as quoted by Bastian, give for the corresponding ages: under 20, 6 per cent.; 21—30, 7 per cent.; 31—40, 11 per cent.; 41—50, 15 per cent.; 51—60, 20 per cent.; 61—70, 23 per cent.; and above 70, 19 per cent. Hippocrates ('Aphor.,' lib. vi, § 57) says that men are most liable to apoplexy between the ages of forty and sixty.

Gout is only indirectly a cause of cerebral hæmorrhage, by setting up granular disease of the kidneys. There is an old belief that apoplexy is particularly common in men with short thick necks and florid faces, but this is very doubtful.†

Cerebral hæmorrhage sometimes occurs first while the patient is making some violent effort, as in running to catch a train. It is believed that sudden emotions, straining at stool, prolonged laughing, coughing, or sneezing are also capable of bringing on the rupture of an artery in the brain, and this may be true provided that the vessel is in a diseased condition. But one must not forget that cerebral hæmorrhage often occurs during sleep, when the pressure in the cerebral vessels is probably low. Watson refers to two cases in which men were attacked by hemiplegia while in the act of sexual intercourse; and a most severe case of apoplexy followed by hemiplegia occurred under these circumstances in a man fifty years of age, a patient of the writer, who remained hemiplegic for more than two years, and then died of a second attack.

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* We are brought by these investigations on sclerous periarteritis to almost the same point to which we are led by the observations of Gull and Sutton on "arterio-capillary fibrosis." Indeed, it seems clear that the French and the English pathologists have been studying the same morbid process, and their general agreement confirms the accuracy of their conclusions. It is strange, however, that MM. Charcot and Bouchard failed to notice the affection of the kidneys.—C. H. F.

† Quæ (animæ) subito revelluntur e crasso corpore multum corporeæ molis secum ferunt: tales autem mittit apoplexia.—*Erasmi Colloquia: Charon.*

The symptoms of obstruction and of rupture of the cerebral arteries differ in some points; but the three most important—*hemiplegia*, *Aphasia*, and *Apoplexy*—are common to both, and will now be separately considered. They are each of great pathological and clinical interest.

HEMIPLEGIA.—The division of the encephalon into two separate hemispheres, each with its own system of blood-vessels, makes unilateral lesions frequent, in striking contrast to the extreme rarity of hemiparaplegia.

Motor symptoms.—The limbs which are affected are always those on the side opposite to the lesion in the brain. This depends upon the fact that the motor columns cross over in the anterior pyramids; those which emerge from under the right half of the pons Varolii passing down into the left half of the spinal cord, and *vice versâ*. Thus any unilateral affection of the nervous centres situated above the decussation of the pyramids, if it causes paralysis at all, affects the opposite side.*

It is remarkable that this fact was well known to the Greek physicians, who correctly explained it as due to the nerves crossing like the letter X; but it was forgotten or doubted as late as the present century. Compare the quotations which are printed at the head of this chapter from Aretæus in the first century, and from Dr James Gregory, professor of medicine at Edinburgh, in the nineteenth.

Although hemiplegia means a stroke paralysing half the body, the extent to which it affects the muscles varies greatly. As a rule, in what may be called "common cerebral hemiplegia"—the *hémiplégie cérébrale vulgaire* of Charcot—the paralysis affects the opposite arm and leg, the lower part of the face and the tongue. In the most severe forms the muscles of the whole of the face are paralysed, and even those of the trunk are obviously weakened; in slighter attacks the tongue and face almost entirely escape, and in some the leg also is so little affected that the case might be mistaken for one of brachial monoplegia. Moreover the hand is always more affected than the arm, and the arm than the shoulder, the foot than the hand, and the leg than the thigh. The ocular and laryngeal muscles, those of mastication, and the diaphragm, always escape. In the facial muscles, commonly all that is noticeable is a little loss of expression about the mouth, which may appear to be drawn over to the opposite side; and not infrequently the face escapes entirely.

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* There are some cases on record which seem to be exceptions to this rule, and these were once brought forward by Brown-Séquard to establish the possibility of hemiplegia on the same side as the lesion on which it depends. But the number of cases of this kind is altogether insignificant in comparison with that of cases on which the rule of decussation is based. In some of the supposed exceptions it is most likely that the reporters have written "right" for "left," or "left" for "right"—in case-taking an easy error. Our records at Guy's Hospital contain two cases in which it is stated that the disease was on the same side as the paralysis; and one of these is in the handwriting of Dr Wilks, who leaves the statement entirely without comment. There can be no doubt that it was a slip of the pen. In other cases probably there really were two lesions: one, the more conspicuous, on the same side as the hemiplegia; the other on the opposite side—perhaps a minute spot of softening—which really caused the paralysis, but was overlooked.—C. H. F.

Another explanation, viz. that in some persons there is no crossing of the anterior pyramids, is unsatisfactory: first, because there is no evidence that the crossing is absent in the cases in question; and secondly, because in man and animals the crossing is of variable completeness, and when deficient is probably made up for by crossing lower down.

Henry W— was admitted into Guy's Hospital on February 20th, 1860, under the care of Dr Gull, for right hemiplegia, which was almost complete. The state of his face was very interesting. When told to shut his eyes he could close them both. But whereas he could wink with the left eye alone, he could not do it with the right alone; and when he was asked to compress the eyelids firmly the left orbicularis palpebrarum alone obeyed the will, the right one merely bringing the upper and lower lids into contact, without any wrinkling of the skin. This, and the further observations which Dr Fagge made on the same patient, led him to the conclusion that "those movements which are performed in harmony by the two sides of the face or body remain unimpaired in hemiplegia." In 1866, in an article in the 'Medical and Chirurgical Review,' Broadbent first laid down this law, and it has received general acceptance.*

The exemption of habitually bilateral movements (not always performed by corresponding muscles of the two sides) is seen in the case of the muscles of the eyeballs and in that of the masticating muscles.

Almost invariably, when recovery takes place, the patient regains power in the leg earlier than in the arm; he may perhaps be able to walk with a crutch at a time when he can scarcely move the fingers at all, and if any part remains permanently paralysed it is the upper limb. These facts may be explained by the movements of the arm being more independent than those of the leg, and furnish a further illustration of Broadbent's rule that the limb of which the movements are more highly specialised and less automatic suffers most and longest.

The highly differentiated movements of fingers are more affected than the coarser movements of the shoulder. The tongue and the lips, however, are only partially paralysed; when protruded from the mouth the tongue is more or less distinctly turned towards the side on which the limbs are paralysed, and the patient often articulates indistinctly and mumbles in his speech. A slight degree of paralysis becomes more evident if the patient pushes his tongue first to the right and then to the left side.

The hypoglossal nerve and the portio dura—arising as they do above the decussation of the anterior pyramids—nevertheless resemble the nerves of the limbs in showing impairment of function on the side opposite the lesion in the brain. This depends on the fact that the fibres from the roots of those nerves undergo an independent decussation in the pons.

In severe cases of hemiplegia the loss of power extends to muscles which are used in association with those of the opposite side. Thus the chest does not expand fully on the paralysed side, at least when a deep breath is being taken. Dr Walshe determined this by actual measurement ('Lancet,' 1849). Broadbent himself pointed out that when a hemiplegic patient is made to raise himself from the recumbent posture by his abdominal muscles alone, the rectus on the paralysed side can be felt to act somewhat less powerfully and a little later than on the healthy side. So, also, if he is told to close the jaws firmly, the masseter and temporal muscles do not contract quite simultaneously or with equal force on the two sides.

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* In 1860 or 1861 I read before the Physical Society of Guy's Hospital a paper in which I developed the same theory; of course, as I did not print it, the priority rests with Dr Broadbent.—C. H. F.

It is remarkable that when there is profound paralysis of the face or arm, movements which the patient is quite unable to execute by his own will may still be called forth by emotional or involuntary stimuli. Thus a hemiplegic may raise his shoulders in yawning, or contract his zygomatic muscles in laughter, when he cannot perform the same movements by a voluntary effort. We may associate these facts with the curious observation that when a man is speechless from apoplexy an oath may be forced from him by some sudden emotion. The escape of the muscles of respiration, of the larynx, and of organic life in hemiplegia points to the same rule, for which there seems to be no anatomical explanation; nor do we see such limitation of peripheral paralysis (say of the portio dura) or of paraplegia, so that we must refer the facts above stated of the limitation of cerebral paralysis to a physiological or evolutionary differentiation of function.

As Hughlings Jackson many years ago pointed out, patients who are aphasic from a lesion situated above the corpus striatum on one side are sometimes quite unable to protrude the tongue from between the teeth. We have several times observed this at Guy's Hospital, most clearly in a patient under the writer's care in Philip Ward, in January, 1890. It seems to be an instance of bilateral paralysis from a unilateral lesion; but we may perhaps attribute it to an inhibitory influence transmitted downwards upon the associated nuclei of the ninth nerves in the pons, rather than believe that both sides are "represented" in each cerebral hemisphere. This is the hypothesis suggested by Jackson to account for the supposed occurrence of paralysis of all the limbs as the result of an affection of one hemisphere.

As an exception to the rule above stated, that the oculo-motor nerves do not share in hemiplegia, we sometimes see that the eyes, instead of being directed straight forward, are turned to one side, and this side is always opposite to that of the paralysis, so that, for the purpose of artificial memory, one may say that the patient "is looking towards the lesion in his brain." This *conjugate deviation* of the eyes is by some writers regarded as due to a paresis of certain ocular muscles—the external rectus on the side of the paralysed arm and leg, the internal rectus on the other side. Others have supposed it to depend upon spasm of the antagonist muscles, and thus to be analogous to the state of "early rigidity" which will presently be described. But the whole face and head may also be drawn over, away from the hemiplegic limbs, and in such cases Prévost found that the sterno-mastoid and the trapezius muscles are not necessarily in a state of contraction. Moreover, Jackson has pointed out that when "early rigidity" comes on in the arm and the leg, the "conjugate deviation" becomes reversed, the eyes being now directed towards the side of the paralysis; a case in point was noted by Dr Fagge in 1867 in a patient of Dr Rees. Thus it seems clear that this remarkable symptom is not of a spasmodic nature, and Vulpian and Prévost are probably right in supposing that it represents, in a rudimentary form, the rotatory movement (*mouvement de manège*) which results in animals from unilateral injuries. Indeed, a woman under the care of Charcot for left hemiplegia with conjugate deviation of the eyes was several times found lying across her bed with her head to the right and her feet to the left. Prévost showed that conjugate deviation of the eyes is not limited to affections of any one region of the brain: among fifty-eight cases which he collected there were four of meningeal hæmorrhage, and four others in which the cortex was alone affected. It is generally transitory, lasting only a few hours or a day or two; but sometimes it persists for weeks or months.

We have seen that in common cerebral hemiplegia there is no complete paralysis of any of the cranial nerves, but in a less common group of cases palsy of the third or of the seventh nerve is associated with hemiplegia, and then the seat of the disease can be determined with more than usual exactness.

The facial nerve (*portio dura* of the seventh) may be affected in two different ways. First, the lesion, if it is situated in the upper part of one lateral half of the pons, may destroy the motor-facial nucleus, and so cause a total loss of power in the face on the same side on which the arm and leg are paralysed: and since the nucleus of the sixth nerve is very close to that of the seventh, paralysis of the external rectus may perhaps be present in addition. Secondly, an affection of the lower part of the pons on one side may cut the fibres of the facial nerve as they are passing across the motor tract, after they have decussated on their way outwards from their nucleus. The paralysis of the face is then on the opposite side to that of the limbs. Or, thirdly, it is not impossible that an extensive unilateral lesion of the pons might cause paralysis of both sides of the face by destroying the nucleus of one facial nerve and the fibres of the other.

Again, when hemiplegia involves the third nerve, the ocular muscles it supplies suffer on the side opposite the seat of disease in the crus cerebri. An instance of this has been placed on record by Sir Hermann Weber ('Med.-Chir. Trans.,' vol. xxviii). A man, aged fifty-two, was attacked with paralysis of the right side of the body and of the muscles supplied by the left third nerve; he died after two months, and an oblong clot of blood was found in the left crus.

Gubler and other French writers have given the name of "*Hémiplégie alterne*" to cases in which either the portio dura or the third nerve is affected on the side opposite to the limbs. "Crossed hemiplegia" is the English equivalent.

Sensation.—Feeling is much less affected than motion; but there is every reason to believe that anæsthesia is present in recent cases, although its recognition may be difficult or impossible in patients whose consciousness is much obscured. It is, however, far more transitory than the motor paralysis, often passing off in a few days, and sometimes still earlier. In this respect lesions of the brain resemble those of the spinal cord and of the peripheral nerves. In slight cases of hemiplegia sensation is apparently unaffected, and even in the most profound it is never completely abolished. The hands and feet are more affected than the proximal part of the limbs, and the face less. But in exceptional cases, as Gull stated in his Gulstonian Lectures (1849), anæsthesia is more marked than motor paralysis. In one case the writer noticed this in each of a series of hemiplegic attacks, some affecting the right and others the left side, in a patient seventy-two years old.*

Subjective sensations of formication and numbness are frequently associated with partial anæsthesia, and sometimes there is a condition of hyperalgesia, or increased susceptibility to painful impressions. Sometimes, too, spontaneous pains are experienced in the joints on the affected side, particularly in the shoulder-joint.

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* The patient was no other than the late Sir William Gull himself.

Trophic changes.—Hemiplegic limbs are often reddened and warmer than those of the opposite side—a difference which is attributed to interference with the functions of the vaso-motor nerves. The inequality of temperature amounts to something less than one degree Centigrade; after some months it ceases to exist. In some cases there is excessive sweating; or slight œdema is discoverable. In a case at Guy's Hospital, dropsy (of renal origin) was limited to the arm and leg that were paralysed; and the late Dr Laycock, of Edinburgh, published several examples of this curious fact. Charcot described as frequent in hemiplegia rapid sloughing of the skin over the gluteal muscles—an acute bedsore (cf. p. 611); and he regarded the affection as of tropho-neural origin. Such bedsores are certainly very rare in cases of hemiplegia seen in this country; and equally so other trophic affections of the skin such as are noted in peripheral paralysis.

As a rule contractions in the paralysed limbs by reflex action are less readily excited than on the healthy side; as an exception, more readily. In the latter case some amount of tonic spasm is often present; the elbow is flexed and resists extension, and the fingers are bent in upon the palm of the hand. This condition was described by Todd under the name of "early rigidity." The muscles do not waste, and do not lose their faradic contractility, or show any other signs of the reaction of degeneration. Subsequent rigidity or "late contraction" of the limbs is among the sequelæ of hemiplegia (p. 734).

Vision in hemiplegia.—In certain exceptional cases hemiplegia impairs the sight; but there is difference of opinion as to the kind of imperfection. According to Jackson it is lateral homologous hemiopia,* corresponding halves of each retina being affected in such a way that the patient is unable to see towards the paralysed side. If, as is probable, only the inner parts of the optic nerves decussate at the chiasma, we have a satisfactory explanation of this "homologous lateral hemiopia." In one instance, in which the paralysis was on the left side, the autopsy showed a single lesion of the right thalamus, the posterior half of which was softened and of a greyish-yellow tint ('Lancet,' May, 1875). The hemiopia had been of the most marked description. The man had sometimes seen only half a word, reading "land" for "midland," and remarking to his son that "Liver" was a queer name, when it really was "Oliver."

Charcot, however, maintained that there is a "crossed amblyopia"—an impairment of vision in one eye on the paralysed side. The acuteness of vision is often diminished by one half: moreover the visual field is narrowed on all sides, and this limitation of the field is more marked for some colours than for others. The first to be lost is violet; then, in succession, green, red, orange: the colours for which vision is retained longest are yellow and blue, but even they may at last be indistinguishable, so that every object appears of a brown colour, as if painted in sepia.

In a case of Dr Bastian's, a lesion limited to one of the anterior corpora quadrigemina caused crossed amblyopia, not lateral hemiopia. He thinks there must be a second decussation of the optic tracts for those fibres which do not cross in the chiasma.

Seat of the lesion.—As Hutchinson long ago showed, hemiplegia is not infrequent when meningitis is limited to one half of the brain, as after surgical injuries; but as a rule even extensive destruction of the cortex of the cerebrum is followed by no definite signs of paralysis, so long as it spares the "motor area." The experiments of Fritsch and Hitzig, and those of Ferrier and Yeo, Schäfer and Horsley (to which we shall more fully refer in the next chapter), have shown that irritation of convolutions of this area causes movements in the opposite limbs; and also that destruction of these convolutions can produce a permanent paralysis. Cortical lesions, to use Jackson's terms, are both "discharging" and "destroying" lesions. Diseases of the ascending frontal and parietal convolutions may, if sufficiently extensive, give rise to permanent hemiplegia, although the corpus striatum remains perfectly healthy. Softening of this motor region of the cortex is an occasional result of obstruction of the Sylvian artery, beyond the origin of its "central" branches, whether by thrombosis or embolism. The paralysis seems to be indistinguishable from that caused by a lesion of the motor tract, and is followed by the same consecutive degeneration of the lateral columns of the cord, which will be described a little further on. On the other hand, similar affections of the sphenoidal, anterior frontal, and occipital regions cause no permanent hemiplegia.

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* These terms (half-sight and half-blindness) are now usually confined to lateral division of the field—whether homologous (or homonymous) or "crossed." The terms *hemiopia*, *hemianopia*, *hemianopsia*, are used indifferently to express the same condition.

We may therefore conclude that lesions of the motor gyri, and of the fibres which run thence through the corona radiata, may cause hemiplegia, just as lesions may which destroy the external capsule, the crura cerebri, or any other section of the motor tract.

As regards the lesions of the *basal ganglia*, Charcot maintained that, whenever a lesion is limited to one of the grey nuclei of the corpus striatum (the lenticulus or the cauda), the hemiplegia is always transitory, and very often incomplete; whereas if the internal capsule is involved it is commonly complete and persistent; but he admits that neither the nucleus caudatus nor the lenticulus can be *wholly* destroyed without other parts being also affected. The limitation of paralysis to a single limb points not to a limited lesion of the corpus striatum, but to a lesion of the cortex. Jackson agrees with this statement, and to explain it, supposes that "the whole corpus striatum is represented in miniature by every single part of it." In their passage through the internal capsule, the fibres which belong to the upper limb and those of the lower may be distinguished. The cortical centre for the leg muscles lies nearer the median plane than that for the arm muscles; and, in the internal capsule, the fibres for the leg are situated further outwards than those for the arm.

Charcot believed that in affections limited to the anterior two thirds of the internal capsule there is no loss of sensation; while in those which involve its posterior third there is hemianæsthesia on the same side as the paralysis.

The bend (*genu*) of the motor tract and its posterior limb between the lenticular nucleus and the thalamus is the most frequent seat of hæmorrhage and consequent ordinary hemiplegia; next its anterior limb between the lenticular and caudate nuclei; and lastly, the external capsule of the thalamus.

After-effects.—Hemiplegia is often recovered from, the patient gradually regaining the use of his limbs, until at length no difference can be made out between the two sides. As already remarked, the rule is for the leg to recover before the arm; the patient may be able to walk with a crutch at a time when the hand is still motionless. Trousseau, however, relates two instances in each of which power was restored in the arm earlier than in the leg, and he maintains that when this is the case the prognosis is particularly unfavourable. The writer has met with one such case, and here also the leg never completely recovered.

In many cases hemiplegia is permanent. The patient may for the rest of his life be unable to move any part of his arm, or he may regain power in it to a greater or less extent.

In almost every case the affected muscles pass, sooner or later, into a state of contraction, which is known as *late rigidity*. Bouchard found it absent in only one out of thirty-two cases that he examined. It generally consists in flexion of the joints; the elbow is bent at nearly a right angle, the wrist is pronated and flexed on the forearm, and the fingers are drawn in upon the palm of the hand, so that the nails sometimes penetrate the skin and produce painful ulcerations. Much more rarely a position of extension is assumed; the elbow may then be straight, the wrist may be thrown back, and the fingers may assume the griffin's-claw attitude which was described as occurring in progressive muscular atrophy (p. 669). The leg is less apt than the arm to become contracted, even when it remains paralysed; if at all, the hip- and the knee-joints are flexed, and the heel drawn up. This form of rigidity sets in so gradually that the exact period at which it begins cannot be determined. Bouchard speaks of a case in which it was present in a marked degree two months after the apoplectic attack which had caused the hemiplegia. Sometimes one can overcome the spasm by a little traction upon the affected parts; sometimes it resists the application of even a painful amount of force. The muscles are gradually wasted from disuse; but this atrophy occurs much later, and never approaches the degree of anterior polio-myelitis.

In 1866 Bouchard suggested, in the 'Archives Générales,' that late rigidity was an indication of certain changes in the lower parts of the cerebro-spinal axis, which had been discovered by Türck in 1853, and had been previously noticed by Cruveilhier as occurring after unilateral lesions of the brain. These changes are to some extent visible to the naked eye; the crus cerebri is smaller than on the healthy side, and presents a grey streak towards its inner edge; the anterior pyramid is grey and wasted, so that the decussation of the pyramids is more conspicuous than usual. Below this point there is no marked alteration on the surface, but transverse sections of the spinal cord show, in the lateral column of the side opposite to the lesion in the brain, a triangular grey patch, which gradually diminishes in size, but is traceable down to the lumbar enlargement. Sometimes it is only after hardening and staining that the change is discoverable by the microscope. It consists in degeneration of the nerve-fibres which pass down the cord from the brain, and also in the formation of new connective tissue. In other words, there is descending sclerosis of a definite tract of the lateral column,* precisely like that which follows spinal lesions (p. 651).

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* Many nerve-fibres escape the degenerative process; and Bouchard's theory was that these unaltered nerve-fibres cause the contracted state of the muscles.

Hitzig proposed a different explanation of "late rigidity." He points out ('Arch. f. Psych.,' 1872) that prolonged rest is followed by a relaxation of the contracted muscles; that, for instance, after a night's sleep the paralysed limbs are often flaccid and supple, and remain so until the patient begins to exert the opposite limbs in getting out of bed. Again, he has observed that voluntary efforts with the unaffected arm often cause an increase in the rigidity of the affected arm. In one of his cases, as soon as the patient was made to lift a heavy weight with his left hand, the thumb and forefinger of the paralysed

Charcot believed that, once set up, the sclerosis may spread as an independent affection to the anterior grey cornua, causing the muscles to become again flaccid and to undergo a rapid degenerative atrophy; or to the posterior cornua, inducing partial anæsthesia; or, lastly, to the opposite lateral column, giving rise to a contraction of the other lower limb, as is supposed to have occurred in a case of Dr Bastian's.

Whatever its explanation, late rigidity seems to be related to certain forms of mobile spasm which may also present themselves in hemiplegic limbs, but more rarely, and only when the loss of voluntary power is incomplete. They vary in different cases. Sometimes a more or less marked *tremor* is observed, either while the limb is at rest, or only when it is made to execute some voluntary effort. Charcot, for example, mentions an instance in which the act of carrying a glass to the mouth was attended with rhythmical movements, so that the liquid in it would be spilt. In other cases *spasms* are seen, which are more or less choreiform in character, so that Weir Mitchell, who described them in 1874, called the condition "post-hemiplegic chorea."

Lastly, some cases of chronic hemiplegia are attended with slow involuntary movements, principally affecting the thumb and fingers, as described by Hammond in 1871 under the term *athetosis*. The name (*ἄθετος*, without a fixed position) is intended to signify that the parts concerned in the spasm cannot be kept still. They are constantly moving, without any voluntary effort on the part of the patient. The fingers are alternately flexed and extended, with varying degrees of adduction or abduction, so as to give the hand a peculiar aspect; and the toes may present similar changes of position. Gowers discussed the "post-hemiplegic disorders of movement" in the 'Med.-Chir. Transactions' for 1876. Those which the writer has seen have been in young patients. Athetosis is often observed without antecedent hemiplegia.

APHASIA.*—Beside hemiplegia another symptom of cerebral obstruction or hæmorrhage is Aphasia, or loss of speech, a condition of great and varied interest. The following is the account of it given more than a hundred years ago by Heberden in his famous 'Commentaries':

"The inability to speak is sometimes owing not to the paralytic state of the organs of speech only, but to the utter loss of the knowledge of language and letters, which some have quickly regained and others have recovered by slow degrees, getting the use of the smaller words first, and being frequently unable to find the word they want and using another for it of quite a different meaning—as if it were a language which they had once known, but by long disuse had almost forgotten. After an apoplectic state for several days, one person was forced to take some pains in order to learn again to write, having lost the ideas of all the letters except the initials of his two names."

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right hand became quite stiff, although they had before been free from spasm. He supposes that the contractions of hemiplegic limbs represent an excess of those co-ordinated movements in distant parts which naturally accompany every action of the body.

* It has been called *alalia* and *aphemia;* but of these names the first is of doubtful authority, and the second would mean in Greek, not loss of speech, but loss of reputation. Aphasia is the correct term: *ἀφασία μ' ἔχει* is a phrase used by Euripides, and in the form *ἀφασίῃ ἐπέων* by Homer.

To express want of power to write the term *agraphia* has been coined, and for want of ability to recognise written characters, *alexia*.

Aphasia is altogether distinct from a mere impairment of articulation, such as occurs in bulbar paralysis (p. 673), and often forms part of common cerebral hemiplegia (p. 729). That is due to imperfection in the movements of the tongue and palate, and the speech is more or less thick and difficult to be understood. Aphasia must also be distinguished from the congenital condition of a deaf-mute, and the emotional paralysis of speech which lasts only a few moments. In aphasia, if the patient can utter a word at all, he pronounces it clearly and distinctly. He may be altogether mute; or he may occasionally, when excited, ejaculate an oath; or he may possess the power of saying two or three words of a short sentence—sometimes correctly, but often only in answer to every question put to him.

Trousseau relates the case of a man named Paquet, who for four months said nothing but *cou-si-si*, *cousisi*, and who kept uttering these three syllables on all occasions, whether he was in a passion, or wished to express gratitude, or wanted to ask for something. Only, if he became very excited, he would sometimes say *sacon*, *sacon*—probably an abbreviation of the oath *sacré nom de Dieu*. Broadbent had a patient who could scarcely say anything but *Oh, shameful! shameful!—Oh, pity! pity!* Of two cases recorded by Broca, one patient had only four words, *oui*, *non*, *trois*, and *toujours*; and another said *tan*, *tan*, to every question for twenty-one years. The patient is apt to use one word for another; sometimes there is a likeness in sound between the two, as when *purging* takes the place of *perjury*, *pamphlet* of *camphor*, *dispersion* of *dispensary*, or *boat* instead of *tub*; sometimes none, as when a patient of Dr William Ogle said *two-shilling piece* for *spectacles*.

A person familiar with two or more languages may lose the power of speaking in one of them, while he retains it in the others. Trousseau gives an instance of this in a Russian, who before his illness spoke French like a Parisian; and Bastian has seen two similar instances, one in a German who had long been resident in England.

Dr Ogle ('St George's Hosp. Rep.,' vol. ii) draws special attention to the fact that grammatical form is always observed; substantives are used in the place of substantives, verbs for verbs, numerals for numerals. M. Broca's patient employed *trois* to express any number, but corrected what he said by holding out the proper number of fingers at the same time. Broadbent's patient possessed only one place-name, Burlington, where she had lived as a child.

Sometimes a patient who can utter only one or two words by himself is able to repeat others if prompted. Thus Trousseau relates the case of a man named Marcon, who could only say *Ma foi* and *Cré nom d'un cœur*, but who, when asked "Are you from the Haute Loire?" answered *Haute Loire*; and then to the query, "What's your name?" repeated *Haute Loire*. "But your name is Marcon?" *Yes, sir.* "What department do you come from?" *Marcon*; and so forth. We shall hereafter see, however, that such a case as this is not *mere* aphasia.

About 1886 we had for several months a patient in Mary Ward who had "common cerebral hemiplegia" of the right side with aphasia, and, excepting a word occasionally ejaculated at random and immediately forgotten again, her only language was the repetition of the syllables *ten-ten*—sometimes with a more open vowel sound, so as to come very near Broca's patient. She seemed intelligent, and was cheerful, taking interest in the slow and gradual recovery of her paralysed muscles; and it was curious to see how perfectly by help of her one word, uttered with perpetual variety of accent and intonation, she was able to convey acquiescence and negation, pleasure and pain, entreaty, remonstrance, and gratitude. Much pains were taken to teach her fresh words; but though she learnt one for a time, she quickly lost it again; and when after several months she left the hospital, able to walk with support and having some power over her arm, she went out repeating *ten-ten*, with a new meaning of farewell.

Agraphia.—In some cases of aphasia the patient loses the power of expressing himself in other ways as well as in speech, but is sometimes able to express his thoughts with his pen. Trousseau relates the case of a carrier of the Paris Halles, who came into the room making signs that he could not speak, and handed in a note in which the history of his illness was detailed. He had written it himself, and it was perfectly well worded. A few days previously he had suddenly lost his senses, and remained unconscious for nearly an hour. When he came to he had no paralysis, but he could not articulate a single word. In the course of five or six weeks he completely recovered; but what was remarkable was that during the whole course of the disease he could manage all his affairs by substituting writing for speech. Such cases are very exceptional. As we shall presently see, paralysis of the right hand is very often present in those who have aphasia, and of course it is then difficult for the patient to write; but we may ascertain whether the power of combining letters to form words is preserved by giving such a patient the loose wooden letters that children play with, asking him to spell his name or to put together a sentence. He may be quite incapable of doing so; indeed, he may not even be able to pick out the letters that make up the one or two words that he is perhaps able to articulate. Trousseau's patient, for instance, whose one word was *oui*, could not point to the letters *o*, *u*, *i*, in a book. This condition of inability to read or spell has been named *alexia*.

The impairment of power to write is sometimes marked when speech is but little affected. Jackson records the case of a partially aphasic patient who could talk pretty well, although she made mistakes in speaking, and called her children by wrong names; but when told to sign her name she wrote *Sunnil Sielaa Satreni*, in which there was no resemblance to the real name either in sound or spelling; and when told to write her address she put down *Sunesr met ts mer tina—lain.* If there is much impairment of the intelligence the value of agraphia as a symptom is diminished; but both Trousseau's patient with aphasia and no agraphia and Jackson's with agraphia and little aphasia appeared to have had their full mental faculties.

Again, it has been shown that in some cases of aphasia the memory of *other modes of expression* is lost, beside spoken and written language. Trousseau says that a patient may be unable to imitate the face of a person who is crying, although when he feels grief the expression of his countenance shows it clearly enough. In the case of Paquet, who said nothing but *Cousisi*, Trousseau first held out his hands and moved his fingers as though playing the clarionet, and asked this man to do the same. Paquet immediately executed the same movements with perfect precision; and when asked whether he knew that the attitude was that of a clarionet player he would assent by nodding his head. Yet when told, a few minutes afterwards, to do it again, he often was unable.

An aphasic patient whose case was carefully studied by Dr Scoresby

Jackson ('Edin. Med. Journ.,' 1867) was totally unable to play the piano. He put himself in the proper position, and placed his fingers on the keys, but he could not play a single bar, not even of a piece of music with which he had been familiar before his illness; yet he could hum the same tune pretty well. He played draughts with skill; and Paquet also could play backgammon and dominoes perfectly, and would cheat when he found himself losing.

Amnesia.—It is important to notice that the very words which an aphasic patient is unable to utter may be perfectly understood by him when spoken by another person. This, indeed, is but an illustration of a broad distinction which is traceable throughout the faculty of speech. A child learns the meaning of words addressed to it by others long before it can itself speak. A person partially acquainted with a foreign tongue recognises many words, if spoken distinctly by anyone else, which he would have been unable to call from his memory if he wanted them in conversation. Nay, we all of us understand the meaning of many English words when we hear them which we never use ourselves. Adopting terms used by Dr Moxon, we may say that there is a great difference between "incoming" and "outgoing" language; or, following Dr Broadbent, that words are to be considered in two distinct aspects: first as "intellectual symbols," and then as "motor processes." In ordinary aphasia and agraphia it is the *outgoing language* and *motor processes* that are interfered with; in amnesia and alexia it is the *incoming language* and *perception* of intellectual symbols.

The same distinction is denoted by Wernicke's terms; *motor* (ordinary) *aphasia*, including agraphia, and *sensory aphasia*, including alexia. Charcot's "verbal and graphic amnesia" describe the auditory and visual forms of sensory aphasia which are denoted by "word-deafness" and "word-blindness" respectively.[*]

The local lesion.—It has now been established that aphasia—including agraphia—is almost invariably dependent upon a lesion of the *left side* of the brain. This was first stated by Broca as the result of a case which he published in the 'Bulletin de la Société Anatomique' for 1861.[†] The association, though previously overlooked, is beyond question. If a large number of hemiplegic patients be taken, it will be found that many of those paralysed on the right side are speechless; probably every one whose left arm and leg are paralysed will be able to articulate as distinctly as the condition of the tongue and lips will allow. In much rarer cases aphasia will be found to exist without any paralysis, and the lesion is almost invariably in the left side of the brain.

Lastly, to complete the argument, the exceptions themselves illustrate the rule, for in left-handed persons (whose right hemisphere takes the precedence in movements which the left does in the rest of mankind[‡]) loss of speech goes with paralysis of the left limbs, and not with that of the right limbs; or, in other words, it depends upon disease of the right hemisphere instead of the left. Examples of this association were recorded by Dr Hughlings Jackson ('Med. Times and Gaz.,' 1866), Dr John Ogle ('Lancet,' 1868), and Dr Wadham ('St George's Hospital Reports,' 1869), and two well-marked cases have occurred in the writer's experience: one about 1870, a man in Stephen Ward with Left Hemiplegia, who, to his surprise, was aphasic; and the other a boy of eleven in John Ward, in 1883, who had Right Hemiplegia and spoke perfectly.

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[*] It may be that perception of the meaning of the words spoken by others, and uttering words for one's self, are functions of different parts of the brain. Or, again, it may be that both "incoming" and "outgoing" language have their seat in the same locality, but that the latter requires for its execution some subordinate nervous centres which have nothing to do with the former. The second of these hypotheses was defended with great ability by Dr Broadbent in the 'Med.-Chir. Transactions' for 1872.—C. H. F.

[†] It was stated nearly thirty years after the event that Dr Marc Dax, of Sommières, read a paper on the subject before a Medical Congress at Montpellier in 1836, but the memoir has never been seen since. Its title was "Lésion de la moitié gauche de l'encéphale coïncident avec l'oubli des signes de la pensée." Certainly no one, either in Paris or in London, was acquainted with the views of M. Dax when Broca published his paper.

[‡] The relation of the left side of the brain to movement and speech was discussed by

Further, it was proved by Broca that aphasia is caused by lesions of a particular part of the surface of the brain on the left side. Bouillaud was long ago led by clinical observation to connect the faculty of language with the *anterior lobes* of the brain,* and, indeed, Gall had previously suggested the same localisation. But it was not until a second of Broca's cases was published, in November, 1861, that anything like precision of localisation was arrived at in regard to loss of speech. The case in question was that of a man named Lelong, aged 84, who after an attack of apoplexy lost the power of uttering words, with the exception of four, although he knew all that was said to him, and could make himself understood by gestures. He died at the end of a year of a fracture of the femur. At the autopsy the posterior third of the second and of the third left frontal convolutions were found to have been destroyed by softening, their place being occupied by a secondary hæmorrhagic cyst.

Ever since, the *third left frontal* has been known as Broca's convolution, and it is now admitted that motor aphasia always depends on a lesion involving that region, if we limit it to the posterior part of the third left frontal gyrus, and extend it to the back of the second frontal and adjacent parts of the ascending frontal and *gyri operti*.

It appears that no disease of the corpus striatum, or even of the internal capsule, is capable of causing loss of speech. The general accuracy of this assertion is proved by the fact that cerebral hæmorrhage, in the position in which it usually occurs, leaves the speech unimpaired. In 1876 Broadbent ('Brit. Med. Journ.,' i, p. 436) knew of no case on record in which lesion of the corpus striatum alone had given rise to this symptom. At first sight this seems inexplicable, since it is difficult to see how any effect can be produced by destruction of a convolution which may not equally follow the division of those conducting fibres which connect it with the basal ganglia, or even with the spinal centres. Broadbent meets the difficulty by supposing that there are two distinct routes by which impulses pass from Broca's convolution to the nerve-nuclei in the pons, to the cortex, and to the spinal cord. One is the straight path through the left corpus striatum, the other is by commissural fibres which go to the third frontal convolution on the opposite side, and thence down through the right corpus striatum. So long as either of these routes remains open, speech is possible; it being, of course, assumed that the nuclei on opposite sides are so closely associated together by cross connections as to be set in action simultaneously. It follows from Broadbent's hypothesis, that if hemiplegia and

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e writer in the 'Guy's Hosp. Reports' for 1870, and more fully and satisfactorily by r Wm. Ogle in a paper on "Dextral Pre-eminence" ('Med.-Chir. Trans.,' 1871).

* Bouillaud, as M. Broca handsomely admitted, had also recognised the special relation the left side of the brain to spoken language. "N'est-ce pas que nous écrivons, nous ssinons, etc., de la main droite? . . . eh bien, serait-il absolument impossible que pour rtains actes aux quels sont affectés les hémisphères cérébraux—la parole, par exemple—ous fussions pour ainsi dire gauchers?" (Discours à l'Acad. imp. de Médecine, 1865.)

aphasia occur together, the lesion must be one which either involves simultaneously Broca's convolutions and the corpus striatum, or else affects an extensive area of the left hemisphere in the neighbourhood of the fissure of Rolando. Whichever is the case, the cause of the disease must almost always be obstruction of the Sylvian artery, whether by embolism, thrombosis, or syphilis.*

Again, aphasia cannot be caused by a lesion of any part of the left hemisphere, except Broca's region. No doubt cases are very rare in which a morbid change is limited to a single spot on the convexity of the brain. But a woman at Guy's Hospital suffering from cancer of the breast had transitory attacks of aphasia; it was conjectured that she had a secondary nodule in the third frontal convolution on the left hemisphere of her brain: and this diagnosis was afterwards verified by an autopsy. As Broadbent observed in 1872, it is remarkable how large a proportion of apparently exceptional cases break down under careful scrutiny.

It is probable that the centre for sensory aphasia, or word-deafness, is in the superior temporo-sphenoidal convolution, and that for agraphia, or word-blindness, in the angular gyrus and occipital cortex. Agraphia without aphasia would depend on a destroying lesion of the centre for the right hand in the left ascending frontal and adjacent part of the middle frontal convolution.

Theory of aphasia.—Reverting now to the physiological causation of aphasia, we ask why loss of speech is always dependent on a lesion of the surface of the left hemisphere—except in left-handed persons, in whom, as we have seen, the lesion is in the corresponding part of the right hemisphere. The late Dr Moxon first threw light upon this curious fact, and suggested an explanation which has virtually been accepted by subsequent writers. His view ('Med.-Chir. Rev.,' 1866) is that the two halves of the brain originally resemble one another in their functional capacities, but that in the course of education one side only becomes stored with those ideas of associated movements which are required for the performance of bilateral actions, such as are concerned in speech; or, as he elsewhere puts it, one side of the tongue *guides* the other; just as the right hand guides the left one when they are made to execute similar motions, for this requires far less attention than the execution of opposed motions by the two hands at once. Dr Moxon did not explain why the process of education should take place on one side of the brain rather than the other; but, as we have seen, the associated movements which make up the act of writing, and in which only one hand is concerned, are so closely connected with those of speech, that agraphia often accompanies aphasia. Now it is true that everyone does not write; but perhaps even a savage from his earliest infancy employs one hand rather than the other in the gestures by which he aids speech, and the hand of the orator is the right hand. Broca himself surmised that this preference of the left hemisphere for so many other purposes led to the education of convolutions of that hemisphere for the "motor processes concerned in speech."

Theory of amnesia.—We have still to ask what are the relations of aphasia to "incoming" language—that part of the faculty of speech which consists in the recognition of words spoken by others. Most modern writers, following Dr Sanders and Dr William Ogle, admit a separate variety of aphasia, which they term *amnesic* or *amnemonic*. The characteristic symptom of it is that the patient is able to utter words, provided that he has first heard them spoken by another person; in fact, one may be able by prompting to make him speak pretty freely, although he may be incapable of saying anything in reply to a question which does not suggest its own answer. But the value of this test is limited to the cases in which it yields a positive result; if the man continues speechless, after one has repeated again and again the word or the sentence which he is asked to utter, one learns nothing by the experiment. That the memory for "incoming" language is not altogether lost may, indeed, sometimes be gathered from the gestures of intelligence which such a person makes if the right word is suggested to him, and from his strongly marked dissent when other words are substituted. Moreover, in those cases in which an attack of aphasia rapidly passes off, the patient may be able to give a complete account of all that occurred during his illness, and to state that his power of thinking was unaffected. Prof. Lordat, for example, who once suffered in this way, afterwards stated that he was able to combine and to distinguish abstract ideas. He thought over the subject-matter of a lecture, and found that he could dispose in his mind the chief points without difficulty, and introduce any changes that he pleased. And yet, when he thought of the Doxology, he was not able to recollect a single word. We must leave it to the metaphysicians to discuss how far it is possible for the mental processes to be carried on without conscious revival of words; but it is clear that in M. Lordat's case the fault lay in the machinery of expression alone—always supposing that he was able to recall past states of consciousness with complete accuracy.

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* If in a given case plugging of the Sylvian artery should be overlooked it might easily be supposed that the only disease was in the corpus striatum, and that this had caused the aphasia. In July, 1877, I made an autopsy in which it would have been quite possible to commit this mistake; the patient was aphasic, and the only obvious lesion of the brain was in the corpus striatum; but the Sylvian artery contained an embolism at its very commencement.—C. H. F.

When aphasia is incomplete, so that the patient can utter one or two words, and uses them in answer to every question, he is often perfectly conscious of his mistakes. Every physician has seen such cases, and will remember the shake of the head, the puzzled look, the smile—half amusement, half vexation—the repeated attempts to find the right word, and the beam of satisfaction if by chance it comes off the tongue at last.

These facts seem to prove beyond dispute that the memory of language may be unimpaired, even when there is complete aphasia.

Mental condition in aphasia.—Let us now look at another side of the question. Many patients affected with partial aphasia go on uttering the same word for months or years, without seeming to know that anything is amiss with them. Trousseau studied very carefully the case of an artist who, according to his own account, suffered from nothing but failure of speech, and was able to understand perfectly all that he read. It turned out that he made the grossest mistakes in reading aloud, that he wrote one word for another, and that when asked to sketch a human figure he drew like a child who had never been taught. In another case Trousseau found that the patient, who read the newspaper, and expressed by signs that he understood it perfectly, was nevertheless in the habit of reading the same page over and over again in the same day. A third patient, a girl under his care, had for a whole year one book in her hands, and almost always read the same page. He advised as a good test of the understanding of an aphasic patient, to take up a book and read a few lines, telling the patient to follow with his eyes, and to turn over at the proper moment; the artist, whose case has already been referred to, could never do this correctly. Trousseau concludes, in spite of the facts mentioned in the previous paragraph, that in aphasia there is not merely loss of speech, but also impairment of intelligence. The lesions which cause aphasia are in most cases such as interfere with the supply of blood to a considerable area of one hemisphere, and therefore it is not surprising that they often produce mental symptoms independent of loss of speech.

That other symptoms have no connection with the aphasia is proved by the fact that there is often *anosmia* in the corresponding nostril. In the 'Med.-Chir. Transactions' for 1870, Dr William Ogle refers to seven instances of this: he supposes that the so-called external root of the olfactory bulb becomes implicated when it is passing to the floor of the fissure of Sylvius.

The question, however, is—not what defect of understanding *may* be found in aphasic patients, but what amount of intelligence they ever retain. Instances of pure aphasia, unattended with paralysis of the limbs, are especially worthy of study in reference to this question. In such cases, while the loss of speech is often absolute, the intellect may be quite unimpaired.

The following is a striking instance:—"About three in the morning, as near as I can guess, I woke and sat up, when I felt a confusion and indistinctness in my head, which lasted, I suppose, about half a minute. I was alarmed, and prayed God that, however He might afflict my body, He would spare my understanding. This prayer, that I might try the integrity of my faculties, I made in Latin verse. The lines were not very good, but I knew them not to be very good. I made them easily, and concluded myself to be unimpaired in my faculties. Soon after I perceived that I had suffered a paralytic stroke, and that my speech was taken from me. I had no pain. . . . I then wrote to Dr Taylor to come to me and bring Dr Heberden. . . . In penning this note I had some difficulty; my hand, I know not how or why, made wrong letters" (Dr Johnson to Mrs Thrale, June 17th, 1783, *anno ætatis* 74). He recovered quickly in a few days from this attack of aphasia with partial agraphia, but without motor paralysis; and the cerebral hæmorrhage (for such no doubt it was) did not return.

But after all, cases like those of Dr Johnson and Prof. Lordat are the exceptions. One sees patients with right hemiplegia who go on for years unable to utter a single word, or to communicate in any way with their friends. It would be sad to suppose that such persons are really in possession of all their mental faculties, and that they are living, as it were, imprisoned behind an iron mask. Happily this is as unlikely as it would be melancholy, for these patients show often by their gestures that they are not miserable, and in most cases they are obviously impaired in intellect. Moreover, those who suffer from left hemiplegia, dependent upon any extensive lesion of the right hemisphere, manifest an equally marked deficiency of intelligence, although they are not deprived of speech.

These considerations lead to the conclusion that, although loss of speech is often associated with inability to understand "incoming" language or recognise mistakes made in "outgoing" language, yet the two things are essentially independent of one another, and that when aphasia associated with amnesia indicates a lesion, we may conclude that the lesion extends beyond the limits of Broca's region.

Broadbent arrived at nearly the same conclusion (cf. 'Med.-Chir. Trans.,' 1872, p. 174). He maintains the truth of Broca's theory, so far as concerns the seat of the lesion when aphasia occurs independently of any failure of intelligence. This theory, however, does not imply that the whole of the faculty of language is localised in any one part of the hemispheres; it only asserts that a certain spot in the left hemisphere contains machinery without the use of which a person cannot utter words, nor convey his thoughts in writing.

Recovery from aphasia.—Although a patient, after a paralytic stroke with aphasia, regains his speech soon after consciousness is restored, and although he regains the articulation which has been damaged before the power in his leg, and long before that in his arm—yet when a patient with right hemiplegia continues aphasic, his recovery of speech comes, as a rule, latest of all, when there only remains slight awkwardness in the fingers or an almost imperceptible halt in the gait.

There is reason to believe that when Broca's region has been destroyed the power of speech may still be recovered, not by regeneration of the lost tissue, but by the corresponding part of the right hemisphere taking on the action of the left; just as a draughtsman after losing his right hand would, in learning to draw with his left, be educating not only his muscles and nerves, but also the motor centres of his right hemisphere.

The writer had several years ago under his care in Stephen Ward a young man who was admitted with complete aphasia and right hemiplegia, probably the result of syphilitic stenosis of his left Sylvian artery. While slowly regaining power of movement and improving in health, the sister of the ward tried to teach him to speak, and found the only way was to begin from the beginning, by naming objects (and printed words—so as to teach him to read again also), and making him imitate sounds over and over again. The result was a good degree of success.

In the same way the monophrasic woman mentioned before, who said *ten-ten*, was taught by two successive clinical clerks to print letters with her left hand, and to recognise printed letters. Here the success was limited, perhaps owing to the greater age of the scholar or the smaller patience of the teachers.

Aphasia in lunatics.—There is still one question which it is necessary to consider: is aphasia, when it occurs in persons of unsound mind, invariably dependent upon a lesion in Broca's region? Dr Bastian mentions the case of a lunatic who did not utter a single word for several years, except during two brief intervals, once when he had an attack of pleurisy that lasted some days, and once when he was suffering from toothache. The late Dr Forbes Winslow met with a similar instance; a person who had been insane for fifty-two years did not speak during thirty years, but recovered speech and answered questions perfectly well during the last fifteen years of his life. In such cases there is surely no reason to suppose that there was any local lesion in the brain.

The same may be said of some cases which Trousseau gives as examples of aphasia; for instance, that of the lady who welcomed her visitors by exclaiming *pig*, *animal*, *fool*, without understanding the meaning of her words; and also of an example of agraphia in a lunatic at Broadmoor, where the inability to write was of an amnesic type, and may be regarded as one of the symptoms of mental disorder ('The Brain as an Organ of Mind,' 3rd ed., p. 660).*

Apoplexy.—This term originally signified a "stroke"† by which the patient falls "like an ox struck down by the butcher;" and we shall here use it in this, its proper sense. Unfortunately the word was transferred by metonymy to effusion of blood upon the brain, which was found to be the most common cause of such attacks when autopsies began to be made; and this use of the word was afterwards extended to hæmorrhages in other parts, so that pulmonary apoplexy, retinal apoplexy, and apoplexy of the supra-renal capsules are spoken of. There is no advantage in miscalling apoplexy as a mere synonym for hæmorrhage; and in clinical medicine it is desirable to have a name for the sudden attack of insensibility which is due to suddenly increased pressure within the cranial cavity—usually but not always rupture of an artery—as distinguished from failure of the heart's action or *syncope* on the one hand, and on the other from narcotic or alcoholic poisoning, uræmia, epilepsy, or external injury.

Symptoms.—A patient in an apoplectic fit lies "deprived of sense and motion." He is not asleep, for he cannot be roused; but there may be varying degrees of insensibility. Sometimes the well-known voice of a wife or daughter may elicit an unintelligible muttering in reply, or a spoon put to the lips may cause them to be closed, or one hand may be raised to rub the side of the face. More often there is not even partial consciousness; the limbs remain in whatever position they happen to lie, and no stimulus excites a response; the breathing and the beating of the heart alone indicate life.

The pupils are torpid, or insensible to light; they are sometimes dilated or minutely contracted, and often unequal. In severe cases of apoplexy, the conjunctivæ can be touched without exciting reflex movement in the eyelids, and the cutaneous reflexes are diminished or abolished. Pinching or pricking the skin seldom leads to any sign of consciousness, but sometimes the hand or foot is drawn away on one side and not on the other, or when the arms are lifted and allowed to drop upon the bed, one only falls as a dead weight; or the arm and leg on one side may be rigid. These are important indications of hemiplegia, as is also the conjugate deviation of the eyes described above (p. 730).

The countenance sometimes is pale, sometimes congested and purple, with livid lips and tongue, while the features are often swollen, and the forehead bathed in perspiration.

The *temperature* is chiefly important for prognosis. As Bourneville found, there is at first a slight fall, about 2° Fahrenheit. In rapidly fatal cases a normal or subnormal temperature continues until death: but if life is prolonged the temperature rises to 99° or about 100°. When death occurs at an interval of more than ten hours from the commencement of the attack it is often preceded by a rapid elevation of temperature, and this is always an unfavourable sign. In one fatal case at Guy's Hospital the thermometer registered 107°. When recovery is to take place, the temperature, if it has risen, returns to the normal point two or three days after the attack began.

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* The reader is referred for a full and interesting discussion of aphasia and its varieties to the Lumleian Lectures of Dr Bastian before the Royal College of Physicians in 1897 ('Lancet,' April 3rd—May 1st, 1897).

† *Gr.* 'Αποπληγία, ἀποπληξία, a stroke, a classical Greek word from ἀποπλήσσω, to strike, to disable (Soph., 'Antigone,' v, 1189, where it is applied to syncope). Used by Hippocrates of a "stroke" of the palsy, but applied to one limb, as σκέλος ἀποπληκτικόν, a paralysed leg.—*Lat.* Sideratio.—*Fr.* Coup de sang.—*Germ.* Schlagfluss.

The *pulse* may be either increased or diminished in frequency; the prognosis is bad if it is greatly above or greatly below the average—say below 60 or above 120. Unfortunately, the converse is not always true. As a rule the pulse becomes more rapid as the case goes on towards a fatal termination; but a still worse sign is irregularity, a succession of frequent beats followed by a series of beats at long intervals. Formerly great stress was laid upon the "full" and "labouring" character of the radial pulse in apoplexy, and it was supposed to indicate venesection. We now refer this character either to want of elasticity from atheroma of the aorta, or to the high arterial tension of chronic Bright's disease.

The *breathing* is often infrequent; there may be as few as three or four respirations in the minute. Towards the last the patient may cease to breathe for perhaps a minute, and a purple flush appears over the countenance, but afterwards a deep breath is again drawn, the face resumes its natural colour, and the respiration goes on as before. This may be repeated several times, until at length the final breath is drawn; but the heart may go on beating for a considerable time still. In some cases the respiration assumes the characters described by Cheyne and afterwards by Stokes, and known by their names.

In many cases apoplexy destroys life by a more gradual increase of obstruction to the breathing. One cause of this is the supervention of congestive œdema, beginning in the bases of the lungs and spreading upwards through their dependent parts—so called hypostatic pneumonia. Another is the accumulation of a thin mucus in the air-passages, which forms a froth with the inhaled air, and may fill the bronchi and even the trachea. Dyspnœa, again, is often caused by the entrance of fluid nourishment into the air-passages; for when a patient is insensible, milk and beef-tea and brandy are very apt to run down into the larynx without giving rise to any warning cough or sensation of choking.

Yet another important cause of dyspnœa is the impediment to the act of respiration which is due to paralysis of the tongue and fauces. Everyone who has made many examinations on the bodies of those who have been suffocated knows how large a quantity of frothy fluid is found in the air-passages, even when death has been rapid; but an apoplectic patient may lie for days in a condition of dyspnœa. This is shown not only by the lividity of his countenance, but also by the *stertor* that accompanies the entrance and exit of air. The causes that lead to the occurrence of this sound were clearly pointed out by Dr Bowles, in the 'Med.-Chir. Trans.' for 1860. He admits that when the mouth is partially open the soft palate sometimes drops upon the tongue, and vibrates as the air rushes beneath it; but he ascribes far more importance to a change in the position of the tongue itself; he shows that when the mouth falls open, the point of attachment of the lingual muscles to the symphysis is carried backwards, and he thinks that the tongue then comes into contact with the posterior wall of the pharynx. As might be expected, this is most apt to occur while the patient lies supine. As Dr Bowles says, turning him over on his side, with the mouth inclined so that the saliva and mucus can drain away, is often followed by disappearance of stertor and by decided improvement in other symptoms.

When the breathing is noisy the cheek is often puffed out at each expiration, in consequence of paralysis of the buccinator muscle; this symptom is unfavourable.

Duration.—The period that an apoplectic seizure lasts is very variable. In some exceedingly rare instances death takes place in a few minutes. Abercrombie relates how a woman who seemed to be in perfect health was one evening at a crowded meeting, when she uttered a loud scream, and fell down insensible. She was immediately carried out, remained pale and unconscious, and within *five minutes* she was dead. Hughlings Jackson mentions a similar case of a woman who was sitting at the tea-table, when she stopped in the middle of a laugh, cried out, "Oh, my head!" fell back in her chair, and died within not more than *five minutes.* In both these cases meningeal hæmorrhage was found.

In 1864 there was brought to Guy's Hospital the body of a woman who had died in a few minutes as she was returning home from the theatre with her children; in that instance also a large quantity of blood had been poured out over the sides and base of the brain.

Dr Fagge once found at an autopsy a large clot in the left hemisphere, bursting into the lateral ventricle. The patient, a man aged forty-one, was in a surgical ward for some laryngeal affection, and had been sent down to the dispensary to fetch the medicines; on his way he had a fit, became comatose, and died within *ten minutes.*

One would have expected that hæmorrhage into the pons would often cause instantaneous death. But Jackson remarks that he has never seen such a case, although he has known a woman lie deeply comatose for *some hours,* in whom this part was afterwards found hollowed out into a mere shell. In the most rapidly fatal case of a clot in the pons recorded at Guy's Hospital death occurred in *forty minutes;* two patients lived each for two hours, one nine hours and a half, one thirteen hours, one sixteen hours, and one *two days.*

Of seventy-eight cases of hæmorrhage into the pons collected by Bode and quoted by Ross, forty-six proved fatal within *twenty-four hours.*

Meningeal, like pontine, hæmorrhage commonly proves fatal within forty-eight hours. But in the ordinary form of apoplexy, in which blood is effused into the neighbourhood of the corpus striatum, life is often maintained for a much longer period. In twelve such cases at Guy's Hospital death did not occur until between the second and the seventh days; and in six cases at the end of ten, twelve, thirteen, sixteen, nineteen, and again nineteen days respectively. It is worthy of remark that two among these six cases were examples of a very rare occurrence, the formation of a clot within the substance of the brain, which was large enough to destroy life, but which yet failed to reach either the lateral ventricle or the surface; and in a third case the seat of the hæmorrhage was quite exceptional, being the interior of one occipital lobe.

In twenty-four consecutive fatal cases of cerebral hæmorrhage under the present writer's care, death took place under twelve hours in seven, under twenty-four in eleven; on the second or third day in three; on the fourth, fifth, or sixth in eight; and on the ninth in two.

In many cases after an apoplectic attack, consciousness is regained more or less gradually. After a few hours one is perhaps able to rouse the patient so that he will give his name, although when left to himself he still takes not the slightest notice of anything. For several days he generally remains drowsy and apathetic, or if he talks his ideas are confused and perplexed. Occasionally he is delirious, talking incoherently, and trying to get out of bed, and in such cases the prognosis is not good, in spite of the fact that the coma has passed off. Nothnagel says it is exceptional for recovery to occur if insensibility persists for as long as forty-eight hours. In cases which terminate favourably, as the patient regains consciousness, hemiplegia, with or without aphasia, becomes manifest.

Prodroma.—The older writers laid great stress upon certain symptoms which they believed to be frequent precursors of apoplexy, and which they therefore designated "warnings" or "molimina." Under these names they included:—epistaxis, ecchymosis of the conjunctivæ after coughing or sneezing; and the frequent recurrence of giddiness in a person advanced in years. These last symptoms are no doubt likely to be due to disease of the cerebral arteries, which may soon lead to their rupture; and arterial disease may lead to other nervous disorders—such as thickness of speech, diplopia, partial ptosis, sensations of numbness and formication in the hand or fingers, partial loss of power in the arm, or dragging of the foot. If any of these occur in an old man or woman, one cannot overlook its possible significance as a warning of apoplexy.

Indeed such symptoms are often the results of a small cerebral hæmorrhage, which has already taken place, and precede the effects of a larger one.

Onset.—Abercrombie, many years ago, observed that cerebral hæmorrhage comparatively seldom leads to sudden loss of sense and motion, or (in other words) to the classical form of apoplectic seizure. A precise clinical history of the cases in which extravasation of blood is most apt to be found is given in his celebrated work. The first symptom, he says, is a pain in the head. The face becomes pale, the body cold, and the pulse very weak; there is nausea, or even vomiting; and he may fall to the ground faint and exhausted. Often a slight convulsion occurs. After a little while he may be able to walk home; he is quite sensible, but oppressed; he remains cold and feeble, with cadaverous pallor of the countenance. By degrees he recovers his warmth, his face regains its natural appearance, and his pulse improves in strength. Then he becomes flushed, answers questions slowly, and gradually sinks into coma, from which he rarely recovers.

All subsequent writers have recognised the truth of this picture. After fracture of the skull, when the middle meningeal artery is torn through, there follows a very similar series of events; and the name of "ingravescent apoplexy" has been given to cases which run such a course.

Cerebral hæmorrhage seldom produces sudden coma. No doubt the patient is often insensible when first seen; perhaps he is picked up in the street, or found lying on the floor, or in a water-closet; or the seizure may have come on during sleep. But Trousseau says that although his attention was for fifteen years directed to the question, he did not in that time meet with a single instance in which, when an attack of apoplexy due to cerebral hæmorrhage occurred in the presence of witnesses, it did not begin more or less gradually. An exception must be made for those rare cases which destroy life in a few minutes (p. 746), and another exception is allowed by Trousseau himself for certain cases which begin with an epileptiform seizure, an exception endorsed by Hughlings Jackson. But ingravescent, not sudden, apoplexy is the rule in cases of large cerebral hæmorrhage.

It must, however, be admitted that the symptoms before coma sets in are more variable than would appear from Abercrombie's description. Pain in the head is often, perhaps most often, absent. Not infrequently the only thing which is noticed by the patient is a numb feeling, or a sense of weight in one of the limbs. Thus Trousseau relates the case of a woman who while returning from market noticed that she dragged her right leg, and that her right arm felt heavy, so that she changed into her left hand a folded newspaper which she was carrying, lest it should fall into the mud; she walked upstirs into her room, took off her clothes, and got into bed; after which she became hemiplegic and comatose, and remained in a state of stupor for three days. Another patient of Trousseau had noticed while at dinner that one of his hands felt heavy; he was not giddy, but faltered a little in his speech; he tried to rise from his chair, but one of his legs being paralysed he fell down. His children lifted him up, and with their help he walked into the next room, where Trousseau found him in three quarters of an hour perfectly conscious; but his left arm and leg were almost powerless. Coma set in in a few hours, and he died the next morning.

Convulsive movements are only occasionally observed in cases of genuine apoplexy from cerebral hæmorrhage—not *congestion cérébrale apoplectiforme*. Dr Pitt found it twice in 79 cases at Guy's Hospital. It is usually, but not always, limited to the paralysed limbs, and when the head is affected by this clonic spasm it is turned away from the seat of hæmorrhage, *i.e.* towards the paralysed side.

Among the early symptoms of cerebral hæmorrhage is a transitory loss of consciousness, from which the patient quickly recovers, so that it is separated by an interval from the final coma. To this Trousseau gives the significant name of "cerebral surprise." The writer once saw a striking instance of this kind. He was sent for to an old lady who, while at supper, "became faint" and slid from her chair to the ground. When he arrived she was on a sofa, and said she was much better, but within a few minutes she became again unconscious, and died in deep coma a few hours later. Next day he found a large hæmorrhage into the ventricle.

The probable explanation is that the first extravasation of blood acts as a shock, and stuns the patient as with a blow; the heart is faint, the blood-pressure falls and the hæmorrhage is arrested. After an interval the heart recovers, fresh hæmorrhage takes place and produces the ordinary ingravescent coma. In some cases the coma is preceded by delirium.

Diagnosis of the seat of the lesion.—Speaking generally, the most probable seat of cerebral hæmorrhage is the most common one—the region of the corpus striatum, and particularly the external and internal capsules (cf. *supra*, p. 724). If a second or third attack has proved fatal, there is very likely ventricular hæmorrhage. If death has come on unusually rapidly there is probably either moderate effusion in the pons, or a very large one on the surface or into the ventricles.

The fact that lesions affecting the cerebral convolutions so often give rise to convulsive seizures has led some observers to suppose that such symptoms are especially apt to occur in cases of *meningeal hæmorrhage*, but our cases do not bear this out. Nor is rigidity of the limbs noted having been frequently present—a fact which Dr Goodhart pointed out the 'Guy's Hospital Reports' for 1876. It seems, however, that the hemiplegia is not often observed in this form of apoplexy; the blood its way too easily along the subarachnoid space at the base of the brain to compress one hemisphere more than the other.

It is doubtful whether extravasation of blood into the *lateral ventricle* can be recognised at the bedside. The recurrence of coma, after recovery from a first seizure, was attributed to this cause by Nothnagel; but we have seen that it is common in all cases of apoplexy. Others have supposed that the presence of ventricular effusion is indicated by paralysis of all four limbs succeeding to hemiplegia; or by early rigidity of the paralysed arm and leg, as maintained by Durand-Fardel. But neither supposition will bear the test of *post-mortem* experience. In 1874 Dr Fagge made an autopsy in the case of a lad, aged seventeen, who had died fifteen hours after having been found lying on the ground unconscious. Not only both lateral, but also the third and fourth ventricles, were full of clot, which was in equal abundance on the two sides. No cause for the hæmorrhage could be found; but there had been repeated epileptiform fits. In 1876 an old woman died in the Clinical Ward of Guy's Hospital of apoplexy without hemiplegia. The right ventricle was full of clot, which had come from the superficial part of the caudate nucleus, the internal capsule being quite uninjured.

In hæmorrhage of *the pons* the pupils are often contracted to pins' points, and the respirations are infrequent—perhaps not more than six or four to the minute; but occasionally we have seen dilatation of the pupils, and the breathing is sometimes hurried and irregular. Large hæmorrhage into the pons almost always produces deep coma with flapping cheeks and insensible pupils. Death is not instantaneous: ten minutes appears to be the shortest period on record—thirty or forty minutes to four or five hours the most common; but occasionally death may be delayed for twenty-four hours and, in a case of the writer's, for forty-eight. Hæmorrhage into the pons sometimes leaves behind it a permanent difficulty of articulation. Crossed hemiplegia can seldom be made out satisfactorily while the patient is insensible, but if he recover from coma this symptom proves that the lesion is unilateral in the pons or the upper part of the bulb.

Hæmorrhage into the *cerebellum* is remarkably rare, and very seldom large; if, however, it occurs near the surface and breaks into the fourth ventricle, death quickly ensues from compression of the respiratory centre of the bulb. The diagnosis of local lesions is more exact in cases of tumour than of hæmorrhage (cf. *infra*, pp. 786-8).

General Diagnosis.—In the diagnosis of apoplexy one has to bear in mind many possibilities; in few diseases is it more important or more difficult to form a right judgment. Error may arise in two ways: sometimes from the absence of any trustworthy history; sometimes from the doubtful nature of the symptoms.

Thus a man may be discovered comatose and stertorous in bed or on the pavement, and there may be no one to say what has happened; often it is impossible to depend upon the statements of those in whose company he is found; and sometimes there is suspicion of foul play. In all such cases the following possibilities must be taken into careful consideration before deciding that the case is one of apoplexy: that the patient's brain has been injured by external violence, as by a blow or a fall, with or without fracture of the skull; that the disease is abscess, tumour, or meningitis; that he has taken narcotic poison; that the poison is alcohol; and that the coma is due to uræmia, to diabetes, &c., or to the effects of an epileptic fit.

1. As regards *injury to the brain*, difficulties arise in several ways.

There may be no bruise upon the face or head, and no displacement of any part of the calvaria, so that it is only at the autopsy that we discover the skull to be fractured, or the brain on one side bruised. Even then, one is by no means justified in concluding that the case was originally one of accident or of injury inflicted by others. The patient may have had a fit or been drunk, and in falling may have fractured his skull. A man was once admitted into Guy's Hospital who was driving in a cart, when he fell and was picked up insensible. He died after four days, and the surface of the brain was found to be extensively bruised; but as he was known to be a subject to epilepsy, it appeared that one of these attacks had been the cause of his falling.

We must bear in mind that meningeal hæmorrhage often occurs as the result of disease; it is, therefore, only when the brain-substance itself is ecchymosed that we are justified in attributing to external violence cases in which blood is found effused on the surface of the brain. On the other hand, we have had several instances in which a severe injury of the skull with laceration of the cerebral convolutions has been associated with hæmorrhage into the central parts. Some of these have undoubtedly been examples of primary hæmorrhage, the superficial injuries having been caused by the patient's falling upon a kerbstone or the like; but it is certain that a fall or a blow upon the head may in rare cases cause rupture of an artery in the interior of the brain.

In 1855 a patient was admitted who had been found lying by the side of a crane, the handle of which seemed to have struck him on the head, for there was a wound of the scalp. At the autopsy the left corpus striatum and thalamus were found to be destroyed by a large mass of blood lying between them.

In 1862 a man was admitted who had fallen several feet upon his head from a platform on which he had been working. His skull was found fractured, but the lateral ventricles were full of blood, and the right corpus striatum was broken up by a large clot which projected through an opening in its surface. His fellow-workmen thought he had overbalanced himself, as he had shown no signs of having a fit; but the question was left open in Dr Fagge's report.

In 1866 a man was admitted into a surgical ward an hour before his death who had been steering a barge on the Surrey Canal, when a steamer came too close and struck the rudder; the tiller knocked him over, and he was picked up insensible. There was no injury to the cranium, nor to the surface of the brain. The right lateral ventricle was found full of effused blood, the septum was broken down, and there was a moulded clot in the fourth ventricle.

In 1868 a patient in the ophthalmic ward missed his footing, fell, and struck his left temple. A large quantity of blood was effused upon the cerebral convolutions, but the right corpus striatum was also extensively ecchymosed, and had in its interior a mass of blood of the size of a hazel-nut.

In 1870 a man came to the hospital with a cut on his forehead, saying he had slipped up on a flight of stone steps at the Victoria Theatre. He ultimately became hemiplegic and died comatose, when the right lateral ventricle was found to contain a large clot, the blood having oozed from a rent in the thalamus.

It will be noticed how closely the appearances found in these five cases resembled those of spontaneous cerebral hæmorrhage. In the reports of three of them it is distinctly stated that the kidneys were granular, or wasted, or cystic. So that, even if the rupture of the artery was the direct result of a blow or fall in each instance, they may fairly be regarded as examples of ordinary apoplexy, the main cause having probably been the existence of chronic disease in the coats of the artery. Most likely, if no accident had happened, these patients would have died of spontaneous hæmorrhage into the brain before long. One cannot be surprised that it is impossible to diagnose such cases in the absence of a history during life, when the right interpretation is so difficult after an autopsy.

2. *Other structural lesions* of the brain may occasionally give rise to a rapidly fatal attack, resembling apoplexy, from hæmorrhage. This is the case, for instance, with cerebral *abscesses* and *tumours*, and occasionally with *meningitis*, whether traumatic, tuberculous, or syphilitic. Sometimes, indeed, cerebral hæmorrhage does really occur as a complication of the softer and more vascular tumours, from the blood-vessels within the growth giving way, and pouring out a large quantity of blood. It is not improbable that in many of the cases in which hæmorrhage has been found in the cerebellum, or in some other unusual seat—if the patient was young and the arteries healthy—the original disease has been a soft tumour of small size, which has been torn up by the extravasated blood and overlooked at the autopsy.

3. Narcotic *poisoning* may be mistaken for apoplexy, or *vice versâ*. We have seen that in some instances in which blood is effused into the pons the pupils are contracted and the respiration is regular and very infrequent, just as in persons who have taken large doses of opium.

I was summoned one night to see a lady some six or seven miles away from London, and found her comatose, with the symptoms just mentioned. There was a suspicion that she had poisoned herself, for the day was the anniversary of the death of a son; and a few hours before her illness began she had said to a sister, who had come to visit her, "Well, have you come to see me die?" But she had been sitting up in bed talking to this sister, and apparently as well as usual, when she suddenly fell back comatose. This fact appeared to exclude the possibility of her having taken opium; and the conclusion seemed clear that there was hæmorrhage into the pons. But the next morning she was quite well again, and never confessed that she knew the cause of her illness. Thus a patient deeply comatose from opium *may* recover without being walked about or swallowing strong coffee or stimulants.—C. H. F.

In one case of suicide from prussic acid, recorded by Dr Stevenson ('Guy's Hosp. Rep.,' 1869), death did not occur for more than an hour and a quarter after the discovery of the fact that the poison had been swallowed, and the symptoms could not have been distinguished from those of apoplexy; but all difficulty was removed by the fact that there was on the table a half-empty, although corked, bottle containing some of the poison. A marked odour of prussic acid was diffused through the room, but the patient's breath did not smell of it.

4. It is often difficult, and sometimes impossible, to distinguish apoplexy from *drunkenness*. A man who really is dead drunk may be supposed to have cerebral disease; but the only result likely to follow this mistake is that a patient who had been expected to die, or at least to become hemiplegic, may in a few hours completely recover.

In 1868 a boy, aged fourteen, was admitted into Guy's Hospital, who with another boy had stolen two bottles of brandy, and, according to his companion's statement, had drunk a reputed quart (twenty-six and two thirds fluid ounces) without any admixture with water. This occurred at about 2.15 p.m.; by four o'clock he was insensible, and he was at once taken to the hospital. He was still completely insensible, but without stertor; his pupils were at first of natural size, but afterwards became contracted. An œsophageal tube was passed, and the contents of the stomach were pumped out, with a most powerful odour of brandy. He was then put to bed, and an enema of coffee was given. He remained unconscious for twelve hours; then he asked for a glass of water, and before long was well. Dr Stevenson, commenting upon this case in the 'Guy's Hosp. Reports' for 1869, says that no doubt the boy owed his recovery to the prompt use of the stomach-pump.

It very rarely happens that anyone swallows a fatal dose of alcohol except for a wager or out of bravado, and then a physician called to the case is sure to be told what has occurred. To mistake intoxication for apoplexy is to commit an error likely to be more injurious to the doctor than to the patient. But to suppose that a man who has apoplexy is merely intoxicated is a most serious matter. This is the mistake which is often made by the police, and into which medical men themselves have too often fallen, from failing to appreciate the real difficulties of the diagnosis. Perhaps the patient is found in a public-house, or is known to have been drinking heavily, or smells strongly of spirits. The only safe course for us is to ignore these facts altogether. The odour of brandy does not even prove that liquor was taken before the attack; it may have been administered afterwards. The only safe rule, which should never be departed from, is that a man who is insensible, and who cannot be roused, is never to be sent away to the police station, nor be left by himself to sleep off the supposed drunkenness; he should always be put to bed and carefully watched. The presence of hemiplegia, convulsions, or inequality of the pupils may make one certain that the case is due to something beyond alcoholic intoxication; and the same conclusion may be drawn, although with less confidence, from the occurrence of rigidity of the limbs, or of stertor; but the *absence* of any or all these symptoms is no disproof of cerebral disease. The most important cases, however, are not those in which coma, or even partial unconsciousness, is present, but rather those in which the patient is noisy and excited, grimacing and throwing his limbs about; one is perhaps sure that such a man is drunk, but it may be impossible to say whether or not he also has hæmorrhage going on within his skull. Rupture of a cerebral artery seems often to be the direct result of indulgence in drink, and to detect its early symptoms must obviously, in such circumstances, be exceedingly difficult. Medical men who have had the largest experience are those who most freely acknowledge the impossibility of speaking confidently, particularly when the patient is advanced in years, and may have chronic disease of his heart, or of his kidneys and cerebral arteries.

5. Again there are cases of coma which resemble apoplexy, but are due to other toxic causes than narcotics or alcohol; to poisons produced in the body by disease. It is possible that the case may be one of *pyæmia*, or some specific fever. Dr Fagge recorded two cases in which profound, rapidly fatal coma was proved to be due to this auto-intoxication.

One was that of a man, aged twenty-six, who was brought to the hospital insensible and died in a quarter of an hour. Very little could be learnt about him; but it was stated that he had recently arrived on board ship in the port of London, and that he had been giving evidence in a court of law on the day of his death. The brain appeared healthy; but there was suppurative inflammation of the peritoneum, and also of one knee-joint.

The other case occurred in a girl eleven years old. She was admitted into the hospital one morning at half-past six, and immediately after being put to bed, she gave one gasp and died. Her mother said that she had been quite well until the previous day, when she complained of pain in the left side on returning home from school in the afternoon. She went to bed, and fell asleep; but at 4 a.m. (two hours and a half before her death) she was found to be unable to speak, and to be rolling over and throwing her arms and legs about. When brought to the hospital she was unconscious, and was breathing heavily with froth upon her lips; the right pupil was dilated, the left contracted. The temperature was 101·2°, the pulse 116, the respiration 32 in the minute. All that could be discovered at the autopsy was that the surface of the right lung was ecchymosed, and that there was a partial hepatisation of the lower lobe of the left lung, with much lymph effused on the pulmonary pleura over a circumscribed area.

6. Another possibility is that the coma may be due to *uræmia*. The older pathologists were familiar with cases in which, finding no blood effused upon the brain, they were driven to suppose that the cerebral

symptoms which had proved fatal were due to the presence of fluid in the ventricles and beneath the arachnoid; and they described the affection under the name of "serous apoplexy." At the present day, however, this name, and the theory on which it was based, have alike fallen into oblivion. Many years ago Wilks suggested that the majority of the cases in question were examples of Bright's disease; and his opinion has been endorsed by later writers.

Nevertheless in reading the detailed clinical reports given by Abercrombie, it is far from clear that the cause of death in his cases was uræmia, rather than some one of the less obvious cerebral lesions which at that time would have escaped recognition. The cases seem to be few in which the symptoms were like those of apoplexy, and the only disease discovered at the autopsy was in the kidneys. On the other hand, it has often happened that albumen has been detected in the urine of a patient who had been brought into the ward in a state of insensibility, and uræmia diagnosed, until a *post-mortem* examination showed that the coma was due to cerebral hæmorrhage. The common occurrence of albuminuria in such cases is partly because the kidneys are often found granular; partly because albuminuria is often met with in patients lying in an apoplectic state, when the kidneys are found after death to be perfectly healthy; so that the presence of albumen is justly regarded as the result of venous congestion.

Among the few instances in which an apoplectiform seizure has been found at Guy's Hospital to have been caused by renal disease, without any cerebral lesion being discovered, the two following were met with by Dr Fagge.

In 1867 a man, aged forty-eight, was brought to the hospital, conscious, but unable to speak; his friends said that he had often suffered from headaches; the day before his admission he complained that his mind was wrong; his manner was strange, but he answered when spoken to. Afterwards he became insensible, passing his excretions under him. At one time he regained his senses sufficiently to say "yes" and "no;" and he could swallow; but he soon relapsed into coma, and died on the following day. His pupils were rather contracted, and his breathing was laborious. The brain was found pale and anæmic; the ventricles were nearly empty. The heart was hypertrophied; the kidneys were extremely wasted, as a result of dilatation of the calyces secondary to stricture of the urethra.

In 1868 a man aged forty-four, who had been intemperate and gouty, and who had been suffering from a severe headache for a fortnight, was brought to the hospital in a drowsy state, from which he could just be roused to speak. He had had a fit, and had bitten his tongue. His body and limbs were in a state of constant jactitation. His pupils were rather contracted. His urine was retained, but his fæces were passed into the bed. Before his death he had two more fits. The only changes found in the brain were that it was tough and wasted, with large ventricles, and with an excess of fluid in them; but the kidneys weighed only two ounces and a half, and were exceedingly granular. At the bedside it would probably have been impossible to say, in reference to either of these cases, that an artery had not given way within the brain.

It has sometimes happened that patients under treatment in the hospital for Bright's disease have died quickly with cerebral symptoms; but under such circumstances hæmorrhage is comparatively infrequent.

In the great majority of cases the stupor caused by uræmia alternates with convulsions, and passes off again and again without leaving hemiplegia—so as to disprove cerebral hæmorrhage.

7. Coma from *diabetes* or from *cholæmia* may simulate apoplexy, but the presence of glycosuria or of jaundice will decide the nature of such cases. In neither case is there hemiplegia, and the symptoms come on more gradually than those of apoplexy.

8. Of cerebral diseases, the one which is most likely to be mistaken for

apoplexy is a form of *epilepsy*, which was described by Andral and other French writers as a separate malady, under the cumbrous name of "apoplectiform cerebral congestion," until Trousseau pointed out its real nature and distinguishing characters. In all probability Abercrombie would have included it under what he termed "simple apoplexy," in which after death no morbid appearance could be discovered in the brain; and Sir William Gull used to teach in his lectures that this "simple apoplexy" was nothing but epilepsy.

Among the cases related by Trousseau are the following:

In 1845 a gentleman, aged forty-two, was found in his bed insensible; his face was turgid and livid, there was stertor, and all power of motion and sensation was lost. How long he had been in this condition his wife could not tell—she had been awakened by a strange snoring noise. Trousseau had the patient placed in a half-sitting posture, threw cold water in his face, and applied ligatures round the upper part of the thighs to retain the blood in the legs. Scarcely one hour elapsed before he regained his senses and the use of his limbs, and on the following day great lassitude was the only remaining symptom.

Some time afterwards the same physician was fetched in great haste to a neighbour, aged seventy, who was said to have been attacked with apoplexy on the Boulevards. He had been unconscious for a quarter of an hour, but was recovering his senses when Trousseau arrived. He did not at first recognise him, and looked vacantly round, throwing his arms and legs about without knowing what he was doing. Within a few hours he gradually and completely recovered.

A solicitor, aged thirty-five, was sent to Trousseau from the country, with the history that in the course of the previous six months he had had three apoplectic fits. They had lasted, however, an hour at the most, and they had left no paralysis behind them. Trousseau accordingly declared the disease to be epilepsy, and his diagnosis was before long found to be correct. But of course there must in every instance be a first attack, and then the criterion fails, and if this seizure should prove fatal the real nature of the disease must remain a matter of inference.

In both these cases the real nature of the disease was afterwards established by the recurrence of the seizures at more or less frequent intervals, attended with all the symptoms of epilepsy. Indeed, it must be observed that in each case the attack began when no skilled observer was present, so that there was no proof of the absence of convulsions; and Trousseau himself goes on to say that in almost every instance of the same kind in which he was consulted, and in which the commencement of the seizures had been seen, "nervous twitches" or convulsions had been present. Thus, after all, the identification of "apoplectiform cerebral congestion" with epilepsy involves little more than the recognition of two facts,—that the spasms which usher in an epileptic seizure may be but slightly marked, so as to escape the notice of an ordinary observer; and that when one is called to a patient who is comatose one must make sure that the disease is *not* epilepsy before committing one's self to a diagnosis of apoplexy.

When we feel reasonable doubt between the diagnosis of apoplexy and that of epilepsy, our prognosis must be most cautiously guarded: for in a few hours the patient may be apparently as well as ever, or he may be dying.

Dr Fagge was one evening called out in great haste to see an old man, and found him lying on the sofa in his sitting-room, comatose and stertorous, with puffing cheeks and a purple countenance. His pupils were dilated, but the general aspect was that of apoplexy. His housekeeper, who had found him insensible, knew that he had once or twice before had epileptic attacks. Before long he completely recovered.

In 1865 a man aged sixty-seven, a tanner in Bermondsey, was brought into the hospital comatose, and died in a few hours. He had followed his occupation until the day of his death, when he was found insensible, having fallen to the ground. He had convulsive movements, and appeared to be paralysed on the right side. It could not be ascertained that he had ever before had a similar attack, but he was said to have suffered much [illegible] his head. No recent morbid change could be found in the brain, but there was chro[illegible] wasting, the convolutions being shrunken, with much fluid in the sulci, and the ventricles being enlarged and their surface granular; the skull also was dense and had no diploë, and the membranes were thickened and opaque.

A very similar case occurred in 1876. A woman, aged sixty-two, was brought into the Clinical Ward, having fallen down in the street in a fit. The right pupil was larger than the left. There was right hemiplegia, with complete anæsthesia in the right arm and in the right side of the face. After a time she partially regained her consciousness, but had several convulsive attacks. The coma then again became deeper, and she died at the end of five days. No morbid change could be found in the brain except wasting with dilatation of the ventricles; and there was only slight wasting of the kidneys.

In 1896 a powerfully built carman, of about forty-five, was brought into the writer's ward, who had been found by a policeman sitting on his box and driving his horses in a state of complete insensibility—speechless and senseless. No convulsions were seen, and these could scarcely have escaped notice in the street. He was admitted as a case of apoplexy, and when seen shortly afterwards was still in complete coma with stertorous breathing and insensible pupils. The urine when drawn off was found to contain albumen. But there was no difference between the limbs on the two sides; the pulse was not small or hard, and although there was no history of previous fits the case was regarded as one of eclampsia, and after about two hours the patient recovered consciousness, and went out next day perfectly well, with healthy urine, and with no sign of apoplexy or uræmia. He had not been drinking, but he had before had less severe attacks of what was no doubt epilepsy.

Diagnosis between the anatomical causes of apoplexy.—We now come to the differential diagnosis of the several lesions, which are each capable of causing apoplexy, as above defined, aphasia, or hemiplegia; namely, hæmorrhage, embolism, and thrombosis with softening from syphilitic or other disease of the arteries.

(*a*) In cases of *apoplexy*, we must be guided to a diagnosis of its exact cause by such instances as the following.

Cerebral embolism found after death.—In 1868 a man, aged forty, was brought into the Clinical Ward with paralysis of the left side, having suddenly fallen down insensible while wheeling a barrow. He presently regained his senses and conversed about his symptoms; then he again became comatose, and remained so until he died a few hours afterwards.

In 1869 a man was taken in for paralysis and partial anæsthesia of the left side. He was reported to have suffered from continuous headache for two or three weeks. A few days before his admission he had suddenly become giddy and lost the use of his left arm and leg; afterwards he was unconscious for two or three hours, and then died.

In the same year a woman, who was in the hospital suffering from dropsy, had a fit, which was followed by aphasia and right hemiplegia, and she died within two days.

In 1874 a woman, who was in the ward for uterine cases under Dr Braxton Hicks, became comatose with left hemiplegia, and remained insensible until she died on the following day.

In none of these four instances was there any hæmorrhage into the brain, nor any change beyond softening of the part which should have received its supply of blood through the obstructed vessel. Yet, if no autopsies had been made, these very cases might well have been quoted as illustrating the chief varieties in the mode of onset of an apoplectic seizure due to the rupture of a cerebral artery. Indeed, there appears to be no absolute difference between the symptoms of embolism and those of hæmorrhage; and there can be no question as to the inadequacy of certain supposed points of distinction—that in embolism the face is pale rather than red; and that cases of hæmorrhage are accompanied by excessive pulsation of the carotids, stertorous breathing, and inequality of the pupils.

Nothnagel quotes Dr Eliza Walker as having shown in her inaugural dissertation at Zürich, in 1872, that embolism is often ushered in by an epileptiform attack; this was the case in twenty-four out of ninety-seven cases which were collected by her. In seventy-nine cases of embolism Dr Pitt found seven in which convulsions occurred.

Thrombosis found after death.—A man, aged forty-eight, was attacked with pain in the head a month before his death. He fell back insensible, with right hemiplegia. Afterwards he could walk, but he ultimately became again semi-conscious and rambling in his talk.

Another man, aged thirty-five, was attacked with unconsciousness and paralysis of the left side six days before his death. He then had convulsive fits, in one of which he died.

Apoplexy due to arterial thrombosis can seldom be distinguished from that caused by cerebral hæmorrhage. The writer had a case of repeated attacks in an elderly woman in 1890, and concluded in favour of thrombosis rather than hæmorrhage, but after death rupture of arteries in several parts of the brain was discovered, with no obstruction of the vessels.

The most frequent cause of cerebral thrombosis is *syphilitic* disease of the arteries; and the cases related by Heubner in his monograph show how closely the symptoms of this lesion may resemble those of rupture of an artery in the brain. In eight cases out of twenty-two collected by him the symptoms were ushered in by an attack of apoplexy. In some rapidly fatal cases the insensibility lasted until death. In others there was a transitory loss of consciousness, accompanied by hemiplegia, and followed after an interval by the supervention of coma from which the patient never again woke; in other words, the clinical features of an "ingravescent" nature were closely imitated.

A man who had suffered from headache and sleeplessness, but appeared to be as well as usual in the morning, was found comatose and paralysed on the right side; he could at first be roused a little, so as to make an attempt to open his eyes; but afterwards the insensibility became more profound, the face flushed, the pupils immoveable, and he died the next evening.

A journeyman furrier, admitted into the Leipzig Hospital in a state of unconsciousness, had been attacked on the previous day with paralysis of the arm; this came on gradually, so that he watched its progress upwards from the thumb to the shoulder, being then in perfect possession of his senses, and able to point with his left hand how high the numbness extended at any particular moment.

In both cases thrombus was found in an artery affected with syphilitic endarteritis.

Thus we find that an attack of apoplexy is by no means always due to extravasation of blood in the brain. Omitting spontaneous thrombosis on account of its rarity, we have embolism and syphilitic arteritis as possible causes of precisely similar symptoms.

The diagnosis is based chiefly upon collateral circumstances. *Age* is an element of the highest importance. In persons more than fifty years old one is seldom wrong in attributing an apoplectic seizure to hæmorrhage: and the younger the patient the greater the probability that one of the other two causes is in operation. To decide between them we must look for indications of cardiac disease on the one hand, and, on the other, for eruptions on the skin or fauces, enlargements of the testicles or lymphatic glands and nodes. It must not be forgotten that cerebral hæmorrhage may be an indirect result of embolism, by the formation of an aneurysm in the obstructed artery (p. 723). In most cases of this kind the primary disease is ulcerative endocarditis, and the embola are septic; so that enlarged and tender spleen, or albumen or blood in the urine, may suggest the correct diagnosis.

Notwithstanding the caution expressed above (p. 753), the *presence of albumen* when combined with a low specific gravity of the urine, a hard pulse, and signs of a hypertrophied left ventricle are of great diagnostic value, for these symptoms of chronic interstitial nephritis much increase the probability of a ruptured artery. If we find evidence of retinal hæmorrhage or albuminuric retinitis by the ophthalmoscope, we feel almost certain of our diagnosis. A very gradual *onset* of coma, with severe previous symptoms of pain, sickness, or vertigo, for a day or two, points to occlusion of an artery (probably syphilitic in a young adult), and consequent softening. A more sudden and profound attack is probably due to hæmorrhage. A shock, like syncope, with revival and then torpor, gradually deepening into coma coming on within an hour, points almost certainly to hæmorrhage, checked by failure of the heart, and returning as the circulation improves.

Pain in the head, with hemiplegia and subsequent coma, suggests cerebral embolism, and in a young subject with a cardiac murmur makes it practically certain.

(*b*) In cases of *hemiplegia* the diagnosis between its several causes depends first upon the question whether coma was present when the attack of paralysis occurred. If so, the considerations stated in the last paragraph are applicable; and in proportion to the duration and the severity of apoplectic symptoms is the probability that there was rupture of an artery. On the other hand, if the seizure was unattended with any, even transient, loss of intelligence, the presumption is strongly in favour of its being the result of a mere arrest of circulation in some part of the motor tract. This may depend on embolism of a cerebral artery, or on syphilitic endarteritis, or on arteritis deformans, apart from syphilis, which also leads to the formation of patches of white softening from thrombosis. One would have expected that a point of considerable diagnostic importance would have been the insidious and gradual commencement of hemiplegia due to arteritis. But experience scarcely bears this out; the attack is generally more or less sudden, as we shall find it may be even when due to such chronic lesions as a cerebral tumour. It is probable that the nervous elements continue to perform their functions up to a certain point, notwithstanding impairment of their structure; and that several of them at length suddenly and simultaneously give way, just as a rope will sustain a weight until the moment before the last strand breaks.

E. [illegible]

The discrimination of the various lesions of the brain that may give rise to hemiplegia depends on a careful study of the other nervous symptoms. We have already seen the diagnostic value of the association of *aphasia* with hemiplegia, as showing that the Sylvian artery is obstructed, whether by embolism, thrombosis, or syphilitic thickening.

It is probable that persistent *hemianæsthesia*, when associated with hemiplegia, points to hæmorrhage rather than to arterial obstruction. For such a combination of symptoms shows that the posterior third of the internal capsule is involved; and this lies towards the periphery of the area of distribution of the Sylvian artery (p. 721), so that it might probably be fed from another source if arrest of the blood-supply were the cause of the hemiplegia. However this may be, most of the cases hitherto recorded, in which loss of sensation has been permanent, have occurred in old people, so that rupture of an artery was probably the cause of the symptoms. In the case referred to above (p. 731) the patient was seventy-one at the time of the first seizure.

On the other hand, *syphilis* is apt to affect several of the large cerebral arteries, simultaneously or in succession. It is therefore not surprising that hemiplegia dependent upon this cause is sometimes accompanied by a series of *incongruous* or *irregular* symptoms, which cannot be referred to a lesion limited to any one spot in the brain. Thus there is sometimes complete paralysis of one or more of the cranial nerves, or the patient may regain the use of the arm and leg which were first affected; and subsequently he may be attacked with loss of power on the opposite side. This, indeed, is not in itself characteristic of the syphilitic affection; for old people with atheromatous vessels are very liable to have patches of softening develop themselves in the two hemispheres in succession, or to suffer from the effects of small hæmorrhages into both corpora striata in turn. But symptoms due to syphilis are more apt to be irregular in their locality and course than those which depend upon other causes; the patient perhaps becomes able to stand, or even to walk, and then after a few days relapses; and such changes may occur again and again.

Heubner laid stress on a peculiar *somnolent* condition, as indicating the presence of syphilis: the patient, he said, is half awake, half asleep; he lies with his eyes shut, taking no notice of anything, and refusing to answer questions; but when one tries to examine him, he resists. That such symptoms are comparatively rare in cases of embolism or of cerebral hæmorrhage is true; but they are common enough when the disease is tumour, and one must always remember that tumours may give rise to hemiplegia if they involve the motor tract.

There is, in fact, no one of all the organic changes to which the brain is liable that may not sometimes induce loss of power in the arm and leg on one side; beside epilepsy, hysteria, chorea, and some other neuroses which will be described in their place.

Conversely, it occasionally happens that affections of the cerebral arteries may run their course to a fatal issue without hemiplegia appearing. Such cases may be attended with headache, loss of memory, drowsiness, delirium, vomiting, thickness of speech, dysphagia, involuntary evacuations, and other symptoms, but none of them are characteristic.

Prognosis.—The gravity of a case of Apoplexy may be judged of first by the depth of the coma. The ingravescent cases are the worst, while gradual recovery of consciousness is of good omen. In cases comatose from the first, death frequently follows on the third day, but it may occur after a week or even longer; and, on the other hand, recovery may follow prolonged unconsciousness if the pulse and breathing are not seriously affected, and if the patient is not fed. Rise of temperature is a bad sign. So are early rigidity, convulsions of the paralysed limbs, flapping of the cheek in breathing, insensitive conjunctivæ, and increasing cyanosis. The longer coma persists, the less hope there is; but the writer once saw a patient make a good recovery after lying ten days insensible. When consciousness is once restored, life is generally preserved, at least until a subsequent attack.

Treatment of Apoplexy.—As Hippocrates said, "to cure apoplexy when severe is impossible, and not easy when it is slight" ('Aphor.,' ii. 41). Moreover, then, there is no disease in which it is more difficult to estimate the effect of any therapeutical measures adopted.

The patient should, if possible, be left in the room in which the seizure occurred; a mattress placed on the floor does perfectly well for a time. His head and shoulders must be raised, and he should be turned well over on his side, so as to prevent the tongue from falling backwards against the pharynx. If death is impending one ought to abstain from all active treatment, for no good can be done by the application of a blister to the neck, or of mustard plasters to the calves. A patient whose coma is so deep as to threaten his life cannot be roused by such means; if he is still sensible, the irritation must be injurious rather than beneficial.

When the case appears not to be altogether hopeless, five or ten grains of calomel or two drops of croton oil should be placed on the tongue, particularly if there is reason to believe that there is an accumulation in the bowels; or an enema of turpentine may be given. If the insensibility last more than a few hours it may be necessary to pass the catheter. Whenever there is serious difficulty of swallowing, the administration of food or drink by the mouth should be altogether forbidden, on account of the danger of its running into the air-passages and setting up pneumonia. No harm results from keeping an apoplectic patient for a day or two without nourishment; and afterwards if needful he may be fed by the rectum.

Bloodletting, whether by venesection, cupping, or leeches, is now seldom practised. Sir Thomas Watson, indeed, speaks of patients so insensible as not to feel the puncture made by the lancet, who have yet emerged from their coma while the blood was flowing. But it may be fairly doubted whether in such cases the disease was not that "simple apoplexy" which is probably a form of epilepsy or else uræmic coma. If the pulse be large and labouring, the face flushed, the carotid arteries full and throbbing, bleeding seems to be indicated. But most patients suffering from apoplexy are advanced in years and impaired in health; while in a younger person the coma may be due to embolism or to syphilitic arterial disease, and not to arterial hæmorrhage.

When the symptoms of cerebral hæmorrhage are slowly ingravescent, it seems probable that treatment may sometimes prevent a fatal issue. If called to such a patient in the early stage (that of collapse), one should keep him recumbent, but with head and shoulders raised, and he should not be allowed to move. The limbs may be warmed by hot flannels or elastic hot-water bottles, but the administration of brandy, and even of ammonia, must be forbidden. As reaction comes on, the question of bloodletting must be seriously weighed. It is true that a rapid death almost always occurs, and that the autopsy generally confirms the opinion that the case was hopeless from the first. But it is possible that free venesection, just at the time when the vigour of the circulation is being re-established, may, by lowering the pressure in the cerebral vessels, prevent further effusion of blood.

The writer has once only had the opportunity of ordering venesection at this crisis. The patient came in already recovering from apoplexy. A second attack occurred a few days later, and a clinical assistant on the watch for it opened a vein within three minutes of its occurrence, and drew off about 12 oz. of blood; but this did not avert the fatal issue.

Hæmostatics, such as ergot, acetate of lead, or gallic acid, are no longer prescribed; but cooling lotions, or a bag of ice, may be applied to the head, if they do not disturb the patient.

When coma is probably due to Embolism, the only treatment is to keep the patient in a state of the most perfect quietude, so as to reduce to a minimum the activity of the nervous centres deprived of their blood-supply.

In cases believed to be due to obstruction from Syphilitic arteritis, inunction of mercury should be commenced with the first cerebral symptoms.

A person attacked with Hemiplegia without unconsciousness should for some days be kept in bed, and as free as possible from all mental or bodily disturbance. If advanced in years, or suffering from heart disease, he should be supplied with soup, beef-tea, milk, and perhaps a little wine.

A syphilitic patient should at first have rather a scanty diet, while the proper treatment is being carried out: say a twelfth of a grain of perchloride of mercury, ten or fifteen grains of iodide of potassium taken three or four times daily.

Heubner relates some remarkable examples of recovery of power in the paralysed limbs under the method of inunction.

A striking case was that of a student, aged twenty-six, who after having suffered from headache and giddiness for some weeks was attacked one night with left hemiplegia during sleep. There were indurated glands in the neck and at each elbow, and a scar on the velum palati. At the end of a fortnight he was no better—feverish, prostrate, sleepless, and delirious; bedsores were forming, and spasmodic movements of the right arm had begun. Mercurial ointment was then rubbed in for seven days, and afterwards iodide of potassium was given in large doses. Six weeks later he was able to leave his bed, and little by little he regained power in his limbs. Several years afterwards Heubner found him perfectly well, except that he walked with a stick.

In most cases of Hemiplegia after apoplexy the limbs regain power to a certain extent, in a few they do so completely, but in many more or less paralysis remains, in spite of the most judicious treatment. In chronic cases recourse may then be had to electricity, and sometimes with benefit. The continuous application of a very weak galvanic current to the head, as advised by Remak, is said by Nothnagel to be occasionally followed by a decided increase of power and diminution of rigidity in the affected muscles.

A more intelligible method is to excite the paralysed muscles to contract by the make and break of slowly interrupted galvanism or by the faradic current. Whichever acts best is to be preferred. As the late Sir Russell Reynolds said, little or no good can be thus effected if the muscles contract normally; but if the contractility is diminished, electricity will often in the course of a few weeks restore it; muscles will become less wasted, and the previously cold limb will regain its temperature. By faradising the extensor muscles of the fingers one may diminish contraction of the fingers into the palm. In general it may be stated that the application of electricity should not begin till two months from the hemiplegic seizure; that it should be at once left off if it causes headache, giddiness, faintness, sickness, or any unpleasant feeling at the epigastrium; and that the current should never be so strong as to cause pain, nor so long continued as to cause fatigue.

Hemiplegia cannot be influenced by drugs, though occasionally laxatives and sometimes hypnotics are required. Nor can one reasonably expect benefit to result from visits to Bath or Harrogate, Gastein, Pfeffers, or anywhere else.

To prevent a recurrence of the attack, temperance both in eating and drinking, a loose state of the bowels, and the avoidance of emotional and other excitement, both of mind and body, are the rational and probably the only measures to be advised.

SOFTENING OF THE BRAIN.—A circumscribed spot of the brain or a less defined area is not infrequently found to be much softer than usual, independent of *post-mortem* maceration, of decomposition (which occurs earlier in the brain and cord than elsewhere), and of accidental injury in opening the skull. The consistency of the cerebral tissue is so reduced from its

ral firmness, that it sometimes becomes diffluent, and is washed away
a moderate stream of water, leaving a shreddy surface. The specific
rity also is lowered, as shown by Dr Bastian in 1866, from about 1040
032.

We have to consider the nature of the process which leads to cerebral
ening, whether the colour be red, brown, rusty, yellow, or white.

The morbid condition named red, brown, or rusty, is not very common,
its nature has been much disputed. It is attended with œdema; the
cted convolutions are broader than natural, and the sulci between them
per; and the corpus striatum or thalamus looks rounded, prominent, and
. There is a marked diminution of the natural firm consistence, and the
ur is altered; the cortex assumes a deep purple tint, and the white
stance becomes pink or red, with numerous minute ecchymoses. Under
microscope the venules and capillaries are found dilated and full of
d; blood-corpuscles are seen among the nervous tissues, and unless
ntly extravasated, are found united in amorphous masses. Notwith-
ding Rindfleisch's statement, pus cells are very rarely if ever present.
on says that he has often been surprised to find how slight were
histological changes in some cases of this kind: the elements of the
ue were softened, but they still retained their form, and no charac-
istic inflammatory products could be recognised. Gluge's large "com-
nd corpuscles" are, however, usually present.

It has been assumed by some pathologists that suppuration of the brain
further stage of red softening. It is true that in cases of pyæmia one
etimes finds reddened patches apparently antecedent to the formation
he pus. But when several suppurating cavities are found in the same
isphere—however small and recent they may be—the intervening
bral substance is pale and firm. Moreover the causes of red softening
different from those of abscess.

One of these causes is injury. Some years ago a woman died in Guy's
pital who had fallen three or four months previously and struck her
d against the wall. Three weeks before her death she had a fit, which
followed by partial left hemiplegia, affecting the side of the face and
gue, and accompanied with ptosis. At the autopsy, all the parts at the
e of the brain were found by Wilks to be in a state of softening,
rtly red and partly white;" one third of the thickness of the pons was
affected, and nearly the whole of the crus cerebri.

Cerebral softening is occasionally found in the neighbourhood of an apo-
ctic clot, or round a tumour, but more frequently in a part of the brain
rived of its blood-supply by thrombosis or embolism. It appears never
occur as a primary and independent process.

In two autopsies the occipital lobe of the left hemisphere of the brain, and a consider-
part of the cerebellum on the same side, presented the appearance of red softening;
in each of them the cardiac valves showed recent vegetations, so that it seems probable
the affection was dependent upon embolism of some of the smaller arteries.

I once made an autopsy in which parts of the right superior and middle frontal
volutions were swollen, soft, and of a pinkish-grey colour. There was no caseation, and
lefinite edge to suggest the presence of a new growth; so that the lesion would have
regarded as primary acute inflammation had not the microscope revealed the presence
large number of oval and round cells infiltrated between the nerve-fibres. It was a
sed glioma.

Some years ago, a woman, aged twenty-six, died in the hospital, after an illness of three
ths' duration. The falx cerebri was found adherent to the anterior parts of both
ispheres by granulation tissue. On section the frontal lobes appeared of a brick-red
ur; *their cineritious substance was* swollen, and the boundary line between it and the

white matter was ill defined. On the left side this red colour extended down to the lateral ventricle. The affected parts were rather harder than natural; but in all other respects the disease corresponded perfectly with the descriptions which writers have given of local inflammation of the brain; and unless there was a new growth which was overlooked, it must have been of that nature. The microscope only showed corpuscular infiltration, with Gluge's compound granule-masses.—C. H. F.

Roston, who in 1820 gave the classical description of *ramollissement rouge*, regarded it, with Lallemand and Cruveilhier, as sometimes inflammatory and sometimes the result of obstruction of an artery, a view advocated by Abercrombie and Rokitansky. Virchow added obstruction by an embolus to that by thrombosis as a cause of red softening. Red softening was held to be inflammatory, and white softening to be necrotic ("gangrenous," as Abercrombie called it), from obstruction of the blood-vessels. But there is no such thing as acute inflammation of the brain, except abscess, which is always septic, and never due to mere obstruction of vessels. "Softening," whether red, white, or yellow, is a passive secondary process due to embolism, thrombosis, or arterial degeneration. The red colour of *ramollissement rouge* is due to the extravasation of blood—capillary ecchymosis—complicating the necrosis, just as it does in cases of embolism of the spleen, kidney, and retina. The reason why white softening (*ramollissement blanc* of French authors) is more common in old people, and red in young, is that in the latter it only occurs as the result of embolism from rheumatic endocarditis; whereas in adult life the larger number of cases are due to local atheroma or syphilitic endarteritis with thrombosis, and there is more venous reflex from the sudden and complete obstruction of an embolus than from the gradual obstruction produced by narrowing of the calibre of an artery.

The yellow softening described by Rokitansky and subsequent pathologists is, in most cases, nothing but a late stage of red softening, which passes through the gradations of rusty, orange, and yellow, as the hæmoglobin of the extravasated blood-discs is gradually converted into hæmatoidin. In exceptional cases, particularly when it occurs round a tumour, the yellow tint may be due to a true fatty degeneration, complicating "white" softening from thrombosis.*

The clinical recognition, the distribution as to age, and the prognosis of red and white softening chiefly belong to embolism and thrombosis of the cerebral arteries, which have been treated in a preceding section (pp. 718—720).

The phrase "softening of the brain" has, however, been applied in popular rather than medical language to a condition of mental imbecility which sometimes comes on in advanced age as a part of senile decay, but which is also observed as a sequel of apoplexy in middle or later life, and of arterial occlusion from syphilis or rheumatic endocarditis in the earlier adult period.

Probably in most of these cases the popular term is not far wrong. Senile dementia depends primarily on chronic changes in the nervous

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* Huguenin describes various retrograde changes as consecutive to red softening. He says that the patches may either subside entirely, or pass into a condition of yellow softening with cavities full of serum, or undergo cicatrisation, or become converted into tough, dirty white indurated masses. It is difficult to tell on what evidence we could conclude that the conditions in question had followed red softening rather than an effusion of blood or obstruction of an artery.—C. H. F.

; but secondarily, on degeneration of the cerebral arteries. To st condition are also due cerebral hæmorrhage and its results; syphilitic endarteritis and embolism from acute endocarditis (or nitral stenosis) account between them for the cases in younger s.

TUMOURS AND ABSCESS OF THE BRAIN

CEREBRAL TUMOURS—*Tubercle—Syphilitic gumma—Meningeal growths—Glioma and other neoplasms—Cysts—hydatids—General symptoms: headache, vertigo, and other cephalic symptoms—vomiting—temperature, pulse, and respiration—optic neuritis—Localising symptoms—for the base—the cerebellum—the motor area of the cortex—the corpus striatum and thalamus—the corpus callosum and other parts—Diagnosis from hysteria, meningitis, etc.—diagnosis between the several kinds of tumour—Prognosis—Treatment.*

CEREBRAL ABSCESS—*Ætiology—Anatomy—Symptoms: general and localising—Diagnosis—Course and event—Treatment.*

THE important group of organic affections of the brain formed by new growths are of great interest from the exact localisation of disease which recent physiological discoveries have rendered possible. The group is not a pathological one; for it includes syphilitic gummata, tubercle, and new growths, and we shall add an account of local suppuration or cerebral abscess. The lesions discussed in this chapter only agree in causing increasing local pressure within the skull; but this is clinically all-important, for they produce similar effects. We will begin with the anatomy of cerebral *tumours*, including *Tubercle* and *Syphilitic Gumma*. Afterwards we will discuss their common symptoms, their localisation, differential diagnosis, and their treatment. Lastly, *Abscess* of the Brain will be separately described.

1. *Tuberculous tumour of the brain.*—The specific bacillus of tubercle may affect the brain in two different ways. Sometimes minute tubercles grow into it from the pia mater, or are scattered through its substance along the vessels. This condition is always associated with meningitis, and will be described in the next chapter. In other cases there is a single caseating mass, which varies in size from that of a pea to that of a walnut; one of the largest is a specimen in the museum of Guy's Hospital, which was received from Dr Hughlings Jackson. Sometimes these tuberculous masses are flattened towards the pia mater; but commonly they are more or less globular, and either touch the surface only at one spot or are completely surrounded by brain-substance. They do not adhere to the dura mater lining the skull, but may be fixed to the tentorium. Their substance is of a yellow colour, usually firm or even hard, but sometimes soft or even diffluent, opaque and generally homogeneous, but the centre may be softening down into a yellowish liquid, or may be partially calcified. They have, in most cases, a narrow, pinkish-grey, soft growing edge, wh separates the yellow caseous material from the brain-tissue around. T edge sometimes consists of an aggregation of miliary tubercles, and mic scopically it contains lymph-like corpuscles like those of tubercles in ot

parts, with delicate connective tissue and specific bacilli. As Rindfleisch observed, the marginal zone of the mass sometimes consists of well-formed fibrous tissue; the same transformation of the tuberculous corpuscles as occurs frequently in the lungs.

Among thirty-two cases that Dr Fagge observed at Guy's Hospital there were only two in which no tuberculous lesion could be discovered in any other part of the body. Tuberculous meningitis (which was common) is rather an accidental result of infection from the caseous mass than independent; but in a large number of instances there was chronic phthisis or disease of the mesenteric or mediastinal glands. In eighteen of the thirty-two cases the cerebellum was the seat of the tumour; three times the pons Varolii, once the bulb, and six times one of the hemispheres: in the other four cases there were several tubercles in different parts of the brain.

Twenty-one of the patients were males and eleven females, a proportion which accords with that given by other writers. In three instances the age was under five years, in six between six and ten years, in twelve between eleven and twenty years, in seven between twenty-one and thirty, in three between thirty-one and forty, and in one forty-two.

In one case, that of a child aged four and a half, there is said to have been a fall upon the back of the head five or six weeks before the occurrence of a fit, which was the earliest sign of cerebral mischief; the seat of the tumour was the pons. Crichton Browne mentions ('West Riding Asylum Rep.,' ii) a similar instance; the same part of the head was struck, symptoms speedily showed themselves, and proved fatal in two months, and a mass of tubercle the size of a walnut was found in the cerebellum. Many other such cases are on record; but, remembering the frequency of falls on the head among children, perhaps not more than mere coincidence would account for. Moreover in some cases of cerebellar tumour, the fall which preceded other symptoms may have been due to vertigo.

2. *Syphilitic gumma of the brain.*—This usually appears as a firm, dry, yellow, caseous lump, which is not always easily distinguished from tubercle. One point of difference is the fact, long ago observed by Wilks, that it almost always grows into the cerebral substance from the meninges. Heubner found among forty-five cases, recorded by different observers, only three exceptions to this rule, and these were not conclusive. Again, when a gumma occupies the convexity of the brain, the corresponding part of the dura mater becomes thickened, adherent, and forms a tough yellow layer inseparable from the tumour; so that the shape of a gumma is more irregular than that of a tubercle, which is more or less globular. Moreover it shows a larger proportion of translucent undegenerated tissue, as a moist greyish-red or grey mass, sometimes as soft as jelly.

The most common seat of a gumma is the base, where it may involve several of the cranial nerves and fill up the sella turcica as well as the diamond-shaped space. Microscopically it consists of granulation tissue, containing some fusiform and stellate corpuscles. It is very vascular, and sometimes presents many ecchymoses. When antisyphilitic remedies have been given during life, the only morbid change found after death may be a patch of superficial softening of the cortex, beneath a local adhesion of the dura mater to the brain; this was the case, for example, in a patient of Dr Dreschfeld ('Lancet,' 1877).

Of ten cases of gumma of the brain at Guy's Hospital, all occurred between the ages of twenty-six and forty. In five of them there was a definite history of constitutional syphilis, or else indisputable syphilitic lesions were found; only one patient distinctly stated during life that he had never had venereal disease, and in that case the liver contained gummata. In one case the patient's illness had been attributed during life to a sunstroke; and in practice sunstroke often turns out to be cerebral syphilis.

Other tumours of the brain.—There is no region of the body in which so many different kinds of new growth are found as in the brain and in its membranes; yet histological distinctions are nowhere else of so little practical importance, because the clinical history and symptoms of a case are not affected by the nature of the tumour.

The *membranes* may present fibrous and some other simple tumours, such as occur almost anywhere, and malignant tumours of all sorts, usually secondary. A favourite seat for malignant growths, usually *sarcomata*, is between the dura mater and the bone; or perhaps it would be more correct to say that they begin within the osseous substance itself, and either push inwards the dura mater or penetrate it, protruding upon its inner surface, and invading the cerebral tissue.

Two or three kinds of innocent tumour are peculiar to the meninges. To one of them Virchow gave the name *psammoma*, from its containing calcified particles, like those which constitute the brain-sand of the pineal body and choroid plexuses. The lime-salts are deposited in curious globular structures, made up of elements arranged concentrically, so as to resemble somewhat the well-known "bird's-nest cells." Robin accordingly described this affection as a form of epithelioma; but such a view of its nature is incorrect. Virchow says it generally appears as a semi-globular mass, of firm consistence, pale red or white, and medulla-like. One examined by Dr Fagge had a loose texture and a flocculent surface; it grew in such a position as to indent the brain above the left Sylvian fissure. According to Virchow such growths are less frequently seated in the tentorium or in the falx than in the dura mater lining the skull—particularly in the anterior fossa.

Another form of tumour peculiar to the membranes is known as *cholesteatoma*, or "pearl-tumour." It forms a dry, hard, rounded mass, occupying the pia mater generally at the base of the brain. Its cut surface has a pearly lustre, and it consists of lobules made up entirely of horny epithelial cells, and supported by a stroma of dense connective tissue.

Lastly, Virchow has described under the name *melanoma* a fourth kind of meningeal new growth; in a case which came under his observation there were numerous black or brown nodules in the pia mater, the rest of the body being entirely free from them.

Of cerebral tumours, properly so called, all but one are found in other parts of the body. Of these, *sarcomata* are the most numerous, especially those made up of spindle-cells. Sometimes, though very rarely, a myxoma or fibroma is met with. Primary carcinoma of the brain is so rare that its occurrence is doubtful, and even secondary cancer is far from common: when secondary nodules develop within the cranium, they are as a rule sarcomata, and they are almost always multiple, whereas a primary new growth is solitary. Among forty-four cases reported by Dr Fagge, in only two was there more than one tumour in the brain, without obvious source of infection elsewhere; and it is quite possible that in those cases there was primary disease of one of the bones, or of some other structure, which was overlooked.

Cysts.—The centre of a cerebral tumour now and then softens down into a *cyst*, and this may become so large that the presence of solid tissue may be overlooked. In five cases at Guy's Hospital the cerebellum contained a large thin-walled cavity, filled with a fluid that was highly albuminous or deposited a fibrinous coagulum. In three of these no structure could be discovered, except the vascular membrane forming the cyst itself, but in each of the other two was a small tumour situated on the fibrous wall.

Other cysts, in which no trace of a tumour can be found, are least rare in the cerebellum, and are sometimes multiple in the cerebrum and associated with cysts in the liver, kidney, or pancreas. Their origin is still a matter of dispute, and perhaps is not identical in the several organs which seem to be similarly affected ('Path. Trans.,' 1885, vol. xxxvi, p. 17).

Hydatids are of very rare occurrence in the brain. In a girl, aged nine, Dr Moxon found a large *echinococcus* occupying the middle and posterior lobes of the right hemisphere; and there was another and no doubt a primary one of the liver. The *Cysticercus cellulosæ* occasionally infests the pia mater or the ventricles.

Glioma.—The one kind of tumour peculiar to the brain, the cord, and the retina,* was named by Virchow *glioma*, because he regarded it as formed from the neuroglia, or interstitial connective tissue of the brain, the existence and structure of which he first demonstrated. He admitted that it was often impossible to say of a particular specimen whether it should be called a glioma or a sarcoma; and recognised intermediate forms, which he termed glio-sarcomata. This origin from the neuroglia, or perhaps in some cases from the endothelium of the perivascular lymph-channels, is recognised, but the exciting cause is quite unknown. Multiple glioma is not uncommon, and sometimes affects the cord as well as the brain. As we have seen in the former situation, cystic degeneration often takes place, producing syringomyelia (p. 631), and such a condition has been conveniently termed a *gliosis*. A myxoma is occasionally found in the cerebrum, or a myxo-sarcoma.

A glioma may be of a pinkish-red colour, or it may look so exactly like the surrounding brain-substance that it can only be recognised by the microscope. Its substance is continuous; indeed, it often assumes the form of the part in which it grows, so that one might imagine the corpus striatum or the thalamus, or some particular convolution, to have swollen to three or four times its natural size. In some cases caseation takes place in these growths; and, according to Klebs, they are also liable to sclerosis, exactly like that of normal brain-substance.

A soft glioma often contains many thin-walled vessels, which may rupture and pour out blood into its tissue, tearing it up so that one can hardly discover any trace of the growth. As Virchow long ago pointed out, the disease may thus be mistaken for a simple cerebral hæmorrhage. Perhaps the most puzzling cases are those in which repeated extravasations occur, and the coagula become converted into tough opaque masses.

Not only gliomata, but all the less circumscribed forms of cerebral tumour are apt to set up in the adjacent brain-tissue morbid changes that can only be regarded as due to irritation caused by their presence. Such an affection sometimes assumes the form of "red-softening." More often it is what Rokitansky first described as "yellow-softening"—a state in which the medullary substance has a faint yellow tinge, and looks glistening like blancmange, but nevertheless retains its form when sliced or cut, and is not, in fact, softer than the rest of the brain. This is due to œdema. The colour depends on more or less of hæmoglobin- or hæmatoidin-crystals, and softening on interference with the blood-supply by pressure of the tumour (cf. p. 762).

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* In the retina during childhood, and perhaps also in the supra-renal capsule.

The presence of a new growth in the brain often causes accumulation of fluid in the ventricles. This hydrocephalus is sometimes due directly to compression of the veins of Galen or of the choroid veins; but Virchow showed that this explanation does not always apply, and his statement is confirmed by several cases in our records.

Statistics of cerebral tumours.—Of Dr Fagge's twenty-two cases in which there was a primary new growth in one of the *hemispheres*, sixteen were on the right side and six on the left. In only five of these was the patient less than thirty years old; the rest were pretty evenly distributed between the ages of thirty and sixty. On the other hand, out of fourteen cases in which a tumour was seated at the *base* of the brain, ten occurred in persons under the age of thirty. The six instances of cysts in the *cerebellum* were all in patients between twenty-one and twenty-seven years old. There were only two cases in which a tumour was found in children under the age of ten, and both these were in the cerebellum.

Tumour of the brain is more frequent in males than in females. Among forty-two cases at Guy's Hospital, the proportion was 27 to 15. This corresponds with the ratio of 10 : 6 given by Obernier. In the cases in which one of the hemispheres was the seat of the affection the preponderance of males was much higher, 16 : 6. Men are much more liable than women to blows and falls, and Virchow believes that new growths affecting the upper parts of the brain are often caused by injuries to the head. He has further stated that tumours in this position are generally gliomata, whereas at the base sarcomata and carcinomata are more often found.

In the forty-third volume of the 'Guy's Hospital Reports' Dr Hale White collected 100 unpublished cases of cerebral tumour, which were found in the deadhouse from 1872 to 1884 inclusive. The new growth was tubercular in nearly half of these cases (45), and it was a glioma in 24. Two of the remaining cases were glio-sarcoma, 10 sarcoma, 5 carcinoma, 5 gumma, 1 myxoma, and 1 lymphoma; while 4 were cystic.

Of the tuberculous cases, 34 occurred in male and 11 in female patients; more than half of these 45 patients were children under ten years old. The hemispheres were the seat of a tubercular tumour in 22 cases, the cerebellum nearly as often (20), the pons in 6 cases, the bulb and the crus cerebri in one each. Nineteen of the tubercular tumours were multiple.

Glioma was widely distributed among patients of different ages, from three years to sixty-two. Here also the male sex predominated in the proportion of 19 to 5, and all the patients under twelve were boys. The seat was the hemispheres in 13 cases, the cerebellum in 4, the pons in 2, the bulb in 1, the pituitarium in 3, and the dura mater in one only.

Of the four cysts, two were situated in the cerebellum. One of the cerebral cysts was a hydatid.

Considering locality only, 7 tumours of all kinds grew from the meninges, 7 were in the hemispheres, 28 in the cerebellum, 10 in the pons, 5 were in the corpus striatum or thalamus, and 3 in the pituitarium.

With these last figures may be compared those compiled by Lebert in the third volume of 'Virchow's Archiv' (1857). He found (beside 3 which began in the bones of the skull, and 40 in the dura mater) 21 in the hemispheres, of which 17 were on the surface, 4 in the cerebellum, 8 in the pons and bulb, and 3 in the pituitarium.

Symptoms.—In proceeding to discuss the clinical symptoms of a cerebral tumour, we must first remark that a *post-mortem* examination occasionally reveals a new growth when there had been no suspicion of any cerebral lesion during life. Thus we have in the museum of Guy's Hospital a specimen of a very hard growth, nearly as large as a pigeon's egg, attached by a pedicle to the ridge of the petrous bone; it was discovered accidentally in the body of an aged pauper woman. Another is a large malignant mass in the cerebellum, taken from a patient who died of cancer of the breast and liver. If she had lived a little longer, the tumour might have shown signs of its presence; and the same remark applies to the few instances in which one or more small tuberculous tumours have been unexpectedly found in the brain in cases of phthisis. All that is proved by such cases is that in the brain, as elsewhere, gross lesions may be latent during the early periods of their development. But of this fact there is abundant evidence of another kind. Thus when tubercular meningitis has been the cause of death, and when the patient had appeared to be perfectly well up to the first symptom of that disease, one sometimes finds one or more caseous nodules in the brain which are obviously of older date. Again, it sometimes happens that a person dies of what appears to be an acute illness, and that the only lesion found at the autopsy is a tumour, with softening of the surrounding cerebral tissue. A man was once admitted into hospital insensible, livid, and in a high state of fever, so that the diagnosis was typhus; he lived only three days from the time when he was first taken ill, but at the autopsy a small spindle-cell growth, the size of a bean, was found projecting into the fourth ventricle from the side of the aqueduct. Nevertheless, excepting one or two instances in which psammomata or other meningeal tumours have been unexpectedly discovered, Dr Fagge never found an example of a tumour in the brain of a patient killed by accident, or who died from some indifferent disease, like pneumonia, or ileus.

Again, while a cerebral tumour may long continue latent, symptoms, when at last they appear, may be not gradual and insidious but sudden in onset; thus a convulsive fit, or vomiting, or intense headache may be the first warning of illness in a person previously in apparent health. We must suppose that in such cases the contents of the cranium adapt themselves mechanically and physiologically to the gradually increasing intercranial pressure until at last some fibre gives way; just as a picture falls suddenly when the last strand of its cord breaks, though the process of decay was very gradual. Or pathologically we may compare the case to that of the compensation of valvular disease suddenly breaking down, so that an apparently healthy man within a few hours exhibits the symptoms of advanced disease of the heart.

The symptoms may be divided into two groups. Some of them are common to all, or nearly all, cases; others depend on the seat of the lesion. It will be convenient to describe the former group of symptoms first; and they may be arranged under three heads:—Cerebral symptoms, such as headache, vertigo, and mental disturbance or coma; vomiting; and lastly, secondary changes in the optic disc.

1. *Pain.*—This is the most constant of all the general symptoms, and

is generally the earliest indication that anything is wrong with the patient. Its seat sometimes, but only sometimes, answers to that of the lesion. The superficial nerves of the corresponding part of the scalp may be tender to pressure or percussion; or (as Romberg noticed) the act of holding the breath or of coughing may increase the pain—no doubt by producing venous congestion. The museum of Guy's Hospital contains a large tumour, three inches in diameter, found many years ago in the left hemisphere of a girl who had suffered severely from pain in the head, and who had declared that when she turned her head to one side she felt something move in its interior. Another preparation consists of a small growth from the dura mater, taken from an old woman who died of bronchitis. She had often expressed a wish that her head should be opened, because for years she had experienced anomalous pains and a sense of coldness in one spot, not larger than a shilling; this corresponded very nearly with the seat of the tumour that was found after her death.

The pain caused by a new growth may be of every degree from a dull headache to the most unbearable agony. It is sometimes constant, but generally undergoes exacerbation from time to time. In some cases, indeed, it is intermittent or paroxysmal, so that it may closely resemble ordinary migraine. Thus Abercrombie relates the case of a boy, aged six, who began to suffer from fits of severe sick headache, recurring at first about once a fortnight, and leaving him in good health in the intervals. After five or six months the attacks became persistent; and when two months later he died, a tuberculous mass was found in the cerebellum. A similar instance is recorded by Lebert.*

2. *Vertigo*, varying from a slight feeling of giddiness on standing up, to reeling and inability to walk or stand upright, is a frequent symptom of a cerebral tumour. It is most constant when the seat of disease is in the cerebellum. This symptom probably depends on disturbance of the branch of the Portio Mollis which supplies the ampullæ of the semicircular canals, but it is seldom associated with deafness or tinnitus aurium as in functional auditory vertigo (cf. *infra*). The effect of this symptom is seen in a reeling or staggering gait, and sometimes in sudden falls.

3. *Vomiting* is a frequent and most important symptom of tumours of the brain. It is independent of meals, and accompanied by more headache than nausea.

The bowels are usually constipated, as in other chronic cerebral diseases, and the abdomen is often hollow, the walls retracted, and the intestines empty. As a rule there is loss of flesh, and extreme emaciation sometimes occurs. But a boy of fourteen, who was in Guy's Hospital in 1867, became remarkably fat during his illness, and remained so until he died; and two or three years previously the body of a young woman in a similar condition was brought down into the *post-mortem* room. In each case there was a tumour at the base, growing upwards into the third ventricle. The writer has had two other cases under notice in which great increase in weight took place in patients suffering from tumour of the brain; both were young, and in both the seat was probably the cerebellum.

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* These cases seem to have an important bearing upon the general theory of the production of "cerebral symptoms" by local affections of the brain. The functional disorders—migraine, vertigo, epilepsy, and some others: "nerve-storms" or "explosive neuroses"—are liable to be set up by a variety of exciting causes. Now my hypothesis is that a tumour or a tubercle causes a transitory vertigo, or an epileptiform fit, or an attack of sick headache in exactly the same way as any other disturbing agent. I conceive that the nerve-storm so produced has precisely the same seat as when it is merely the result of over-fatigue, or irritation of the generative organs, or disorder of the stomach. And I think it is probable that frontal headache, even when unattended with the other characteristic symptoms of an attack of migraine, is yet very often of that nature. If so, pain in the forehead would point less directly to the anterior part of the brain as the seat of a tumour than occipital pain to the cerebellum or posterior lobes.—C. H. F.

4. *Changes in the optic disc and retina.*—In 1860 von Graefe communicated to the German 'Archiv für Ophthalmologie' a short paper, in which he stated that inflammation of the optic nerves within the eye sometimes occurred as a complication of cerebral diseases. It was already well known that *blindness* was a frequent effect of tumours of the brain, even when they did not involve the optic tracts, but the condition of the optic disc in these cases was unknown. It was soon afterwards ascertained that there may be redness and swelling of the optic papilla although the patient sees perfectly well.

Von Graefe himself believed from the first that there were two ways in which these changes in the disc could arise, and that these could be distinguished by the ophthalmoscope. Either an increase of intra-cranial pressure, acting through the cavernous sinuses, might mechanically obstruct the return of blood through the retinal veins; or else an inflammatory process at the base of the brain might extend as a "neuritis descendens" along the optic nerve to the disc. These two conditions are often seen together. The one, known in German as the "Stauungspapille," has been translated by Dr Allbutt as the "Choked Disc," or "Ischæmia of the Disc." The other is called "Optic Neuritis," "Papillitis," or "Neuro-retinitis descendens." There is also a third change, "Atrophy of the Disc," which is usually consecutive to neuritis, but may have an independent origin.

The choked disc.—There is no better description of this affection than that which von Graefe gave in his account of the first case that came under his observation. "The papilla," he says, "was greatly and irregularly swollen, rising steeply on one side and falling gradually on the opposite side to the level of the retina. . . . . . Instead of being transparent its tissue looked grey and opaque, with an extremely deep reddish tint, and the adjacent part of the retina had the same appearance, so that the choroidal margin was completely hidden. The opacity was diffused, except that with the direct method of examination one could perceive a striated appearance following the course of the fibres of the optic nerve. The retinal veins were dilated, exceedingly tortuous, and obscured here and there by dipping into the opaque tissue; the arteries were comparatively small. The turbidity of the retina diminished gradually from the disc outwards over a zone of rather more than 2 mm. in breadth, so that it occupied an area 5 mm. in diameter (including the disc itself)." In a second case there were ecchymoses in the part of the retina near the disc.

The prominence formed by the optic disc in a case of this kind appears to be easily recognised after death when the eye is removed and laid open; an excellent illustration of it is given by Dr Allbutt. In one instance in which an intercurrent attack of smallpox destroyed life at an early stage of the ocular affection, Cornil found that the microscopical appearances consisted in an infiltration of the connective tissue with serum, and ecchymoses in the adjacent part of the retina ('Arch. Gén.,' 1868, ii, p. 679). The absence of grave morbid changes is proved by a case of Mr Lawson's, in which the presence of a hydatid cyst within the orbit caused an extreme state of "choked disc." but four days after puncture of the tumour the engorgement had almost disappeared. A drawing in 'Pagenstecher's Atlas' (pl. xxxi, fig. 7) shows the nerve-fibres bulging outwards, so as to separate the peripheral layers of the retina from the choroid. In some more advanced cases of von Graefe's, examined by Schweigger and Virchow, the connective-tissue elements, the vessels, and the nerve-fibres were all found swollen, and the latter were beginning to degenerate, while in the outer coat of the vessels there was an overgrowth of nuclei.

The choked disc was attributed by von Graefe to increased pressure in the cavernous sinus, aided by the constricting action of the sclerotic ring. He argued that if from any cause the flow of blood along the retinal veins was obstructed, the unyielding structure would give rise to a sort of strangulation at the point where they pass through it, and so he explained the fact that the congestion was limited to the intra-ocular termination of the optic nerve; but (as Dr Hermann Schmidt urged in vol. xv of the 'Arch. f. Ophth.') the ophthalmic vein communicates so freely with the facial vein that it is difficult to see how pressure upon the cavernous sinus could appreciably interfere with the escape of blood from the eyeball, nor does it appear probable that the wall of the sinus would yield to any moderate force. These considerations are confirmed by a case under Dr Fagge's observation, in which the sinus on one side was completely obstructed by softening thrombus; the corresponding optic disc had been noticed during life to be perfectly normal.*

Optic neuritis.—In this condition, as compared with choking of the disc, its swelling and redness are less marked; it looks more opaque, and the morbid process involves its middle and outer as well as its inner layer. Dr Allbutt endeavours to describe the appearance of the disc by saying that there is not "a circumscribed intense redness, or brownish grey, but rather a wash of reddish lilac or a grey tint;" and he adds that one does not perceive "a multitude of minute branches and capillaries," such as give a "mossy" look to the choked disc, but that the main trunks become distended and tortuous.

On microscopical examination of an inflamed disc, the nerve-fibres are found enlarged and beaded. The trunk of the nerve in its whole length shows an accumulation of cells and nuclei between its fasciculi.

As stated above, von Graefe ascribed neuro-retinitis to direct extension of inflammation downwards; as, for instance, from a basal meningitis; but this explanation is not always applicable. In other cases it is secondary to a choked disc, in the same way as those chronic affections which occur in the lungs and liver and kidneys, when there is obstructive disease of the heart. As Hulke long ago remarked, mixed forms are more commonly seen than typical specimens of a "Stauungspapille" or of neuro-retinitis. Indeed, Gowers holds that congestion of the disc is not the result of intra-cranial pressure at all, but it is always inflammatory or irritative in origin, and that the distinction between the choked disc and optic neuritis is one of degree only. This opinion has long been adopted by Mr Higgens, who for some time had followed von Graefe's lead.

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* Probably Schmidt's explanation is correct; he found experimentally that an injection of Prussian blue into the arachnoid cavity (subdural space of the brain) makes its way through the optic foramen into the space between the two sheaths of the optic nerve, and fills a fine network of lymphatic channels within the lamina cribrosa (formed by the sclerotic ring) between the very fibres of the nerves themselves. He therefore suggests that some of the fluid which is always present in small quantity in the arachnoid cavity is driven into the optic nerve in a similar manner whenever the intra-cranial pressure is from any cause increased. As a matter of fact, a watery liquid has more than once been found distending the sheath and giving it a bulbous appearance, and this liquid readily escapes as soon as a puncture is made. One difficulty is in explaining on Schmidt's theory why cerebral hæmorrhage does not cause choked discs. Is it because the great force suddenly exerted flattens the sheath of the nerve, and closes the channel through it?—C. H. F.

Atrophy of the disc.—Neither congestion of the disc nor neuro-retinitis is a permanent affection. They are not even stationary; for the process, if the patient lives long enough, must either subside or end in atrophy. In cases of cerebral tumour the disc becomes less and less swollen, and at length is quite flat, or even sinks below the level of the surrounding retina. Its red and grey tints become a dirty white. The tortuous veins diminish in size; spots of hæmorrhage fade and are absorbed. The outline of the disc remains for some time blurred and irregular, and streaks of exudation are to be seen in the course of the retinal vessels. This description of "consecutive atrophy" is taken from Dr Allbutt, and accords with general observation. He goes on to say that these appearances also are transitory. Little by little the disc clears up; its edge becomes sharply defined; its surface dead white and glistening. Its condition is then undistinguishable from one which has been preceded by no swelling or inflammation of the disc—the primary or "simple white atrophy" of writers.*

It is surprising how little impairment of vision may attend these changes in the optic discs. There is neither pain nor photophobia, and if the patient is aware of his condition at all, before he is completely blind he sees objects more or less indistinctly, as if through a mist. When blindness sets in, it sometimes seems to come on almost suddenly, although the changes in the discs no doubt developed gradually. This occurred, for instance, in a patient under Dr Fagge in 1878; about three weeks before her admission to hospital she went to sleep one afternoon, and on waking up found that she was totally blind.

When amaurosis is complete, the pupils are widely dilated and motionless. But while the impairment of sight is partial they are of normal size, and their movements are sometimes not even sluggish.

It must be remembered that although the optic disc is constantly more or less affected in cases of cerebral tumour, it is also liable to similar changes from abscess of the brain, from Meningitis and from Tabes or general paralysis, as well as from some infective disorders, as Enteric fever.

When a choked disc, or a case of optic neuritis, passes into a state of atrophy, the patient's sight often becomes progressively worse; but sometimes it shows improvement. Thus a patient of Dr Goodhart's, who at one time could see absolutely nothing with her right eye, was ultimately able to read Snellen's 2½, though with difficulty, at a distance of about a foot. When vision has been but slightly impaired during the early stage of the disease, it is sometimes regained later on; but, if actual neuritis has been present, the disc invariably becomes more or less atrophied, and it never resumes its normal appearance.

5. *Mental symptoms*—including irritability of temper, depression of spirits, loss of memory, mania, or impairment of intelligence—are the least constant and characteristic effects of a cerebral tumour. They may sometimes be observed when a tumour lies at the base of the brain. Thus we have in Guy's Museum a specimen of cholesteatoma, as large as a pigeon's

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* Pagenstecher first showed that all the nervous elements may disappear, and be replaced by coarse connective tissue.

egg, which compressed the under surface of the pons and cerebellum; the patient was at one time in the hospital with paralytic symptoms, but he became maniacal, had delusions, and was unmanageable, so that he was removed to Colney Hatch Asylum, where he died. On the other hand, the mental faculties are not infrequently retained nearly to the last, when one of the hemispheres is the seat of the tumour. As a rule the mind is not disturbed until near the close of the disease.

Friedreich and Obernier speak of excessive sleepiness as a principal symptom in two cases which severally came under their observation. The most usual termination of cases of tumour and cerebral abscess, as of the vascular lesions described in the last chapter, is by stupor, which gradually passes into *coma*. Sometimes the patient lies for weeks without taking the slightest notice, passing all his evacuations under him, and showing no sign of intelligence, except that he slowly swallows the food that is put into his mouth. Many years ago Sir William Gull had under his care a boy with other symptoms of cerebral tumour, who manifested a remarkable retardation of intelligence. If a question was put to him he seemed not to heed it; but after many seconds, when the questioner had passed on to talk to some one else, he would deliver, word by word, a reply which showed that he perfectly understood what had been said. In other cases the mental condition undergoes changes possibly depending on varying degrees of pressure by ventricular effusion; after lying in a stupor for a week or more the patient may recover his senses, remain conscious for several days, and then relapse into his former state.

The *temperature* of the body is often one or two degrees below normal; but before death pyrexia may appear. Obernier speaks of a rapidly advancing case in which every exacerbation of the headache was associated with a marked fall in the rate of the pulse.

Other general symptoms which may be sometimes observed in cases of cerebral tumours are nystagmus, or oscillation of the eyes, most frequent in lesions of the cerebellum; contracted, dilated, or unequal pupils; incontinence or retention of urine; and, in the latter stages, bradycardia with slow and irregular respiration, which sometimes takes on the Cheyne-Stokes character.

The heart may continue to beat for some little time after breathing has ceased; in one case it went on for thirty-five minutes, while artificial respiration was kept up. In such cases it is wonderful how quietly life departs, without a gasp or moan, or the movement of a limb.*

Localising symptoms—(*a*) *Convulsions*.—We now turn to other symptoms, which not only indicate in general the presence of a tumour, but also point more or less definitely to its exact *seat*; they are what Jackson terms "localising symptoms."

The first of these is the occurrence of muscular spasms, not constant but occasional, or coming in fits. Sometimes the involuntary spasmodic movement is confined to a single group of muscles, those of one thumb, or of the angle of the mouth, or of the eyelids, or of one foot; but more often several muscular groups are involved and follow an orderly sequence, more or less accurately repeated in each fit. The spasms are clonic and are free from pain. While these convulsions resemble those of epilepsy, as Hughlings Jackson first recognised, they are distinguishable from idiopathic epilepsy. We may term the fits "epileptiform," or, as is common on the Continent, "Jacksonian epilepsy," or may use the convenient term "eclampsia," which has long been applied to the epileptiform fits of the puerperal state or of Bright's disease.

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* I was once called to see a patient, whose symptoms pointed to the presence of a cerebral tumour. On my arrival at the house the doctor came out to speak to me, leaving her husband and two women with her. When we went to her bedside a few minutes later we found her dead, except that a slight flickering of the pulse was still perceptible. Not the slightest change had been observed.—C. H. F.

The characteristics of these convulsions distinguishing them from true epilepsy are the following:—First, they do not affect the whole body, but one limb (monospasm) or perhaps two, or one hand or one finger. Secondly, there is no loss of consciousness; or if there is any, it follows, not begins the fit. Thirdly, the spasms are succeeded by loss of power in the muscles affected, varying from almost complete paralysis to a slight diminution in power; this paresis is in most cases only temporary. Minor points of distinction are that the tongue is not bitten, and that the patient does not fall to the ground or foam at the mouth.

Beside these clonic convulsions, tonic muscular spasm is also an occasional symptom of cerebral (particularly of cerebellar) tumours, leading sometimes to spastic paraplegia, or rigid hemiplegia, but more often to strabismus, or to athetosis, or, most frequently of all, to retraction of the head from spasm of the splenius capitis and complexus.

(*b*) *Paralysis.*—Apart from the temporary loss of power which follows spasm, tumours of the brain often produce decided and permanent hemiplegia, though it is seldom so marked as that described in the last chapter which is caused by cerebral hæmorrhage. Ptosis or strabismus is not uncommon, and occasionally loss of power in the lips or tongue, or monoplegia of one limb or a part of it. More frequently seen than any local paralysis is a general loss of power over the limbs on both sides, shown by tottering gait, feeble grasp, and considerable loss of muscular strength when tested by the dynamometer.

Local anatomical diagnosis.—The symptoms above described give us no help in judging of the *nature* of the lesion. They make it certain that it is a tangible "gross" anatomical condition, and that this condition is not hæmorrhage, embolism, or thrombosis; but whether we shall find after death a tuberculous or gliomatous tumour, a cyst, or an abscess, must be decided on purely pathological grounds—the frequency of this or that growth at different ages, the presence of a source of suppuration, or of a malignant growth, or of tubercle, or of syphilis in other parts.

In deciding the probable *seat* of a tumour, we are guided by the localising symptoms above described, spasm and paralysis of muscles.

It is, however, often difficult to decide whether the symptoms are due to irritation or destruction of centres and nervous tracts. In the latter case they would be those of unbalanced action of the corresponding parts physiologically antagonistic. Hence we cannot attach an exact meaning to inequality of the pupils.

It is remarkable how great a size may be reached by a slow-growing mass in the brain, without its affecting surrounding structures; while, again, a small tumour may so compress adjacent parts as to produce striking symptoms.

The base.—Taking the base of the brain first, and passing from before, we meet with the simplest of localising symptoms, those which depend on paralysis of the several cranial nerves.

It is easy to see that complete *anosmia* points to the presence of disease of the ethmoid bone, or involving the inner and lower parts of the two anterior lobes; while loss of smell on one side may be due to a lesion implicating one olfactory bulb, or the root of the nerve adjacent to the Sylvian fissure.

A growth pressing upon the optic chiasma produces different effects according to its exact situation. There may be more or less complete loss of sight in one eye; but, since atrophy of the disc is almost certain to be present, this symptom would scarcely help us. More significant is the limitation of vision to half the field in each eye, or *hemiopia*. Assuming that the inner fibres of each optic tract cross at the chiasma and the outer ones do not, three varieties of hemiopia may be described as follows:

(1) Compression of the centre of the chiasma, cutting off the decussating inner fibres of each optic nerve, will cause loss of vision over the temporal field in each eye (*double temporal hemiopia*); (2) compression of one optic tract will deprive the patient of the nasal field in the corresponding eye (*homologous lateral hemiopia*); (3) loss of vision in the nasal fields of the two eyes (*double nasal hemiopia*) can only be due to a double lesion, symmetrically placed so as to interfere with the non-decussating outer fibres on each side. Examples of all these forms of hemiopia are known to occur. Homologous lateral hemiopia is very common in attacks of migraine; cases of double temporal hemiopia have been recorded in which a large tumour lay in the middle line, involving the chiasma, and a remarkable one by Dr Weir Mitchell due to pressure of an aneurysm of an abnormal anterior communicating artery ('Journ. Nerv. and Mental Dis.,' Jan., 1889); and cases of double nasal hemiopia, in one of which, recorded by Knapp, the pressure necessary was effected by dilated and atheromatous branches of the circle of Willis.*

Intra-cranial growths sometimes produce *deafness* when they occupy, or extend into, the space between the internal auditory meatus and the side of the pons. Jackson has met with one case in which there was loss of hearing from this cause without any facial paralysis. In another instance complete deafness in both ears was imperfectly explained by the autopsy, for the only tumour was one the size of a hazel-nut on the *left* side.

With regard to the other cranial nerves, there is little to add to the remarks made when describing the symptoms of their primary lesions (see pp. 577—590). We can sometimes infer the seat of a growth with great accuracy from the implication of some nerves while others escape. Thus, in one instance, the fact that all the muscles of the eyeball were paralysed, while no part of the fifth nerve was interfered with except its ophthalmic branch, proved that the seat of disease was in the sphenoidal fissure. On the other hand, it is sometimes clear that no single lesion will explain every feature of the case, a conclusion which (as shall presently see) may be of considerable diagnostic importance. Paralysis of the third nerve, if due to an intra-cranial tumour, points parti larly to a syphilitic gumma of the base, but strabismus or ptosis may be due to a tuberculous tumour or to basal tuberculous meningitis.

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* Those who still hold that in man, as in some animals, the optic nerves undergo complete decussation, must assume that "double temporal hemiopia" would be caused pressure on the *front* of the chiasma, while pressure on the *back* of the chiasma by a single growth would account for "double nasal hemiopia." Both these kinds of hemiopia are very rare, and the only explanation of the far more common "homologous lateral hemiopia" would be the untenable one of a lesion being so placed *on one side* of the chiasma as to compress a part of the optic nerve, and also a part of the optic tract on t[illegible] side.

The cerebellum.—The localisation of a tumour in the cerebellum is often attempted, and is not infrequently successful. Beside vomiting, convulsions, occipital headache, contraction of the muscles at the back of the neck, blindness, nystagmus, and vertigo—no one of which symptoms is peculiar to lesions of the cerebellum—there may often be observed a reeling, staggering gait, like that of a drunken person (cf. p. 708). The early experiments of Flourens on pigeons showed that excision of the cerebellum rendered the bird unable to walk or to fly; and he concluded that the function of the organ was one of co-ordination. But this was an erroneous conception of the nature of co-ordination no less than of the functions of the cerebellum. Indeed, we have already seen that a large part of the process by which the actions of the individual muscles are harmonised and combined takes place in the spinal cord. The peculiar function of the cerebellum, if it have any, is still unknown.

When the staggering is accompanied by a tendency to fall in one particular direction, we might expect that this would indicate which side of the cerebellum is affected. But tumours of the cerebellum often attain a great size, and compress adjacent parts or interfere with their supply of blood. Moreover the same lesion may produce opposite effects, according as its action is "destroying" or "discharging."

In 1877 an autopsy in a case of Dr Frederick Taylor's showed a mass of yellow tubercle in the cerebellum, rather to the right of its mid-line; the right lateral lobe was universally pale and softened, and full of granule-masses. The patient was a boy five years old. It so happened that the exact seat of the mischief had been indicated several months before his death by a bulging on the right side of the occipital bone, which was so much thinned at one spot that it would yield on pressure, and rebound like a piece of tin or cardboard. At the same time it was noticed that when he sat up in bed he had no balancing power; but he did not roll over on one side rather than on the other. The eyes were affected with a peculiar form of nystagmus, which might perhaps have been turned to account as a localising symptom. "It came on only when he lay on the left side; the eyes would then gradually fall over to the left, and be suddenly jerked back to the right; and these movements would be repeated rhythmically. When he sat up in bed there was a continuous rhythmical movement of the head from left to right." Afterwards, six weeks before he died, "he lay on his back, with his head turned to the left; his eyes were then directed to the left side, and jerked from time to time upwards."

Hitzig, following Purkinjé, showed ('Reichert's Archiv,' 1871) that by passing a galvanic current through the head of a healthy person, from one mastoid process to the other, one can obtain oscillating movements of the eyes like those observed in Dr Taylor's patient; and they have been referred to disturbance of the cerebellum. Ferrier, too, found that galvanising the exposed cerebellum of monkeys and other animals caused the eyes to deviate in different ways, according as the poles were applied to different regions of its surface. His experiments seem to indicate, as a rule of localisation, that when any part of the cerebellum is the seat of irritation the eyes become turned in the corresponding direction, whether to the right or to the left, upwards or downwards. In many cases analogous movements of the head accompanied those of the eyes. Thus a tendency to fall backwards, which has sometimes been noticed in cases of cerebellar disease, would point to the upper surface of the organ as its seat; for Ferrier found that galvanising that part in monkeys led to upward movements of the eyes, and to throwing back of the head. Dr Taylor's patient had also left hemiplegia, which was no doubt due to compression of the right side of the pons by the tumour. Hemiplegia in cerebellar disease appears to be always produced in that way, and it is usually "crossed," affecting the limbs on the opposite side of the cerebellar lesion. But the functional relations of each half of the cerebellum are nevertheless chiefly with the limbs of the same side of the body through the connections of the fibres of the middle peduncle with nuclei in the opposite half of the mesencephalon. This is proved, not only by anatomical and physiological investigations, but also, and still more convincingly, by the pathological fact that when there is congenital unilateral atrophy, affecting one hemisphere of the brain, one crus cerebri, and one side of the deep part of the pons, the wasting is found in the opposite side of the cerebellum, as it is in the opposite side of the spinal cord.

On the whole, it may be said that when there are general symptoms of an intra-cranial tumour, namely, headache, vomiting, and optic neuritis, especially optic atrophy and amaurosis; when there is no hemiplegia, but only general muscular weakness; when there is nystagmus, spasm of the muscles at the nape of the neck, and decided vertigo and loss of power to maintain equilibrium; the diagnosis of the cerebellum as the seat of the lesion is almost certain.

This last symptom, disturbance of equilibrium, is said by Nothnagel to depend on lesion of the superior vermiform process, and this belief is shared by other observers. Nystagmus also is most constant when the tumour or other destroying lesion is in the middle or vermiform lobe.

A tumour in the inferior vermiform process may press on the veins of Galen or on the aqueduct, and so cause secondary hydrocephalus, as in a case of the writer's where a large mass of tubercle was found at this point in a boy who had been watched from the age of four to nearly six.

Tumours of the lateral lobes of the cerebellum appear to have no special symptoms, unless paresis or spasm limited to one side should coincide with more general cerebellar symptoms.

The motor area of the cerebral cortex.—Passing now to the regional diagnosis of lesions involving the motor tract, we may observe that the greater part of the description of hemiplegia given in the last chapter applies to the same symptom when it is produced by a new growth. But whereas we have seen that certain parts of the brain are far more liable than others to suffer from the effects of obstruction or rupture of a cerebral artery, the distribution of tumours is irregular and capricious. It is wonderful how large a tumour may be found in the very substance of the motor tract without abolishing its functions.*

A growth situated in the superficial convolutions of one *hemisphere* may affect the movements of the opposite side of the body, usually as above described by causing spasms, followed by a transitory paralysis. Bright observed in the first volume of the 'Guy's Hospital Reports' (1836), that in fits due to a local lesion consciousness was not lost. We have seen how Jackson's investigations thirty years later carried on the inquiry from this point. He insisted on the fact that *hemispasm*—the "mobile counterpart" of hemiplegia—must indicate a condition of "instability" in the convoluons which discharge through the corpus striatum, and he set to work to etermine the exact seat of the lesions when this symptom had been present.

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* In 1869 a boy, aged four years and a half, died in Guy's Hospital under my care after an illness of fourteen months' duration. Three months before his death he could sit up in bed, although he rolled about and his balance was easily upset. A month later he "could move his legs a very little." Dr Moxon found that the pons was occupied by an immense caseous tubercular mass, which consisted of two halves fused together, and preserving almost exactly the normal shape of the part; only a thin shell of nervous matter remained. It seemed marvellous that life could have been maintained while the mass was growing to such a size.—C. H. F.

In 1870 Fritsch and Hitzig made known the fact that, instead of the urface of the brain being insensible to electrical stimulation as physiologists ad hitherto believed, there were in the dog certain parts of the convoluions which reacted to the make and break of galvanic currents in a very efinite way, and each set in motion some particular limb. They laid down he position of "cortical centres" for the movements of the neck, face, fore- nd hind-leg respectively. Three years later Ferrier repeated these xperiments with the faradic current, employing various animals, and at ength monkeys, in which the principal convolutions are comparable, one by one, with those of the brain of man.

Ferrier placed his motor centres in the posterior central (ascending parietal) as well as in the anterior central (ascending frontal) convolution, and also in the postero-parietal lobule; and his sensory centres, in the back part of the third frontal (Broca's) gyrus for speech, in the angular for vision,* and in the superior temporo-sphenoidal convolution for hearing.

He agreed with Hitzig in placing the centre for the leg close to the median line. He made the centres for the arm and hand occupy the ascending parietal convolution nearly s far outwards as the fissure of Sylvius, as well as the ascending frontal and the superior frontal convolutions outside and in front of the leg-centres. The movements of the lips, ongue, and mouth he connected with a series of centres occupying the lower part of the scending frontal, and with one in the supra-marginal convolution. Centres for the movements of the eyes were assigned in the two upper ("first" and "second") frontal onvolutions. Stimulation of the angular gyrus, or of the superior temporo-sphenoidal onvolutions, also caused the eyeballs to move, with dilatation of the pupils; but he upposed these movements to be reflex from excitement of the senses of sight and earing.

Since the publication of Ferrier's book in 1876, Nothnagel, Munk, Goltz, nd other experimenters in Germany, and G. F. Yeo, Schäfer, Horsley, Beevor, and Sherrington in England, have repeated and varied his experiments.†

The practical difficulties have been—in faradic stimulation, the danger of a current spreading, either too widely or too deeply; in destructive operations, the appearance of much wider and more complete ablation than proved to be the case; in pathological observations, the infrequency of locally circumscribed single and uncomplicated lesions; the mistake of ascribing the immediate instead of the permanent symptoms to a destructive lesion (whether operative or morbid); and, generally, the fallacies resulting from inhibition of distant centres, and from direct or indirect mechanical pressure.

These difficulties have now been overcome, and it is certain that there are excitable areas in the neighbourhood of the fissure of Rolando which are connected with definite movements. The exact locality of these motor centres has been accurately ascertained in monkeys as well as in dogs and cats, and has been confirmed by the crucial proof of operation upon human patients.*

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* In the second edition of Dr Ferrier's work, 'The Functions of the Brain,' 1886, he ncluded the occipital lobes in the visual area (pp. 271 *et seq.*).

† At the International Medical Congress which met in London in 1881, Prof. Goltz rought a dog from Strasburg which had survived extensive destruction of successive ortions of the cerebral cortex, and this living witness was confronted by a monkey which was brought forward by Dr Ferrier. After observation of the functions of both animals hey were destroyed, and the brains were subsequently submitted to most exact and horough anatomical examination by Dr Klein, Mr Langley, Mr Schäfer, and Dr Gowers. The account of the discussion in the 'Transactions' of the Congress (vol. i, pp. 218—240), nd the detailed reports by Langley and by Schäfer, published in the 'Journal of Physioogy' (vol. iv, pp. 248—326), furnish an admirable example of the difficulty of ascertaining nd interpreting scientific facts and of success in at least the former task.

The motor or Rolandic area includes the ascending frontal with the closely adjacent part of the third, second, and first frontal convolutions, and the ascending parietal, together with the inner aspect of the same, which forms part of the marginal gyrus, and part of the superior parietal lobule (postero-parietal of Huxley and Turner). Irritation of these parts causes definitely distributed clonic spasms on the opposite side of the body—a "discharging" lesion; ablation of the same parts causes definitely distributed loss of power in the same muscles on the opposite side of the body—a "destroying" lesion.

The area which corresponds with certain movements of the *leg* comes first, occupying the borders of the fissure of Rolando close to the median line, and extending over the adjacent parts of the ascending frontal (or præ-central), the ascending parietal (or post-central), and the inner surface of the same part of the hemisphere, which corresponds to the marginal convolutions immediately behind the calloso-marginal fissure. The area which corresponds with movements of the *arm* is below and external to the last, and occupies the ascending frontal and parietal convolutions opposite the second frontal gyrus in front and the angular gyrus behind. The area which corresponds with movements of the *facial muscles* appears to be lower down in the same direction, in front and behind the lowest point to which the fissure of Rolando reaches. Lower still, at the angle between the ascending and horizontal branches of the Sylvian fissure, opposite the third frontal convolution, this region marches with the gyri operti, the insula, and Broca's aphasic region. Lastly, high up and internal is a motor area which lies in front of that for the lower extremity, occupying the back part of the first frontal convolution, which is believed to be associated with movements of the trunk on the opposite side.†

These results of experiment have been confirmed by clinical experience. Some of the earlier cases may, from their historical interest, be briefly cited, but every year many similar ones have been since recorded by Jackson, Gowers, Bastian, Ross, Hadden, Sharkey, and numerous other observers in this country; and a still larger number in France, Germany, and America.

(1 and 2) Two cases of Jackson's (one recorded in the 'Med. Times and Gaz.' for 1872, the other quoted by Ferrier), in each of which the spasms always began in one thumb, and a lesion was found in the opposite first frontal convolution. (3) A case at Guy's Hospital where we found in the back part of the first frontal gyrus a small glioma on the right side; there had been repeated fits without unconsciousness, starting mostly in the left foot, but occasionally in the left arm. (4) A patient of Griesinger's, who was attacked with

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* In the congress of physiologists which met at Bâle in September, 1889, Mr Horsley and Dr Beevor demonstrated on a monkey under anæsthetics the accuracy with which predicted movements of the thumb, forearm, and shoulder, the eyes, and the tongue, could be produced by faradic stimulation of definite areas of the cortex.

† Centres for the muscles of the eyeballs, for movements of the tongue and jaw, and for movements of the tail, have been ascertained with more or less exactitude in the monkey and the dog. The difficulty of comparing the results, even in the former animal, with its different mode of progression and of prehension from human physiology, is obvious, and is fully recognised by Ferrier. Disturbance of the ocular muscles in man appears to be more often connected with lesions of the motor nerves, of the bulb or of the cerebellum, than with those which affect the cerebral cortex.

sms in the right leg, afterwards affecting the right arm, the face, and the
ticercus lay close to the left side of the falx cerebri, in such a position that
xtremity coincided with a line drawn vertically upwards from the ear.
by Dr Dreschfeld, of Manchester ('Lancet,' 1877). The convulsive move-
with a sudden clenching of the left fist, flexion of the wrist, and pronation of
while the left angle of the mouth was at the same time strongly drawn down-
l syphilitic lesion was found in the adjacent parts of the ascending parietal
rginal convolutions. (6) A case observed by Hitzig. The patient was a
r who was wounded on the right side of the head near Orleans on December
On February 4th, 1871, he was attacked with clonic spasms affecting the left
outh and nose, the eyelids, and afterwards the fingers on that side. After
as found to be a local necrosis of the parietal bone, and an abscess in the
g frontal convolution. (7) Another instance, very similar, has been recorded
'Virchow's Archiv,' lvi, p. 289). The patient, who had fallen from a railway
acked with convulsions limited to certain muscles on the right side, especially
ngle of the mouth, the ala nasi, the eyelids, and the tongue. After death the
bone was found fractured, and the surface of the brain crushed on each side
fissure at the lower end of the fissure of Rolando. (8) A case of Dr Gowers
ourn.,' 1874) in which spasms began in the left angle of the mouth, involving
e frontal muscle on each side, but in which the only local lesion appeared to
ood situated above the right lateral ventricle, just inside the gyrus fornicatus.
ainly does not correspond with the observations of Hitzig and Ferrier; but it
radict them, as if, for example, a small tumour were to be found in the
when limited spasms had been present during life. (9) A case reported by
n,' April, 1880) of convulsions in the left arm and leg following hemiplegia,
ubject of phthisis. At the autopsy a tubercular growth was found occupying
the fissure of Rolando on the external aspect of the right hemisphere, and
the internal aspect as well. (10) In a case of Gowers' there was thrombosis
r longitudinal sinus and of some of its afferent veins, with hyperæmia of
three frontal convolutions, situated further forwards than any recognised
or centres;" but in that instance the fits were general, although they began
ovement of the hand to the head; and death occurred within two hours from
cement. (11) In a patient of Buzzard's, a girl of eighteen, a tumour of
walnut was found in the white substance of the *left* hemisphere, extending
grey matter of the gyrus fornicatus; she had fits beginning with an aura
st, but no spasm.

of the early cases reported by Ferrier, Jackson, Bourneville, and
rs quoted by Bastian, there were first spasms of the thumb,
r leg, and afterwards paralysis—and this is certainly the rule.
however, circumscribed convulsions supervened in a paralysed

instances have been observed by Gowers of congenital absence
, and by Bastian of a wasted arm, associated with atrophy of the
cending parietal gyrus; and by Dickinson of an amputated
imilar atrophy of its motor area in the cortex.

gia or paralysis of a part of a limb only, associated with spasms, is
ly caused by a lesion of the motor area of the cortex. If spasms
paralysis affect the face we fix the cortical lesion in the lower
Rolandic area; if the hand, in the middle third of the ascending
arietal convolutions; and if the lower extremity, in the upper
same region. Monoplegia, or very limited motor paralysis, as
nb, is almost certain to be due to a cortical destructive lesion.
is at all affected it appears only to affect the sense of touch,
ccompanied by pain, and to be of only temporary duration. As
ry symptoms are absent even at first.

brum outside the motor area.—Localising symptoms are far less
n a lesion is seated in the cortex beyond the motor area.

n is little affected by the most extensive lesions. We have
ordinary paralysis, whether hemiplegia or paraplegia, affects

movement much more than feeling; and the cases of most marked anæsthesia are functional, in which no lesion can be expected even if we had the opportunity of searching for it. There is, however, reason to believe that the motor area above described is to some extent a sensory area also. Special cortical centres for tactile sensibility, pain, and sense of temperature cannot at present be recognised, but the internal aspect of each hemisphere, particularly the gyrus fornicatus, appears to be a seat of sensory perceptions.

Of the special senses, the olfactory centre can be localised in the uncinate gyrus, and that of vision in the angular gyrus and occipital lobe, including its inner surface, the *cuneus*. Lesions in various parts of this extensive area have been known to produce hemiopia, or less frequently, crossed amblyopia. The auditory centre in the superior temporo-sphenoidal gyrus has been found atrophied in deaf mutes.

No special localising symptoms can be connected with cortical lesions of the front part of the frontal lobes. Rather their absence in a case of probable tumour would lead to this part of the cerebrum being fixed on as a probable seat. The most frequent symptoms in cases of frontal tumours are lethargy, listlessness, and unwillingness to speak, or loss of memory and impairment of intelligence. The same kind of symptoms are found in monkeys and dogs to result from extensive destruction of the frontal lobe anterior to the motor area, although in some cases conjugate deviation of the head, or eyes, or both, have been observed.

Cerebral hæmorrhage, the commonest of "destroying" lesions, is, as we found (*supra*, p. 726), rare in these "non-motor" regions compared with the external capsule, the corpus striatum, the pons, and the white substance. But tumours and cerebral abscesses are much less rare, so that we have sufficient evidence of their negative effects.

In a case under the writer's care in which the symptoms pointed to a cerebral tumour, its locality was ascertained by absence of other than general symptoms of tumour and by signs of pressure on the left orbit; the skull was therefore trephined, and a large growth was found in the anterior part of the frontal lobe.

A patient of Dr Shaw, in 1891, whose sense of smell was noted to be decidedly deficient during life, died in Guy's Hospital with symptoms of growth in the brain, and a cyst was found in the temporo-sphenoidal lobe.

A tumour of the frontal lobe, successfully removed by Prof. Durante, of Rome, had been localised by the absence of motor symptoms, by slight mental weakness, loss of smell, and displacement of the left eye. (Trans. Internat. Med. Congr., 1887.)

Affections of the parietal lobe are for the most part without localising symptoms, but the presence of "sensory agraphia" or "word-blindness" (cf. *supra*, p. 742) would point to a lesion of the lower part of the left parietal lobe behind the ascending convolution.

The occipital lobe and the cuneus appear, from the experiments of Ferrier, Munk, Schäfer, and other physiologists, and from the few cases of human pathology which bear on the point, to share with the angular gyrus a perceptive visual function.

The superior temporo-sphenoidal gyrus is the centre of hearing, and a lesion of this region is likely to be followed by sensory aphasia or "word-deafness."

The corpus striatum.—We have seen that ordinary hemiplegia depends, as a rule, on interruption of the motor tract as it passes between the caudate nucleus and lenticulus (anterior limb of internal capsule); or between the alamus and lenticulus (the internal capsule behind the genu), or upon wering of the white fibres of the external capsule as they run from the otor area of the cortex, between the lenticulus and the claustrum. Detructive lesions, if strictly confined to the lenticulus or the cauda, cause no pecial symptoms.

But cases of what has been called pseudo-bulbar paralysis have sometimes een associated with bilateral lesions of the *lenticulus;* and Aronsohn, ale White, Ott, Nothnagel, and others have found both experimental and linical evidence that the corpus striatum, particularly the *cauda,* contains hermotoxic centres.

The *thalamus* is seldom the seat of disease, and the symptoms produced re not characteristic. In the case of a boy in Guy's Hospital, which was ublished in the 'Pathological Transactions' for 1884, a tumour occupied he right thalamus. There was headache and double optic neuritis, with tosis and slight motor paralysis of the opposite side affecting the arm ore than the leg; no marked anæsthesia, if any, and no blindness or trabismus. The clonic spasms known as "post-hemiplegic chorea" are aid to be more frequent after lesions of the thalamus.

Lesions of the back of the thalamus have caused hemiopia, but there as probably pressure exerted on the optic tracts or the corpora quadriemina. In the anterior part of the thalamus, islands of degeneration have een found by Brissaud in cases of cerebro-spinal sclerosis associated with ental symptoms, and particularly with fits of laughter (p. 713).

The corpora quadrigemina.—We should expect a destructive lesion of ese tubercles to cause crossed hemiopia. Only one or two such cases have en recorded, but Nothnagel believes that a reeling gait followed by uble ophthalmoplegia are the best localising symptoms, and this stateent was remarkably confirmed by the result of a case in a child which as brought before the Clinical Society by Dr Frederick Taylor in 1894 Trans.,' vol. xxvii, p. 18).

The *pituitary body* is not infrequently the seat of a glioma, or other umour. Three cases are noted in Hale White's list (p. 772, *supra*): one f these patients, a man of forty-five, with symptoms of tumour of the base, ad a secondary growth in the lung. Another was a cystic tumour. umours of the hypophysis have also been recorded in which the special ymptoms were due to pressure on the third and sixth nerves in their course o the orbit. The remarkable connection between hypertrophy of this organ nd the condition known as acromegaly has been referred to already (p. 548).

The *pinea* or conarium may contain a psammoma or sarcoma. Dr Heinich Reinhold collected four or five cases in a monograph ('Tumor der irbeldrüse,' 1886). In his patient it was a glio-sarcoma.

Corpus callosum.—The late Dr. Bristowe's interesting chapter on the ubject of diagnosis of lesions of the corpus callosum ('Brain,' vol. vii) as based on four cases, in all of which a tumour of the great transverse mmissure was found after death, and in all of which there was little no optic neuritis, headache, or vomiting; but gradually increasing hemiegia with slight paresis of the opposite side, but without spasms, drowsiess and mental obfuscation, ending in coma. This experience has been nfirmed by Bruns (1886) and since by Grieffe (1892), but has, unfortuately, not been borne out by other observers.*

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* See Dr W. B. Ransome's paper in 'Brain,' 1895, and Dr Ferrier's account in 'Allbutt's stem,' vol. vii, pp. 325–7.

General diagnosis of cerebral tumours.—To the student—who perhaps knows the difficulty of distinguishing between tumours and other surgical affections of parts that can be seen and handled—nothing is more striking than the confidence with which the physician can sometimes assert the existence of a tumour in the cranial cavity.

Cases do, indeed, occur which are only cleared up in the *post-mortem* room. A man of whom little is known, or who has hitherto shown no marked symptoms of cerebral disease, may die in a succession of fits, or in coma of a few hours' duration, and it may be difficult to decide between tumour, cerebral hæmorrhage, thrombosis of an artery, and uræmia; or, if he lives for two or three weeks, between tumour, abscess, and meningitis.

But, as a rule, the illness caused by a tumour in the brain begins gradually, and goes on for several months. In cases at Guy's Hospital the duration of well-marked symptoms has generally been from three to nine months, sometimes longer, and once four years. Jackson has recorded the case of a woman who had optic neuritis and staggered in walking in 1865, and who did not die till the summer of 1872, after she had become insane—there was a growth springing from the base of the skull. It may be affirmed that when the cerebral symptoms described in the present chapter have lasted for more than five or six weeks, they are due to a tumour. Abscess is secondary to injury or some source of suppuration, and rarely lasts long; while the symptoms of embolism and hæmorrhage are very different.

There is already some help to be derived from the "physical signs" obtained by percussion of the skull, and probably we have more to expect. For many years local tenderness on percussion has been recognised as a help to the localisation of a cortical tumour, or a superficial abscess. The pain is probably due to local meningitis, as in most cases of syphilitic disease of the dura mater and external pericranium.

As a localising method, percussion has been applied by Macewen, of Glasgow, who finds that a tympanitic as distinct from an "osteal" percussion note, or a *bruit de pot fêlé,* may be a useful sign of the presence of a cerebral tumour if it is near the surface.

In spite, however, of all that has gone before, it must be remembered that the symptoms of a cerebral tumour are sometimes so vague that they have been mistaken for *hysteria.* The writer remembers a young woman in Mary Ward who had every symptom of hysteria, and who no doubt was highly hysterical; but Dr Wilks, whose patient she was, always asserted that there was more than hysteria; and one morning, to the astonishment of nurses and students, she was found dead in her bed, and at the autopsy we found a cerebral tumour. Another mistake was once made in the case of a plumber who died in Guy's Hospital with epileptiform fits, which we believed to be the effects of lead-poisoning; but a spindle-cell sarcoma of the size of a marble was found in the right hemisphere, with extensive yellow softening around it.

In such cases the routine use of the ophthalmoscope is a great safeguard. At the same time it is not pathognomonic. When neuritis, or choking of the disc, affects one eye only, it is probable that the cause lies in the orbit rather than within the skull. Mr. Lawson brought before the Clinical Society in 1876 a case in which a hydatid cyst pressed upon the nerve behind the eye, and so affected the disc on that side. Again, unilateral neuritis may be due to pressure on the optic nerve between the chiasma and the optic foramen; a gumma at the base seemed to have acted in this way in a case recorded by Hulke in the 'Ophthalmic Hospital Reports.' Jackson has met with two cases in which the ophthalmoscope revealed an affection of one eye, dependent upon the presence of a tumour in the opposite cerebral hemisphere; and in a patient of the writer's there was much more severe optic neuritis on the same side as a cerebral abscess than on the other. But it is a rule to which there are few exceptions that both optic discs suffer together.

Bilateral optic neuritis alone is not a certain sign of tumour. In a series of unselected cases, recorded by Hulke in the 'Ophthalmic Hospital Reports,' there were several in which optic neuritis, terminating in atrophy, was attributed to such vague causes as child-bearing, lactation, leucorrhœa, sexual excesses, or to an antecedent attack of fever.

Dr Hughlings Jackson, who believes that double optic neuritis is almost certain evidence of what he terms "coarse disease" within the cranium, admits that he has himself met with a few instances in which, after death, no such disease could be found. The following is one of them:

A woman, aged thirty-four, had for about a year been subject to attacks of severe headache accompanied with vomiting; and for three months she had been blind. Her illness began with vertigo and momentary unconsciousness, after which she had headache for four days. At another time the pain lasted for three weeks. She was admitted on December 19th, 1874. On January 6th, 1875, she had an attack of pain so intense as to make her toss her head from side to side, holding it in her hands, and crying, "Oh, my head!" She retched and vomited frequently. Both optic discs were greatly swollen, and the veins in them were dilated and tortuous. After the 10th she sank gradually into coma, and on the 12th she died by failure of the respiration. A tumour or some similar disease was confidently anticipated, but Dr Sutton, who made the autopsy, found only certain microscopical changes in the cortex.

Compare with this case one brought before the Clinical Society in 1876 by Dr Goodhart and Mr Higgens. A girl, aged twenty-one, was attacked with intense headache and vomiting on December 21st, 1874, when Dr Jackson's patient had just entered the London Hospital. Some months previously she had been stunned by a severe blow from a stone on her right temple. On the 24th there was well-marked double optic neuritis, her pulse was irregular and only 52 in the minute, and she had no fever. Afterwards she had paralysis of both sixth nerves, suffocative attacks in which she could hardly breathe, constipation, transient hemiplegia, delirium, and an affection of the speech, so that her mother could not understand her. But between the 6th and 14th January, 1875, all her symptoms began rapidly to subside, and before long her impaired sight seemed to be the only thing that troubled her, except that she was unable to take solid food without vomiting.

In practice, we may remember that double optic neuritis most often indicates a tumour, less commonly meningitis or hydrocephalus or tabes, rarely or never cerebral embolism, thrombosis, or hæmorrhage. It may occur in Bright's disease or after fever. Single optic neuritis rarely points to a cerebral lesion. Acute optic neuritis is often caused by a chronic lesion, as a tumour. Primary optic atrophy is not so often a symptom of local cerebral lesions as of more general diseases, such as tabes, general paralysis, and insular sclerosis; but when secondary, atrophy has the same significance as the descending optic neuritis, of which it is the result.

Pathological diagnosis.—The distinction between the different kinds of tumours of the brain depends chiefly on our knowledge of the most frequent, and therefore the most likely lesions in a given case. Again, if we are able to localise the lesion, we may then form a more or less probable conjecture as to its nature. In a child there is a strong presumption that a cerebral tumour is tuberculous. The older the patient, the greater the chance that the lesion is some other form of tumour; and above the age of forty tubercle is unlikely. If localising symptoms point to the cerebellum the diagnosis of a tuberculous growth is more likely to be true, and so also if symptoms of tubercle in other organs are present, but (excepting tuberculous meningitis) this is seldom the case. Glioma is of very slow growth, often multiple, and liable to secondary hæmorrhage with apoplectic symptoms. Malignant disease is rapid in its course, and sometimes softens or actually perforates the skull.

Glioma and secondary malignant tumours are often multiple, and the presence of a primary growth elsewhere is apt to be overlooked. Thus the fact of tuberculous ulcers, or joints, or glands has to be looked for, and a patient may never mention a tumour in the breast, or that a testicle was excised some months before. Out of sixteen cases at Guy's Hospital of secondary growths in the brain, the starting-point of the mischief was a sarcoma surrounding the root of one lung in six, and in five of them this was not discovered during life.

The distinction between tubercle, glioma, and sarcoma is of little practical consequence; what is really important is that one should never overlook syphilis. Gummata may be present in the brain although no nodes on the bones can be discovered, nor any indications of past or present orchitis, iritis, or cutaneous eruptions. A history of venereal disease must not be expected. One must notice a muddy complexion, and search for pigmented scars, bullet glands in the neck or groin, cicatrices in the tongue or fauces, enlarged epididymis, and periosteal nodes. A gumma, like a tubercle, often sets up secondary meningitis of the base, and hence is apt to cause ptosis, mydriasis, and squint.

We should always think of syphilis as a probable cause of obscure nervous symptoms when they cannot all be referred to a single lesion. Jackson long ago insisted on this principle, and two of Buzzard's patients had paralysis of one arm and of both legs at the same time from a syphilitic affection of the cord as well as the brain.

Prognosis.—All that we know of the progress of cases of cerebral tumour would tend to show that they are inevitably fatal. The following cases of apparent recovery are quite exceptional.

A boy, aged fourteen, was under Dr Fagge's care in Guy's Hospital in 1867. About two years previously he had been taken ill with "pain in the back of the head, loss of sight, and fits in which he used to clench his hands." On May 1st, when he had been in my ward for three weeks, it is noted that "he lies apparently unconscious of everything, and cannot be roused. His head is constantly thrown backwards; and when he is touched there is a sort of opisthotonos. He is completely amaurotic; his pupils are equal and slightly dilated." On May 4th the report is, "He is slightly more conscious, he recognises his mother, and will raise his hand into the air when told to do so. His evacuations are passed involuntarily." During the next three months he slowly wasted away, until he was reduced to a mere skeleton; the only sign of intelligence was in lifting his head. But one day, in going round the ward, I spoke to him; and to the astonishment of every one he slowly articulated a few words in reply. From that moment he began to improve. He took food well, regained flesh, talked more and more every day, got up, walked about the ward, and at last was discharged perfectly well except that he was blind. Afterwards he attended among my out-patients, and complained of paroxysmal headache and of epileptiform fits. He was readmitted and died. The notes of the autopsy have unfortunately been mislaid; but I recollect that there was an irregular calcareous mass, of about the size of a marble, embedded in the floor of the third ventricle.

The present writer had a patient, a little girl of five or six years old, in Guy's Hospital with symptoms of a tumour of the brain. She recovered, although with amaurosis and double optic atrophy; and four or five years later came in again with enteric fever, of which she recovered. She was still blind then, with optic atrophy, but with no other symptom of cerebral disease.

The following case the writer is inclined to regard as an error of diagnosis rather than one of recovery from a cerebral tumour. He was once asked to see a patient

with Mr Rand, of Lordship Lane. She was a young lady from the west of England, remarkably healthy in both body and mind. While on a visit she was attacked with most severe and continual headache, vomiting, giddiness, and inability to walk; loss of flesh, and double optic neuritis was present. There was no trace of tubercle in herself or her family, and no sign, as well as the strongest unlikelihood, of lues inherited or acquired. The diagnosis of a cerebral tumour, with the gravest prognosis, seemed inevitable; but full doses of potassium iodide were prescribed, with purgation, and after about a fortnight improvement began, which ended in the patient returning home fully recovered from her dangerous illness.

Treatment.—When the local lesion is ascertained to be a syphilitic gumma, active treatment by mercurial inunction and large doses of potassium iodide is often followed by striking results: consciousness is regained, paralysis is removed, headache subsides, and convulsions cease to recur. But after an interval the symptoms return; perhaps the opposite limbs are now paralysed, or the affection may assume a paraplegic form. A second course of medicine may again be successful, but at last our efforts are too often baffled, and the patient succumbs. Such cases are often prolonged over a period of several years. On the other hand, it sometimes happens that the cure is permanent; and more often that each return is milder than the last, until the disease gradually wears itself out. It is most important that a prolonged course of the perchloride of mercury should follow after symptoms have disappeared in order to prevent a relapse.

There is no doubt that in some cases active treatment by mercury or by iodide has been followed by success even when there is no evidence of syphilis; and therefore it is often advised that in every case we should give a patient "the benefit of the doubt," and treat him without labouring to make out a precise local or pathological diagnosis. But like all treatment independent of diagnosis, this is, in the writer's judgment, not only unscientific but usually unsuccessful. If an abscess is present, such treatment will only waste valuable time; if there is a tuberculous growth, or glioma, or a cyst, it will do harm rather than good; and if we give mercury on the mere chance of a gumma being present, we shall speedily abandon the treatment unless immediate success follows. But if we have made a careful and complete diagnosis of a syphilitic lesion we shall persevere with the only method of curing our patient, increasing the dose or varying the form of our drugs, but steadily persevering in the only path of safety.

When a tumour is not syphilitic, our object is the relief of symptoms. If there be eclampsia, full doses of bromide of potassium should be given. The same remedy will often relieve headaches, or it may be necessary to administer morphia subcutaneously. Reynolds speaks highly of Indian hemp, and also recommends the local application of ice.

The recent achievements of antiseptic surgery, and the power of localisation which we have seen that experiment and observation have now conferred on the physician, have led to the bold attempt to remove a cerebral tumour by trephining and enucleation. Macewen, of Glasgow, is remarkable for the cases he has successfully treated, some of them as early as 1883 (see his address, 'Brit. Med. Journ.,' August 11th, 1888). Horsley published a series of ten cases in which the seat of a cortical lesion was diagnosed and its removal accomplished ('Brit. Med. Journ.,' April 23rd, 1887). In half of these cases the morbid condition was not a new growth, but some inflammatory or degenerative change in the brain or meninges. In one it proved to be a tuberculous tumour, in a second a glioma, in a third a large Pacchionian body, in a fourth a tumour weighing 4½ oz., and in a fifth, again, a tuberculous tumour. In the last of these cases the operation was fatal, in the second the glioma returned and proved fatal six months afterwards; in the three others the patient recovered from the operation, the convulsions were rendered less frequent, or the pain was removed.

It must, however, be confessed that only a small number of cerebral tumours would be amenable to surgical treatment even if we could always accurately determine their situation. Many are too deeply seated, others are multiple, others not sufficiently circumscribed, others of malignant nature, and others only come under notice too late for treatment. The syphilitic growths, moreover, may be better cured by drugs.

A hundred cases from the records of Guy's Hospital were reviewed, with reference to the possibility of operation, by Dr Hale White ('Guy's Hosp. Rep.,' vol. xliii, p. 117). He concluded that not more than three tuberculous tumours, not more than four or five gliomata, and only one sarcoma, two cysts, one myxoma, and two tumours of doubtful origin—in all ten certainly, and four more probably, of the 100—were removable by operation, provided that their exact seat had been previously ascertained.

Cerebral Abscess.—This disease, though pathologically so different from a cerebral tumour, resembles it in its clinical characters, and hence is for practical purposes treated of here. It is not a common malady, but is very dangerous, and affords a most instructive example of the unforeseen practical benefits which follow from purely scientific investigation.

Ætiology.—The important fact was established by Gull* that this affection is never primary or idiopathic; it is due either to general pyæmia or to injury or disease of the skull involving local purulent infection.

Its most frequent origin is from *diseases of the ear*, especially suppurative inflammation of the tympanum. Otorrhœa, if prolonged, always involves the risk of extension of mischief through the bone to the internal surface of the skull. Hence no one with a purulent discharge from the ear should be accepted for life assurance. In the 'Medical Times and Gazette' for 1863 a case is recorded of a patient who for several years had a discharge from the ear, but who lived to the age of sixty-six, and then died of a cerebral abscess; and it often happens that an aural affection which had been present from early childhood kills a grown-up man or woman. In many instances the several stages of the morbid process are plainly traceable after death. The tympanic cavity is found to be bare and carious, or necrotic; the dura mater over its roof is raised from the bone by pus, or it is softened and sloughing; the pia mater is adherent at this point, and close to it an abscess is found in the temporo-sphenoidal lobe. Or the caries may pass from the mastoid sinuses or the petrous bone to the posterior fossa of the basis cranii, and form an abscess in the corresponding half of the cerebellum. Among eighteen successive cases at Guy's Hospital there were twelve in which the temporo-sphenoidal lobe of the cerebrum was the seat of the abscess, three in which it lay in the cerebellum, two in the centrum ovale, and one in the pons.

Toynbee believed that abscess of the cerebrum is the most common result of caries of the tympanum, abscess of the cerebellum of phlebitis of the lateral sinus, and abscess of the bulb of caries of the labyrinth. The ird situation is too rare to be taken into account; but subsequent writers nfirm this connection between disease of the middle ear and abscess of e cerebrum, and between the disease of the mastoid sinuses and abscess the cerebellum.

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* In his papers on cerebral abscess in the 'Guy's Reports' for 1857, 3rd series, vol. iii, p. 251; and in 'Reynolds' System,' vol. ii, 1872.

According to Huguenin and Meyer, the right side of the encephalon is uch more apt than the left to be affected with abscess from disease of the r; and, among twenty-four cases collected by Gull and Sutton, the right le was affected in no less than eighteen. But it is curious that of the ghteen cases that have since occurred at Guy's Hospital, in nine the scess was on the left side, and in nine on the right.

There is more than one route by which septic infection may reach e brain from the ear. It may pass along the bony canals which transmit e superficial petrosal and other veins, or through the spaces in the diploë ich convey vessels from the tympanum to the dura mater. In many stances there is a tract of healthy cerebral substance between the wall the abscess and the petrous bone, showing that the pyogenic micrococci ve not been conveyed by direct continuity. The suppression of the scharge from the external meatus and a sudden increase of pain in the r are bad indications, for they denote swelling of the mucous membrane d increased tension in the tympanum. But in some cases there is no in at all: there may even be no otorrhœa, for the secretion may be ained behind a perfect membrana tympani.

Another, but a far less frequent cause of abscess of the brain, is chronic *ease of the nose.* Two such instances were recorded by Gull in the uy's Hospital Reports' for 1857. Each patient had suffered from a charge from the nostrils; in one case the abscess was in the middle lobe, the other in the anterior lobe. Other writers have given cases in which *nasal polypus* was the starting-point of the mischief. Dr Fagge once de an autopsy in which an ulcerating *epithelioma* of the lip and cheek tended to the base of the skull along the third division of the fifth rve, and set up an abscess in the middle lobe of the brain. Necrosis or *ries of the calvaria* from any cause, if attended with sloughing of the ra mater, may have a like effect. Another source of suppurative ningitis or of cerebral abscess is *carbuncle* or *phlegmon* of the face, rticularly of the upper lip, of which Gull reported a case in a boy of teen, and the present writer saw a very similar one in a girl of the me age.

In twenty cases of cerebral abscess, which were not secondary to disease the ear or nose, and not traumatic, there were fifteen in the white sub- ance of the hemispheres, two in the corpus striatum, one in the thalamus, e bulb, and the cerebellum respectively.

General *pyæmia* sometimes leads to the formation of one or more scesses in the brain, as in other parts of the body. In records of exami- tions at Guy's Hospital (during the same period as the eighteen cases ove mentioned of cerebral abscess from disease of the ear) there were nine tances of abscess as a part of septic infection.

One remarkable case was that of a sailor twenty-five years old, who d with pulmonary and cerebral symptoms. There were found two scesses in the liver, probably of dysenteric origin, one in the lung and one the brain, which had burst into the ventricular cavity.

There is a curious group of cases of cerebral abscess, which are secon- ry to *suppurative inflammation* in a remote part of the body, but in which there are no symptoms of pyæmia, and in which no abscesses are found anywhere but in the brain. Gull first showed the ætiology of such cases, although a similar instance had before been recorded by Abercrombie. It is remarkable that the *lung* is generally the seat of the primary lesion which leads to the abscess in the brain. At Guy's Hospital within a few years Dr Fagge observed six cases of this kind; six others were given by Gull and Sutton in the second volume of Reynolds' 'System of Medicine,' and others were soon after recorded in Germany and elsewhere by Biermer, Huguenin, and Meyer. Apparently the cerebral inflammation is set up by a portion of thrombus washed out of a pulmonary vein, and carried to the brain in the blood; and Böttcher is said to have found in the floor of an abscess of the brain, which was secondary to a pulmonary abscess, some pigment which he was able to identify as having come from the lung. The nature of the thoracic disease has varied in different instances. Adding Dr Fagge's cases to those related by Gull and Sutton, we obtain a series of twelve examples of this form of abscess of the brain. Among them there are three in which the primary affection was empyema (cf. 'Path. Trans.' vol. xxviii, p. 4), two of phthisis, two of pneumonia, and one of bronchitis; in each of the remaining four it seems to have been pulmonary cirrhosis (chronic interstitial pneumonia), with dilatation of the bronchial tubes, or a sloughing cavity in the indurated tissue. Huguenin found bronchiectasis with stagnant putrid secretion to be the most frequent pulmonary lesion which gives rise to cerebral abscess. It is important to note that the mischief in the lung may be altogether latent: in one of the above cases Moxon almost despaired of finding a primary lesion, until at last he discovered that the mucous membrane of the right bronchus was ulcerated, with its cartilages exposed and necrosed.

We must remember that general pyæmia may have an internal source: as septic endocarditis, dysenteric ulceration, or acute osteomyelitis. Such sources of infection may lead to abscess of the brain with or without meningitis. Dr John Ogle once met with a cerebral abscess, secondary to suppuration of the appendix cæci.

Another cause of intra-cranial suppuration is direct *injury to the head*, as from a fall or blow. Generally the skull is fractured, and serious symptoms are present from the first. Such cases come under the care of the surgeon, and do not require further mention here; but occasionally the fact that the head has been injured might be overlooked unless inquired for.

There appears to be scarcely an authentic case in which a cerebral abscess is primary and spontaneous.*

Of 100 cases collected by Mr Barker ('Lancet,' June 11th, 1887), 29 were due to disease of the ear, 27 were traumatic, 20 were associated with suppuration in the lungs or liver, and 7 were due to general pyæmia.

In his 'Gulstonian Lectures' for 1890, Dr Pitt recorded 56 cases of cerebral abscess from the records of Guy's Hospital, and of these 18 were due to disease of the ear, 1 to disease of the nose, 10 to injury of the skull, and 3 more to cranial periostitis or caries; 3 to invasion of tumours of the skull; 8 to primary disease of the lung, 9 to general pyæmia, and in 4 no certain origin could be ascertained.

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* Among seventy-six cases of cerebral abscess from all causes collected by Gull and Sutton for Reynolds' 'System of Medicine' there are a few in which no cause was found; but the only one of which it can be said that the autopsy was complete is the last of the series, that of a man who died in Guy's Hospital in 1863, and whose body was examined by Dr Wilks. He was not known to have injured his skull, but he had led an irregular life, and might easily have forgotten such an injury.

Cerebral abscess, consecutive to disease of the ear, is about equally frequent in males and in females; of the cases secondary to chronic pulmonary affections the larger proportion seem to occur in men.

The great majority of cases, from whatever cause, are met with in persons between fifteen and thirty years old. Of Dr Pitt's cases, only 4 of non-traumatic origin occurred in children under ten; and only 9 in patients above forty.

Anatomy.—Abscesses of the brain which result from injury are almost always solitary, as are those which are secondary to caries of the ear or nose. Out of twenty-seven cases due to aural disease, collected by Gull and Sutton, there are only two in which more than one abscess was present; in one the cerebellum contained three abscesses, in the other there was one in the cerebellum, as well as one in the cerebrum.

On the other hand, out of fourteen cases of general pyæmia, in which the brain had suffered, in eight the abscesses were multiple, and in most of them there were four, five, or more in different parts. So, again, in seven out of eleven cases consecutive to affections of the lungs, the abscess was multiple. The presence of numerous centres of suppuration may therefore go far towards determining the real origin of an abscess in an autopsy, and this might be of forensic as well as of pathological interest.

In abscesses of recent formation the pus is often greenish and viscid, with an acid reaction; and in those of long standing it may be as thick as mucus, of a bright green colour, and alkaline. It may be odourless, but when arising by extension from necrosis of bone it is horribly fœtid. Mixed with the pus corpuscles is much granular matter and oil-drops; and in very old cases one can hardly recognise any pus-cells; they have undergone disintegration.

The abscess-wall is made up of spindle-cells, which form a layer of fibrous tissue, while the cavity is lined with indifferent (or "embryonic") connective tissue, often opaque and yellow under the microscope, from fatty degeneration.

The abscess caused by disease of the ear or nose may be ill-defined, its wall shreddy, and surrounded by softened cerebral substance; but sometimes, when of long duration, it is enclosed in a thick capsule. In pyæmia the abscesses seem to be very rarely circumscribed; but in one of Moxon's cases, which lasted eighteen months, the limiting membrane was so firm that it could be lifted out of the brain-tissue in which it lay. When the affection is secondary to disease of the chest there is almost always a well-marked capsule.

It is only in cases arising from injury that the commencement of the cerebral suppuration can be fixed with sufficient accuracy to decide as to the length of time required for the production of a capsule. The evidence collected by Lebert and Meyer on this point went to show that by the end of the third week the abscess cavity may be found circumscribed, but that a membranous cyst-wall is not found before the sixth or seventh week, and sometimes not until much later. As Gull observed, this might become a medico-legal question in some cases of a man dying of cerebral abscess after receiving a blow, if an interval had elapsed so that no direct clinical connection could be traced between the supposed cause and the effect. He cites one instance in which the absence of a limiting membrane was taken as proof that the disease could not have been the result of a severe fall eighteen months previously; and another, in which its presence showed that the suppuration in the brain was not due to an attack of smallpox which occurred within the last three or four weeks before death. A cerebral abscess without a capsule may form in seven days.

The abscess, when solitary, may reach a considerable size before death, as large as a hen's egg, and sometimes larger. It is often close to the surface of the hemisphere, and occasionally breaks through beneath the pia mater. It is said that pus from an abscess in the brain has been discharged through the auditory meatus, producing "cerebral otorrhœa;" and certainly an abscess in the frontal lobe set up by caries of the ethmoid bone may break through into the nostrils. In four or five cases of suppuration following caries in the ear, at Guy's Hospital, an abscess opened into the lateral ventricle; and this is by no means infrequent in cases secondary to empyema or bronchiectasis. The pus may be found collected in one of the cornua, into which it has trickled, or the whole of both lateral ventricles may be full of pus, with the ependyma red, thick, and velvety; and the process may spread through the third and fourth ventricles to the subarachnoid spaces of the brain and spinal cord.

Symptoms.—It has been said that abscess of the brain may be absolutely without symptoms, so that it is accidentally found after the death of the patient from some other cause. This is more than doubtful; but it is quite true that symptoms are more variable than in other cases of organic disease of the brain. Moreover most patients show symptoms for a much shorter period than that during which the abscess must have been forming; in other words, the disease is often latent during part of its course. This latent period may be only a week or a fortnight, but occasionally may last for twelve weeks or more. In cases of general pyæmia the symptoms of cerebral abscess may be masked under the delirium and stupor of septicæmia.

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Huguenin found that about one fifth of his collected cases of cerebral abscess are situated in the frontal lobe, one fifth in the temporo-sphenoidal, one eighth in the occipital, a very few in the parietal, fewer still in the pons or bulb, and most of the remainder in the cerebellum.

In Dr. Pitt's 52 cases from Guy's Hospital, abscesses from all causes were found 18 times in the frontal lobes, 14 in the temporo-sphenoidal, 6 in the occipital, 5 in the parietal, 8 in the cerebellum, and once in the pons.

Mode of death.—The condition above described may last for several weeks, but sooner or later, and sometimes without any warning, more acute and obviously dangerous symptoms appear. Violent delirium with intense headache may set in, followed by stupor and coma; or a severe epileptiform fit may lead to the same result.

In 1876 a girl, aged eighteen, was admitted into the Clinical Ward one afternoon at five o'clock. She had for six days been suffering from sickness and diarrhœa, with severe headache, so that she was sent in as a case of typhoid fever. She then spoke rationally and answered questions, but seemed strange in her manner. At eight o'clock the same evening she suddenly made a great noise; the house physician was hastily summoned, and found her partially insensible, but capable of being roused so far as to say that she was going to die. She seemed to have loss of power in her left arm and leg. An hour later she ceased to breathe. Artificial respiration was kept up, and the heart continued for some little time to beat rapidly, but it soon slackened, and in ten minutes she was dead. We found four or five abscesses in the right hemisphere.—C. H. F.

The prolongation of the heart's action after breathing had ceased, which was noticed in this case, was recorded by Dr Fagge in other cases of organic disease of the brain ('Guy's Hosp. Reports,' 3rd series, 1879, vol. xxiv, p. 346). His observations have been confirmed by cases related by Prof. Macewen ('Pyogenic Infective Diseases of Brain and Cord,' 1893), by Mr Horsley ('Quart. Med. Journ.,' July, 1894, and 'Trans. Internl. Med. Congr. of Moscow,' 1897), and by Sir Dyce Duckworth ('Edin. Med. Journal,' February, 1898), who reports four cases of death from disease of the brain (three of them abscess) in which the breathing ceased before the pulse; in one of these artificial respiration was kept up for three hours before death, in another for four and three-quarters, and in the third for five hours.

The final stage of a cerebral abscess is seldom of long duration; but it may last for a week, and in exceptional cases longer still, during which time the patient is alternately delirious and in a state of stupor, and passes his evacuations under him. Sometimes there is a transient recovery from such symptoms, followed by their return, and by speedy death.

Diagnosis.—That the recognition of abscess of the brain is sometimes difficult is apparent from what precedes. In cases of supposed ague, or hysteria, of enteric fever, and of neuralgia, it is needful to bear in mind that cerebral abscess is possible. Between abscess and other organic diseases of the brain, particularly tumour and meningitis, there is no positive criterion. Cases of tumour of the brain run a more gradual course than even the most chronic of cerebral abscess, and optic neuritis is more constant. Cases of acute meningitis, particularly septic leptomeningitis following otorrhœa or injury, are comparatively seldom accompanied by hemiparesis, and often by ocular palsy; they are more often marked by high fever, and are never prolonged, as are some cases of abscess.

In practice, however, we recognise abscess of the brain not so much by its symptoms as by our pathological knowledge of the causes to which it is commonly due. Since it seldom, if ever, arises spontaneously, the chief difficulty in diagnosis is the fact that both the patient and his friends often deny the existence of its several causes—previous injuries to the head, otorrhœa, deafness, rhinorrhœa, bronchorrhœa, and other sources of pyæmia. It must, however, be remembered that the same causes which lead to abscess of the brain, may also lead to other forms of cerebral disease. Thus injury is not infrequently followed by meningeal apoplexy or by suppurative meningitis. Chronic phthisis is sometimes accompanied by solitary tubercle of the brain, and very often by tubercular meningitis, which is itself an insidious malady, and attended with the most varied symptoms. Lastly, disease of the ear may either be the starting-point of general meningitis, or may cause thrombosis of the lateral sinus.

Treatment.—Since the prognosis of cerebral abscess is fatal unless the pus is evacuated, the only possible treatment is surgical.

Hilton used to quote in his lectures a case in which Dupuytren trephined the skull, expecting to find pus under the bone; he then incised the dura mater, and finally thrust a bistoury into the brain, gave exit to a quantity of pus, and cured the patient. Similar cases have occasionally been recorded since. In one recorded in the 'American Journal of Medical Sciences' for January, 1850, Dr Detmold, after successive minor operations, which prolonged life for several weeks, opened the lateral ventricle. The case was one of traumatic abscess of the frontal lobe. In 'Ziemssen's Cyclopædia' Huguenin mentions twenty-six cases of supposed success in opening abscesses of the brain. The operation, however, was a desperate one before the methods of antiseptic surgery were adopted.

Among the earlier cases of successful operation for cerebral abscess may be mentioned those of Dr Truckenbrodt in 1886, of Mr Barker (1886 and 1888), Mr MacEwen (1887, who reported twelve cases), Mr Horsley (1888, who reported eleven), Prof. Bergmann (who reported four cases from Berlin), Sir Wm. Stokes (from Dublin), and many from the United States.

In a patient under the writer's care in Guy's Hospital in July, 1879, the symptoms and previous history pointed strongly to an abscess in the anterior parietal region of the left side. Mr Lucas trephined, opened the dura mater, and incised the brain, but without result. No harm was done, and the symptoms of pressure were somewhat relieved; but when death occurred a few days later we found a focus of red softening with infiltrated pus in the corona radiata, close to the part operated on.

In March, 1890, a girl of about twenty was admitted into Mary Ward with otorrhœa, severe headache, vomiting, low temperature, and acute optic neuritis, particularly on the side of the diseased ear. Mr Jacobson trephined the skull and opened an abscess in the temporo-sphenoidal lobe, which, after a second operation, ceased to discharge. The symptoms disappeared and the patient recovered.

At the present time, when an abscess has been recognised, the skull is trephined, and the pus, as a rule, is successfully evacuated.

With improved means of accurate localisation and advancing surgical experience, the number of cases of cerebral abscess successfully treated by operation increases every year.

was taken as proof that the disease could not have been the result of a severe fall eighteen months previously; and another, in which its presence showed that the suppuration in the brain was not due to an attack of smallpox which occurred within the last three or four weeks before death. A cerebral abscess without a capsule may form in seven days.

The abscess, when solitary, may reach a considerable size before death, as large as a hen's egg, and sometimes larger. It is often close to the surface of the hemisphere, and occasionally breaks through beneath the pia mater. It is said that pus from an abscess in the brain has been discharged through the auditory meatus, producing "cerebral otorrhœa;" and certainly an abscess in the frontal lobe set up by caries of the ethmoid bone may break through into the nostrils. In four or five cases of suppuration following caries in the ear, at Guy's Hospital, an abscess opened into the lateral ventricle; and this is by no means infrequent in cases secondary to empyema or bronchiectasis. The pus may be found collected in one of the cornua, into which it has trickled, or the whole of both lateral ventricles may be full of pus, with the ependyma red, thick, and velvety; and the process may spread through the third and fourth ventricles to the subarachnoid spaces of the brain and spinal cord.

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may be raised to 1010; it becomes more or less distinctly opaque on boiling, and shows leucocytes under the microscope. The ependyma is often granular, and the adjacent parts of the brain softened. The fornix and septum lucidum may be almost diffluent, and even the corpus striatum and thalamus may fall into a shapeless pulp as soon as the brain is removed from the skull. This "white softening" appears not to be due to inflammation, for no exudation cells are discoverable; nor does it seem to be mere œdema, the result of obstruction to return of blood from the choroid plexus by the veins of Galen, for the effusion into the ventricles is not pure lymph, as in chronic hydrocephalus. It is certainly not a mere result of *post-mortem* maceration, and may be most probably regarded as a passive necrotic condition, like that of white softening from arterial thrombosis. The seat of obstruction is no doubt in the arterioles and capillaries of the choroid plexus, which are compressed by minute tubercles and fibrin in the lymph-sheaths of the arteries.

More or less numerous tubercles are to be found in the affected pia mater. They are most easily recognised in the Sylvian fissures or in the folds of pia dipping into the sulci, where they appear as minute grey dots, adherent to the smaller arteries or to filaments of connective tissue. When abundant they may become fused together, so as to ensheathe an artery. Some are generally found caseating, and their opaque yellowish colour makes them more easily recognised. If the tubercles are present in large numbers, they may be seen thickly scattered as milk-white spots beneath the visceral arachnoid, especially on the under surface of the cerebral lobes. They may also appear as minute transparent granules upon the inner aspect of the dura mater lining the fossæ of the skull. Within the ventricles they occur only in the choroid plexuses. They sometimes grow from the processes of the pia mater which pass down between convolutions, appearing as whitish yellow streaks along the small arterial branches in the cortex. Dr Gee mentions a case in which the capillary vessels throughout one hemisphere were studded with miliary tubercles; and these remained after the softened cerebral substance had been all washed away.

As a rule both hemispheres are equally affected, but Dr Fagge in one instance found a hemisphere affected on its convexity with tuberculous meningitis, while the left side of the brain and its base seemed to be entirely free. Huguenin described two similar cases limited to the territory of a Sylvian artery; and Dr Pitt records others ('Brit. Med. Journ.' 1890, i, p. 772).

The meninges covering the bulb and cord are often affected as well as those of the brain, and this explains some of the spinal as distinct from the cerebral symptoms of the disease. In the vertebral canal tubercles are found both on the smooth side of the spinal dura and on that of the visceral arachnoid.

In the other organs of the body, tuberculous lesions are, perhaps, always to be found—most often caseous tubercle in the lymph-glands of the neck, thorax, or abdomen, or in the vertebræ, petrosal bone, the hip, or other joints. Miliary tubercles are present in more than half the cases in the membranes of the cord, almost as often in the spleen, and very frequently in the liver, kidneys, lungs, peritoneum, pleura, and pericardium.

Pathology.—Tuberculous meningitis very seldom occurs as the single result of tuberculous infection. In almost every instance it is either secondary to chronic local tubercle, or is only part of a general acute tuberculosis. Often it is both consecutive to a local chronic lesion, and also associated with the simultaneous development of recent tubercles throughout the body.

Among 65 cases collected by Dr Fagge in patients under twenty, there were 13 in which the cerebral affection was secondary to disease of the hip-joint or spine, or some other malady capable of recognition during life; while among 59 above twenty, there were 28 such cases, mostly pulmonary phthisis. In 86 of the total 124 cases there were miliary tubercles in the viscera or in the serous membranes. In only 9 or 10 cases is it distinctly stated that recent tubercles were present in the pia mater and nowhere else.

An interesting question concerns the relation of the tubercles to the lymph and other associated products of inflammation. The opinion of Rilliet and of Huguenin is that the miliary granulations are first developed, and that the pia mater tolerates their presence for a time, but that they afterwards excite inflammatory reaction. But, as Wilks and Moxon argued, if such a view were correct, one ought sometimes to discover meningeal tubercles in small number, unmixed with inflammatory products, in those cases in which acute tuberculosis destroys life by invading the lungs or other organs, without cerebral symptoms.

Dr Fagge met with two instances of this kind. One occurred in a woman, aged thirty-two, who was admitted with an abdominal tumour that turned out to be the omentum indurated by tubercle. She was attacked by hemiplegia, coma, ptosis, and delirium, and died in seven days. At the autopsy the brain and its membranes looked healthy, except for a single minute granule on a fold dipping into one of the sulci, and a little filmy material round one Sylvian artery. So slight was this change that if it had been observable on both sides it would have been passed by. But the microscope showed that even the apparently healthy artery of the opposite side, which had been set apart for the sake of comparison, had a distinct growth of tubercle about it; and the one white granule in the pia mater was already caseating in the centre.

The other instance is that of a man, aged fifty, who became suddenly unconscious on May 2nd, 1876, and was brought to the hospital in a state of coma, with stertor and right hemiplegia. He afterwards partially recovered, and on May 5th he was sensible enough to answer questions that were put to him; but in the following night he was attacked with another fit, of which he died in twelve hours. Tubercles were found thickly scattered about the Sylvian arteries and in the adjacent parts of the pia mater, but without any lymph being present; both lungs contained many grey clusters. In this case it is probable that the tubercles had been formed more slowly than usual, for it was stated that a fit had occurred twenty-two days before the man's death, and that afterwards he was always drowsy and stupid. It ought to be added that he had Bright's disease.

This question was once raised in a trial for murder at Cudham, known as "the Penge case," in 1877. Harriet Staunton, the wife of one of the prisoners, had died in a state of neglect and emaciation, which led to suspicion that she had been starved or poisoned. The medical men who made the autopsy gave their opinion that death had been due to deprivation of food, and yet stated that they had discovered in the membranes of the brain what they believed to be tubercles, but attached no importance to them. What were the exact appearances must remain uncertain, for one report spoke of "small patches of rough, millet-seed-like deposit in the meshes of the pia mater," and another of "a small recent patch of tubercular deposit upon the arachnoid membrane on the upper part of one hemisphere, about the size of a fourpenny piece." The husband and three other persons were convicted of murder. But before the time fixed for their execution the leading pathologists of London addressed a memorial to the Home Secretary, and the lives of the prisoners were spared. If there really were tubercles in the meninges, their presence proved that the cause of the woman's death was disease, and not starvation.

The fact is that the discovery of tubercles by the microscope is of crucial importance. Just as in a case of concussion of the brain, the presence of obvious ecchymoses of one or two convolutions (which cannot be regarded as themselves the cause of death) is nevertheless of the highest significance, so the development of even the smallest tubercles in the pia mater is a sign of invisible changes in the cerebral tissues that are incompatible with the maintenance of life. Obviously, meningitis alone cannot produce cerebral symptoms except by affecting the supply of blood to the brain, and this no doubt takes place by pressure from the exudation, causing thrombosis of the minute vessels of the pia which pass directly into the grey substance of the cortex.

There is, however, little doubt that the brain is not only ill-supplied with blood, but also histologically altered; and occasionally obvious lesions are discovered in the brain as well as in the pia. Rindfleisch speaks of the superficial layer of the cortex—patches of which often remain sticking to the pia mater when it is stripped off—as infiltrated with leucocytes, and Huguenin and Bastian have independently described inflammatory products in the white substance of the hemisphere. Indeed, some part of the brain is now and then found in a state of red or yellow softening. Several instances of this have occurred at Guy's Hospital, and others are mentioned by Rindfleisch and Huguenin. Sometimes the softened part has been the island of Reil on one side, sometimes the frontal or temporo-sphenoidal lobe. In one case Dr Goodhart could detect no granule-masses in the softened parts, but "the tissue was very fatty and granular, and the nerve-fibres seemed to have undergone destruction, scarcely any of them being visible."

Ætiology.—The predisposing causes of tuberculous meningitis are those of tuberculous affections in general—want of sunlight, fresh air, and exercise, scanty food, and inherited proclivity. It often attacks in succession children of the same parents at about the same age. The exciting cause is the entrance of Koch's bacilli into the circulation, and in children this is probably by way of the intestinal tract from the milk of tuberculous cows.

Over-study, mental excitement, intoxication, or blows upon the head have each been supposed to give rise to tuberculous meningitis. A policeman in one of our wards attributed his illness to fatigue in attending a review in Hyde Park in June, 1860, but he had before complained of pain in the head. Three children died in Guy's Hospital in whom the same disease was attributed to a blow or fall upon the head. Such "causes" are, there is little doubt, as imaginary as the pretended causes of syphilitic eruptions; but they are worth recording as a warning against accepting coincidences for causes in other diseases.

A possible exciting cause of tuberculous meningitis is an operation on tuberculous bones and joints. That removal of such sources of infection is on the whole beneficial cannot be doubted, but Dr Pitt has observed several cases in which acute tuberculosis of the meninges and other organs was set up about three weeks after excision of a bone affected with "pulpy disease" of the synovial membrane.

The determining cause of cerebral rather than intestinal, articular, or pulmonary tubercle is quite unknown.

Sex and age.—Tuberculous meningitis is much more frequent in males than in females. At Guy's Hospital the proportion has been as eighty to thirty-seven; and it does not seem to have varied very much at different periods of life, although Huguenin says that in children the preponderance of males is much more marked than in adults.

The relative frequency with which persons of different ages are attacked is not yet accurately known. There are no hospitals to which children and adults are brought in numbers corresponding with their ratios to the population as a whole; and until *post-mortem* examinations become far more general than at present, the Registrar-General's 'Reports' will fail to show the liability of grown persons to tuberculous meningitis. It has always been known to be much less common in infants under two years than in older children, and we now see that this is due to their less chance of tuberculous infection.

Of one hundred and twenty-four cases of tuberculous meningitis at Guy's Hospital, 65 occurred under the age of twenty; 59 between twenty-one and sixty.

Dr Gee, in 80 cases, found that 57 occurred under twenty, and 20 between twenty-one and fifty, 2 between fifty and sixty, and 1 at sixty-eight.

Three cases have occurred at Guy's Hospital during twenty years in infants aged six months, ten months, and one year respectively. Of children between two and four years there have been 14 cases; between five and seven and a half, 9 cases; between eight and ten, 12 cases: between eleven and fifteen, 11 cases; and between sixteen and twenty, 16 cases—altogether sixty-five patients under twenty years old. Of adults between one-and-twenty and thirty there have been 31 cases; between thirty-one and forty, 14 cases; between forty-one and fifty, 11 cases; and between fifty-one and fifty-six, 3 cases—altogether fifty-nine patients above twenty years of age.

Among 100 cases in children under twelve, collected by Dr George Still, 3 occurred in children under six months old, 10 between six and twelve months, 35 in the second year, 13 in the third, and the same number in the fourth year, 9 between four and five, 5 between five and six, 4 between six and seven, and again between seven and eight, and only 4 more between eight and twelve.

Clinical course.—The symptoms of tuberculous meningitis are not essentially different in patients at different ages, but as the best writers have based their descriptions mainly upon observations of cases occurring at an early period of life, and as such cases present certain minor clinical peculiarities, we will begin with the disease as it is seen in *children.*

Premonitory stage.—Before any definite cerebral symptoms appear, a general failure of health is often observed, which may last for several weeks. The symptoms of this period are termed "premonitory." In the well-known work of Rilliet and Barthez on the diseases of children an admirable sketch is given, which has afforded materials for all subsequent writers. Probably these symptoms are due in some cases to a scanty early formation of tubercles in the lungs or other organs, which is afterwards followed by a more abundant crop; in others to the slow progress of chronic caseation of the mesenteric or bronchial glands. Foremost among them is emaciation; the limbs waste and lose their roundness, the muscles feel soft, the ribs are too easily felt and seen, and the skin is lax and flabby.

The cheeks often retain their plumpness of outline, so that the loss of flesh is first observed by the nurse, who sees the child undressed; but the face becomes pale, the eyes are dull, and there is a want of animation in the countenance. The appetite is diminished or capricious. The bowels are disordered, being generally constipated, but with intervals of diarrhœa. The child is dull, apathetic, and slow in his movements; he is easily fatigued, and quickly tired of toys. He complains of headache, and wants to lie down. At night he is restless, lying with the eyes half closed, rousing at the slightest noise, and unable to sleep with a candle in the room; he often grinds his teeth, and starts or cries in his dreams. Pyrexia is usually absent.

Premonitory symptoms do not always occur, but Dr Gee says that among twenty-six cases collected by him there were only two in which they were not noticed. Their duration is very variable, sometimes not more than a fortnight, but generally longer, and occasionally much longer. They may subside, and the child's health appear to improve, before the disease breaks out in a characteristic form.

Onset and early symptoms.—In marked contrast to the uncertain length of the prodroma, the subsequent course of the disease is seldom prolonged beyond twenty-one days from the occurrence of the invasion.

The symptom that first excites alarm is most often *vomiting.* Sometimes the child is sick only when it takes food, sometimes it brings up bile. The sickness from an empty stomach generally lasts for two or three days, but it may go on for a week. If it ceases for twenty-four hours it seldom returns. In some cases there is no vomiting for the first day or two, and occasionally it is altogether absent.

The initial symptom next in frequency is a *convulsion,* more or less epileptiform in character. In one instance mentioned by Dr Gee there was general rigidity, which recurred several times; in another the attack took the form of temporary unconsciousness. In a single one of his twenty-five cases there was neither vomiting nor convulsions, but only increase of the headache, drowsiness and wasting of the premonitory stage.

Severe *headache* is now almost invariably present; the child keeps its hands pressed against the forehead, or may go on rubbing the scalp, first in one place, then in another. Trousseau noticed a peculiar inarticulate "hydrocephalic cry," which is sometimes heard from the first, sometimes only towards the end; he describes it as a single, sharp, loud sound, like that of a person exposed to some sudden danger; it may be repeated every hour, or every five minutes, for several days together. We may hear the hydrocephalic cry without hydrocephalus, and many children die of tuberculosis without giving the cry; but the symptom is marked and characteristic enough to deserve mention.

For several days there may be no marked impairment of the intelligence; the child continues to talk rationally, but his answers may be rather slow, and his memory and power of perception somewhat impaired. He is apt to lie in a drowsy state, half asleep, with his eyes staring vacantly, or he may go on talking to himself, repeating some phrase over and over again, or singing and shouting.

The *pupils* are often dilated, sluggish, and very commonly one is larger than the other. Slight strabismus is a frequent early symptom. Trousseau relates a case in which there was transient hemiopia; the child was sitting window when he called out, "Oh, mamma, look at that little boy; as only half a blouse and half a face!"

uch are, as a rule, the symptoms of tuberculous meningitis for perhaps t days after its invasion. During this period there is more or less r, the evening temperature rising perhaps to 102° or 103°, while in the nings it may be 101° or 100°, or not above normal. The pulse may be tle quickened, or natural, or slow; and is easily made more rapid by least excitement. The frequency of respiration is but little altered. tipation of the bowels is almost always present, and the tongue is often, not always, furred.

ater stage.—When about eight days have passed, the condition of the patient undergoes a change, now and then almost sudden, but more gradual. Its most striking feature is loss of consciousness; and the d which follows has been called the stage of cerebral pressure, in dis- ion from the preceding stage of cerebral irritation. The child now es to take notice of anything that goes on in the room. He often lies ne side, curled up with the knees drawn close to the abdomen, and the ls folded over the pudenda. Sometimes the head is drawn backwards, the muscles of the nape of the neck may be felt hard and rigid. Some- s he keeps grinding his teeth. The pupils now become dilated and nsible, and often one or more of the cranial nerves are paralysed: the d, for instance, so that the eyelid drops; the sixth, or more rarely the io dura. There may be loss of power in the limbs, tickling the of both feet may cause only one leg to be drawn up, and the evacua- s are often passed into the bed.

he order in which these symptoms make their appearance is uncertain; e of them may begin during the earlier period of the disease, be- coma sets in. This is particularly the case with certain characteris- changes in the patient's aspect; a frown upon the brows, and deep around the nose and mouth; injection of one cheek, or of both. If countenance is pale, it is liable to flush when the child is disturbed, or n anything is given it to drink. So, also, any part of the body which been pressed upon shows a marked injection of its capillary blood- els. A particular instance of this was made into a leading symptom of rculous meningitis by Trousseau, under the name of the *tache cérébrale.* observed that if one draws one's finger-nail gently over the patient's h, or abdomen, or face, a bright red line is produced, and that this rs from the effect of an equally slight scratch in a healthy person by earing earlier (within thirty seconds), by lasting longer (eight, ten, or en minutes), and by being broader and of a deeper colour.

The *ophthalmoscope* may show ischæmia of the discs, or marked optic ritis, or tubercles in the choroid. Dr Allbutt found some affection of retina in twenty-nine out of thirty-eight cases.

At this period vomiting is generally absent, and the bowels remain tinately constipated; but for some unknown reason the intestines no ger contain the usual quantity of gas; and the belly becomes deeply lowed (*scaphoid*), the rib cartilages, the iliac crests, and the pubic sym- sis appearing prominent.

The *tongue* may now be red and dry, but it often still remains moist. *temperature* seldom rises above 101°; and Dr Gee remarks that it may days together remain at between 96° and 98°. Thus, if the case is anced when the child is first seen, the disease may appear to be apyretic. The *pulse* during this period is generally infrequent, 60 or even 50 per minute; it is still apt to be irregular in time and unequal in force. A similar irregularity and inequality of the *respiration* are frequently present; the child perhaps breathes rapidly three or four times in succession, and then the chest may remain motionless for some little time. Trousseau recorded a case in which the breath was held for as long as fifty-seven seconds. Typical Cheyne-Stokes respiration is sometimes observed.

Final stage.—The "stage of pressure" may continue with but little alteration until it terminates in the patient's death, which is often preceded by a convulsive seizure. But in certain cases the symptoms change during the last twenty-four or forty-eight hours. Thus there may be a brief return of consciousness. Dr West related how a girl, aged seven, who had been in a state of stupor for six days, and profoundly comatose for two days, became conscious, swallowed some drink, spoke sensibly, and said she knew her father; in the course of an hour and a half, however, she became worse again, and a little later she died. The pulse often becomes rapid during the last two or three days; and, as Dr Gee observes, the temperature may steadily rise until it is above 107°. But in some cases the pulse remains infrequent up to the time of death; and the temperature even in the rectum may fall very low. In one case, three days before the fatal termination the thermometer registered 97·8° and 96°; next day the highest temperature was 96·2°, the lowest 93°; the day after they were 82·8° and 82·1° respectively; and on the day of death 80·5° and 79·4°. In other cases the face and limbs are livid and cold, and covered with a clammy sweat, while the thermometer shows that fever is still present. Towards the last a peculiar fœtid earthy smell is often perceptible; and it is strange that Rilliet and Barthez, after quoting this symptom from Whytt, go on to say that they have not themselves recognised it.

Symptoms in adults.—The onset of tuberculous meningitis in adults is seldom preceded by marked prodromal symptoms. It is often secondary to phthisis, and when this is the case the pulmonary affection is rightly regarded as the cause of any general failure of health that may have been noticed. Then, again, few grown-up persons are watched as carefully as children are by their mothers and nurses; and in adult life loss of flesh may be due to so many other causes that tuberculous meningitis is very unlikely to be thought of.*

The invasion is less often marked by vomiting in adults than children; and eclampsia is still less frequent.

But although in adults the disease generally begins gradually and insidiously, yet we have had at Guy's Hospital more than one case in which the sudden occurrence of convulsions has been the precursor of a fatal termination within a day or two, and in which the autopsy has shown that the disease was tubercular meningitis. It is not at all uncommon for death to take place after an illness of twenty-four or forty-eight hours only. In 1868 a man aged thirty-two, a patient of Dr Wilks, died within two days of having been about his business as a draper; on admission he was so restless that he had to be held down in bed, but he quickly became comatose. In another case there was violent delirium two days before death.

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* It is my impression that most adult patients whom I have seen have been fairly well nourished at the time of death. I have no recollection of having ever observed a very marked degree of wasting, unless when it was obviously referable to some co-existing visceral disease; and in the case of Harriet Staunton, already referred to (p. 799), I should have been reluctant to regard the extreme emaciation as dependent upon the presence of meningeal tubercles.—C. H. F.

In some cases of tuberculous meningitis in adults the first symptom is well-marked *local paralysis*, which is seldom seen in children. In 1871 a woman discharged from a surgical ward with advanced phthisis, returned the same day to seek readmission as a medical patient. No symptoms of cerebral disease had been observed, but now there was paralysis of the left facial nerve, which must have developed within a few hours. She was admitted into the Clinical Ward, and was intelligent enough to be able to say that she could not hear the ticking of a watch with the left ear. But she soon became drowsy, then comatose, and at the end of six days she died. At the autopsy no special affection of the facial nerve could be found either in the petrous bone or elsewhere; but sometimes suppuration of the sheath has been found.

Lastly, tuberculous meningitis in adults may have *hemiplegia* for its earliest, and even for its principal, symptom. Three instances of this were recorded by Dr Fagge. One patient was a man aged thirty-three, who was admitted on account of an abdominal tumour, which afterwards proved to be a tuberculous omentum; some days before his death he lost power in the left arm and leg. In the other two patients the right limbs were affected, and the paralysis was accompanied by well-marked aphasia. One of them was a woman, aged twenty-six, who came under Dr Wilks's care in 1867; until the real nature of the affection was revealed at the autopsy there was not the slightest suspicion of its being due to anything but disease of the Sylvian artery. The other, also a woman, aged forty-one, had been attending as an out-patient for phthisis, when she was seized with right hemiplegia and loss of speech; afterwards she became semi-delirious, and her paralysis changed sides. In none of these three cases was any well-marked change found in Broca's convolution or in the adjacent parts of the brain. But in a boy aged nine, in whom right hemiplegia and aphasia were combined with the more ordinary symptoms of tubercular meningitis, there was reported red softening of the left third frontal, and of the inner ends of the two left ascending frontal gyri. Huguenin relates three similar cases in which paralysis of the right limbs and loss of speech were the chief symptoms; in two of them many tubercles were present in the left Sylvian fissure, but none in the opposite one, and in the third case the pia mater in the left fissure was more thickened than anywhere else.

Diagnosis.—This may be very easy or very difficult, either in children or adults. During the first few days of a case which is to last two or three weeks one is often unable to speak positively, but there is seldom much uncertainty when the disease is once fully developed.

All writers lay stress on the importance of distinguishing meningitis from *enteric fever*, and no doubt many cases, which for a week or ten days are supposed to be examples of that disease, afterwards develop such well-marked cerebral symptoms that the diagnosis is forthwith altered to that of tuberculous meningitis. Thus Rilliet and Barthez describe a special form as having a *début typhoïde*. But although to the clinical observer the meningitis masks the other symptoms, yet the pathologist almost always finds it to be only a part of general miliary tuberculosis, to which the early febrile symptoms were due. In our 'Reports' at Guy's Hospital, however, two instances are recorded in which the first diagnosis was that of fever, and in which the tubercles were found in the meninges and nowhere else. Sometimes the opposite error is committed, enteric fever being attended with strabismus and irregularity of pupils, vomiting, and constipation, as well as with headache, delirium, and coma, so as to be taken for meningitis.

The late Mr Stocker, the last resident Apothecary at Guy's Hospital, used to tell the present writer, when a student, that if, when you pulled down the bedclothes to look for spots, the patient lay on his back and took no heed, you were sure to find some—it was "fever;" but if he swore at you, pulled up the sheet again and turned over on his side, it was "brain."

The writer has known a case of convulsions in a child due to the onset of scarlatina which simulated tuberculous meningitis for a time. The *tache cérébrale* affords little help in doubtful cases. The diagnostic value of ophthalmoscopic changes in the optic discs is considerable; but a normal state is no proof of the absence of meningitis. The presence of a tubercle in the choroid is conclusive, but it is not very frequent. The writer had a well-marked case in December, 1896, but the other symptoms rendered it needless for diagnosis.

In all cases of suspected tuberculous meningitis one should carefully search the lungs for indications of acute tuberculosis, and examine the testes, the lymphatic glands, and other organs, for chronic lesions of the same nature. In a patient fifty years of age, who was brought comatose into John Ward in April, 1875, the writer found evidence of phthisis, and this led to a correct diagnosis.

A condition that formerly was often mistaken for tuberculous meningitis is one, not infrequent among young children, which has been rendered classical by the descriptions given of it by Marshall Hall (1825), Abercrombie (1828), and Gooch (1829). The first of these writers called it "*the hydrocephaloid disease;*" Watson gave it the name of "spurious hydrocephalus;" and Gooch stated that he had "invariably found it attributed to and treated as congestion or inflammation of the brain." A case related by Marshall Hall is that of a girl under three years old, to whom sixteen leeches had been applied for an attack of influenza: and he states that all his patients were in a state of exhaustion before they were attacked by the cerebral symptoms; many of them had had protracted diarrhœa after weaning.

The child's aspect is characteristic; it lies on its nurse's lap, unwilling to raise its head, drowsy, or even comatose, with sunken, half-closed eyes, dilated insensible pupils, irregular and sighing respiration. The face is pale, and the skin cold. Depression of the fontanelle is an important symptom of exhaustion in children, although it seems formerly to have escaped notice. The proper treatment is to give ammonia and brandy, but, above all, to see that suitable food is supplied. To prescribe leeches and calomel would probably be fatal. At the present day we are not likely to fall into the error of diagnosis which was frequent seventy years ago.

The writer, nevertheless, once did so, in a case which furnished a good example of this form of disease. A little girl about three years old was, he supposed, suffering from tuberculous meningitis. There were wasting strabismus, a cry which was called hydrocephalic, and a *tache* which was called cerebral. There were also vomiting and ingravescent coma. Diarrhœa was present, but was attributed to tuberculous disease of the bowels. The fontanelle was already closed, and though the temperature was subnormal, this is no uncommon symptom in the latter stages of meningitis. After death, however, the brain and its membranes were found to be perfectly free; there was no tubercle in any organ, and the cause appeared plainly (after the event) to be one of chronic gastro-enteritis in an ill-nourished but not otherwise diseased child. Happily neither calomel nor leeches had been prescribed, on account of the feeble condition of the patient and the presence of diarrhœa.

At Guy's Hospital we have had cases, supposed to be *mania, delirium tremens,* or *epilepsy,* which have turned out to be tuberculous meningitis. Again, a painter came saying that he had "lead colic," and that his bowels had not been open for a fortnight. In the evening after his admission he had convulsions and became insensible, and on the next day he died. There was caseous disease of two of the lower dorsal vertebræ, with a large abscess in front of the spine; and the membranes of the brain and cord showed the characteristic appearance of tuberculous meningitis. The same disease was found after death in two cases of boys admitted into the surgical wards for symptoms supposed to be due to vesical calculi; no cause could be discovered for the irritability of the bladder from which they had been suffering before signs of cerebral mischief appeared. A very similar case occurred in a woman who was taken into a medical ward for vesical symptoms; but here, beside tuberculosis of the pia mater there was a tumour in the spinal cord.

The diagnosis between tuberculous meningitis and other *organic affections of the brain,* particularly abscess and tumour, is often difficult, and sometimes impossible (cf. *supra,* p. 794). Probably not a few cases in England have been recorded as examples of sporadic *cerebro-spinal fever,* which have really been tuberculous; and it is of practical importance to remember that *syphilis* may be the cause of the most varied cerebral symptoms.

In adults, however, the most serious error of all is to mistake tuberculous meningitis for *hysteria.* Many such mistakes have occurred, and sometimes with lamentable results. A woman, aged twenty-five, had long been "odd in her mind and scarcely to be trusted;" she was attending as an out-patient, with hysterical symptoms, when she sought admission to the hospital, and died three days later. There were several yellow tubercles in the brain, as well as the usual appearances of tuberculous meningitis.

Prognosis.—Tuberculous meningitis is one of the most fatal of all diseases. Rilliet recorded a single instance of recovery, in which the child died five and a half years afterwards of a second attack, and at the autopsy the remains of the former disease at the base of the brain were clearly recognised. One similar case is mentioned by Trousseau, and the late Dr Carrington recorded a third and a most conclusive case. But such instances are extremely rare.

It is, however, by no means very rare for recovery to take place in cases in which tubercular meningitis had been diagnosed more or less positively, and for the nature of the disease to remain uncertain, as it must necessarily remain in such cases, unless an accident should lead to an autopsy being made at a future time.

How closely a case which terminates in recovery may resemble tubercular meningitis is illustrated by a clinical history which we owe to the late Dr West. A child aged three years and a half, a member of a phthisical family, was attacked by a disease which ran the ordinary course of acute hydrocephalus, unchecked by the customary treatment. Convulsions took place, coma succeeded them, deglutition was very difficult, the pupils were dilated and almost motionless, the pulse was very feeble and very frequent, and everything portended a speedy death. A younger brother had died a year before of the same disease. Food was still given, as the power of swallowing was not entirely lost, and ammonia and ether were administered, and after a time quinine. For days the child remained unconscious, but at length she began to raise her hands to steady the cup that was put to her lips. Next she recovered her sight; after some weeks she became able to speak; and after many months she began to walk with a tottering step. Three years afterwards, although her intellect was not defective, she still had a vacant smile, and had never regained flesh, nor recovered the look of health; her gait also remained unsteady.

Whatever the real nature of this case may have been, one cannot be wrong in learning from it not to make an absolutely fatal prognosis in what appears to be a case of tubercular meningitis. However confident we may be in our diagnosis, and however threatening may be the symptoms, our opinion should be given with some reserve.

The writer has had two such cases under his own care in which death, after an interval of many months since recovery, has shown by adhesions of the membranes at the base of the brain that there had been basal meningitis. In one of these cases death occurred more than a year after recovery from an illness with all the symptoms of basal meningitis, and a tumour which had been recognised as the cause of the second attack was found to be tuberculous.

The fact, now well established, that not only lupus and tuberculous disease of lymph-glands, joints, and bone is cured every day, but also phthisis and tuberculous peritonitis, should surely forbid an absolute incredulity of the clinical cases of which the diagnoses are not verified by subsequent accident. Nevertheless it is perfectly true that these cases are rare exceptions; enough to justify treatment, but scarcely to alter the almost hopeless prognosis of an individual case.

The *treatment* of tuberculous meningitis will be discussed when the other forms of inflammation of the pia mater have been described.

ACUTE SEPTIC MENINGITIS.—That some cases of idiopathic meningitis in children are not due to tubercle has now been well ascertained. The first group to be distinguished was that of cases of secondary pyæmia due to infection with pyogenic microbes—usually strepto- or staphylococci.

Anatomy.—On turning back the dura mater, the cerebral convolutions are completely hidden by a green purulent exudation. This looks as if it were spread out over the hemisphere in the subdural space; but by scraping the surface one generally discovers that little, if any, of it is really free. Moxon says that even when some of the exudation had appeared to come off upon the edge of the scalpel, he often found only epithelium and detritus on examining it with the microscope. No doubt in some cases there is a considerable quantity of subdural pus, but this is only when the disease started from the skull; and even when meningitis is set up by compound fracture of the skull, caries of the petrous bone, or necrosis of the calvaria, there is only occasionally exudation in the "arachnoid cavity." In the great majority of these cases no subdural pus is found; and whenever it is widely diffused over the hemispheres it is also abundant in the meshes of the pia mater.

The pia may be swollen to many times its normal thickness, appearing either soft, green and gelatinous, or firm, yellow and felt-like, according to the consistence of the exudation. From such a condition there may be every gradation, down to a point at which only slight traces of pus can be discovered along the principal vessels, at the base, in the Sylvian fissure, or the cerebellum.

In some of the more severe cases the convolutions themselves are softened, so that when the pia mater is stripped off it carries with it portions of the cerebral tissue, leaving a ragged surface behind. Leucocytes may be found throughout all the layers of the cortex. The vessels often contain but little blood, in consequence of the increased pressure which precedes death. The ventricles may be either empty or contain a turbid liquid, or sero-pus.

Some of the sinuses in the dura mater are occasionally found plugged with *ante-mortem* clots, even when the meningitis is not secondary to disease of the skull. In a little girl aged three, for example, who died in Guy's Hospital, and in whom both hemispheres were covered with a thick layer of yellow lymph, each lateral sinus was filled with a greenish softening clot.

How often acute meningitis of the brain spreads to the membranes of the cord it is impossible to say, for in most recorded cases the vertebral canal has not been opened; but we have had at Guy's Hospital many cases in which inflammatory products have been found beneath the spinal arachnoid. In none of them was there any reason to believe that the disease was of epidemic origin; in several it was directly caused by fracture of the skull or severe injury to the brain, or arose by extension of mischief from the cranial bones.

Ætiology.—The causes of acute suppurative meningitis are various. Most frequent among them are *injuries to the head.* These generally come under the notice of the surgeon, but the physician must remember that the disease sometimes follows a blow or fall which may not have produced any external bruise, and about which the patient may say nothing.

Some years ago a man was admitted into Guy's Hospital for a fractured thigh, caused by his having fallen into a cellar while drunk. He died at the end of five weeks, having been delirious all the time. He was believed to have delirium tremens; nothing was known of any injury to the head; he had been able to get out of bed and stand upright. At the autopsy it was found that there was general acute meningitis affecting the base as well as the surface; a small part of one parietal bone, over an area an inch in diameter, was of a greenish colour, and its diploë was reddened. Both the periosteum and the dura mater seemed healthy, but it was thought probable that the bone had been injured by the accident.

Another common cause of meningitis is the extension of inflammation from *chronic disease of the skull.* Syphilitic caries or necrosis of the calvaria sometimes kills in that way, and very rapidly.

Many cases of septic meningitis are due to *affections of the ear*, although Dr Pitt has found that meningitis without abscess or thrombosis of a sinus is the least frequent of the results of disease of the ear (1890). A history of a blow often precedes the attack, but the inflammation really starts from a diseased temporal bone.

A woman aged twenty-seven died from meningitis, and no cause could be found until the late Mr Hinton found pus in the labyrinth on one side. To this accordingly the disease was attributed; but it must be remembered that in epidemic cerebro-spinal fever inflammation often spreads to the ears from the membranes, and there seems to be no reason why the same thing should not occur in the simple form of the disease.—C. H. F.

In a case of the writer's ('Path. Trans.,' vol. xxxix, p. 228) a youth of eighteen was struck on the left ear in a fight with another lad. He was giddy and vomited at the time; a little blood ran from the ear, but he showed no further symptoms until the following day, when headache came on, and by evening he was delirious. When brought to the hospital next morning (two days after the injury) he was unconscious, with moderate pyrexia (101° F.), pulse 68, paresis of right arm, and conjugate deviation of eyes to the left. We found the left tympanic membrane destroyed, although there was no otorrhœa present, and the meatus was blocked with granulations. The fundus of both eyes was normal. Two days later there was marked left facial palsy, the temperature had risen to 102·8°, and he was still unconscious. Two days later the temperature was 103°, and he died the following day, just a week after the accident. I thought it probably meningitis, not abscess, and no operation was performed; indeed, the ill nourishment of the lad, and the presence of considerable bronchitis, made me incline to regard the case as more likely tuberculous than traumatic. After death, however, we found purulent meningitis, with a transverse fracture of the skull from the lambdoidal suture of the occipital to the foramen spinosum of the sphenoid bone, running through the necrosed petrosal. There was no tubercle in any of the organs. There was no suppuration of the lateral sinuses, though they were touched by the line of fissure. The epicrisis was chronic, quiescent caries of the petrosal from old suppurative tympanitis, disturbance by a severe blow which caused immediate concussion, and subsequent septic meningitis.

Numerous other local affections may occasionally give rise to septic meningitis. *Local pyæmia* from carbuncle of the face may cause it, as in a case which the writer saw many years ago in a girl about seventeen. In two cases an epithelioma, commencing in the lip, extended with ulceration along the pterygoid region until it passed through the foramen ovale into the interior of the skull. In another, a little girl had necrosis of the upper jaw after measles, and the cavernous sinus was full of a dirty brown fluid. Or the disease may be set up by suppuration of the eyeball, travelling in all probability along the sheath of the optic nerve. Trousseau relates a case in which its starting-point was a chronic affection of the first two cervical vertebræ. Suppurative meningitis occasionally accompanies phlegmonous *erysipelas* of the scalp.

In many of these cases there is phlegmon of the face, and the micrococci have spread inwards, either by the veins or along the sheaths of the nerves of the orbit. More often the meningitis appeared to be part of general *pyæmia;* once pericarditis also was present; once the meningitis was the only evidence of blood-poisoning, but such seemed to be the most probable explanation of its occurrence, as it came on six days after an operation for imperforate anus in a child; once it was associated with an abscess in the lung, and with suppuration in the mediastinal connective tissue and in the substance of one leg, there being, however, no obvious primary lesion.

Endocarditis.—In 1871 a man, already in the hospital for disease of the aortic valves, died after two days' illness of cerebro-spinal meningitis: and the spleen contained infarcts. In 1874 a woman, aged forty-eight, who had been admitted for chronic jaundice caused by biliary calculi, died from septic meningitis consequent upon ulcerative endocarditis of the aortic valves, which was discovered at the autopsy. Since then many such cases have been observed.

There remain a few cases in which meningitis can be traced to none of the causes mentioned, and must be called *idiopathic.* Dr Fagge found in the *post-mortem* records at Guy's Hospital nine cases of this kind. In three of them pus or lymph covered the whole surface of the brain, both hemispheres and base; in three the amount at the base was much less than upon the convexity; and in one the base was free. Once the under surface of the brain and the subarachnoid space of the spinal cord were bathed in pus, while there was but little on the hemispheres. In the two remaining cases the inflammation was confined to the base, as in tuberculous meningitis. In every instance the cause of the disease remained a mystery.

Course.—Acute suppurative meningitis commonly begins suddenly; it has no prodroma. The patient, if an adult, may be seized with a rigor; in children this is less common. Headache is generally present from the first: it may either be referred to the forehead, or affect all parts alike. There may be exacerbations from time to time, in which piercing cries are uttered: the agony appears to be altogether intolerable. Giddiness is frequently an early symptom; the ground seems to give way beneath the feet, and the legs feel so weak that to stand upright is impossible. Vomiting, perhaps, occurs once or oftener. There is an extreme irritability to light and sound. The eyes are brilliant and injected, the face is flushed, the head is hot, the carotids are felt to throb violently. The temperature rises, and may reach 104° by the third day. The pulse is quickened as a rule, but this is not always the case; and as the disease goes on it often becomes slow. Epileptiform convulsions are not uncommon, and sometimes mark the commencement of disease. The back of the neck is often rigid, so that the patient seems to bore his head into the pillow. Sometimes his limbs are stiff. He generally lies coiled up in bed, anxiously avoiding notice, and most unwilling to be disturbed. His mind may for a time be perfectly clear, but afterwards he becomes delirious, and contrary to what Jenner observed in fever, the headache does not go as the delirium appears. In some cases there is violent maniacal excitement from the very first. Sleep is altogether wanting, or very broken and disturbed. The pupils are, as a rule, contracted; the ophthalmoscope may show optic neuritis, but this is less common than in cases of cerebral abscess.

The second stage of the disease is characterised by stupor, which more or less quickly passes into coma. The pupils are sluggish, or even dilated and insensible; they are often unequal in size. There may be loss of power in the limbs of one side with or without a similar affection of the corresponding half of the face. Sometimes the patient squints; but even when the base is involved one can seldom make out a definite paralysis of any of the cranial nerves; they of course remain unaffected when the inflammation is limited to the convexity of the brain. Epileptiform convulsions may return again and again until one of them proves fatal, or again, there may be attacks of spasm confined to certain muscles or to one limb. The temperature generally remains high, ranging from 102° upwards, but the face is now pale, and the extremities may be cold to the touch and bathed in a profuse sweat. Towards the last the evacuations are passed involuntarily.

The duration of septic meningitis is decidedly shorter than that of the tuberculous disease. Those cases in which the whole surface of the brain, including the convexity, is inflamed, scarcely ever last more than a week, and generally terminate within three or four days.

Diagnosis.—Septic inflammation of the meninges may be as difficult to recognise as we have seen tubercular meningitis to be. There may be little or no pain in the head, and thus the disease may be overlooked. When it is impossible to discover any of the recognised causes of meningitis, and when the symptoms are obscure, one may be unable to distinguish it from alcoholism, mania, or epilepsy. On the other hand, if a source of septic infection is discovered, we may diagnose cerebral thrombosis or abscess rather than meningitis.

In 1877 I made an autopsy in the case of a patient of Dr Frederick Taylor's, who had died with what appeared to be clear symptoms of meningitis secondary to disease of the ear, including convergent strabismus, a swollen œdematous condition of both optic discs, delirium, and coma. The only sign of any disease of the encephalon was a blackened state of the arachnoid over a small part of one lobe of the cerebellum, but there was a putrid thrombosis of the lateral sinus and jugular vein, and this had set up numerous pyæmic abscesses in the lungs.

In the 'Med. Times and Gazette' for 1877 will be found a precisely similar case, under Dr Wilson Fox, in which the patient had all her limbs flexed, and suffered from headache, photophobia, and hyperæsthesia of the surface, but in which the brain and its membranes were perfectly healthy. It would therefore appear that pyæmia dependent upon thrombosis of a lateral sinus is capable of simulating meningitis.—C. H. F.

Prognosis.—We have seen that tuberculous meningitis, with a few well-established exceptions, is a fatal disease. So is septic meningitis of whatever origin; but even this condition allows of the possibility of hope.

Two cases were recorded by the late Dr Andrew in the 'Med. Times and Gazette' for 1875. One was that of a youth, aged sixteen, who had had a discharge from the ear, and who became drowsy and heavy, with frontal headache, giddiness, sickness, and blurring and œdema of the optic discs. The other occurred in a girl, aged nine, who had also had ear disease, and who was attacked with pain in the head, vomiting, and delirium, so that she screamed and started in her sleep. Meningitis was diagnosed, but each patient recovered after an illness of some weeks' duration, in the course of which there was distinct evidence of pyæmia, one of them having pneumonia, the other having an abscess in the thigh.

At the present day, when operative treatment is bolder and more successful, we have often good reason to believe that meningitis has already begun from disease of the ear in cases which recover after operation.

BASAL MENINGITIS WITHOUT TUBERCLES.*—When the symptoms and course of an illness have been like those of tubercular meningitis, the membranes at the base of the brain and in the Sylvian fissures are sometimes found after death with well-marked signs of inflammation, but with no evident tubercles. Dr Fagge records six or seven cases of this kind in Guy's Hospital. In all of them, with one exception, the lungs (and often other viscera as well) contained miliary tubercles; while in the one exceptional case the bronchial glands were caseous. Here the pulmonary lesion was conclusive as to the real nature of the disease.

There is, however, a form of acute basal meningitis in which no tubercles are to be found in any part of the body. Huguenin relates the case of a female child, aged eleven months, who died on the fourth of an attack of measles, attended with convulsions and other cerebral symptoms. Flattening of the convolutions, injection of the choroid plexuses, distension of the ventricles, and softening of the central parts were the only obvious morbid appearances; leucocytes were abundantly present in the pia mater.

A similar case occurred at Guy's Hospital in 1859, in the practice of the late Dr G. H. Barlow. A boy, aged nine and a half years, died after an illness of twelve days' duration, which began with intense headache and ran its course with convulsions, grinding of the teeth, strabismus, and coma. That the disease was tuberculous meningitis was doubted by no one who saw the child, and when the skull was opened the brain looked flattened, as if by effusion; but, except that its tissue was soft and that there was a slight increase of fluid in the ventricles, no morbid changes were discovered. There were no tubercles in other organs.

Several similar cases have since been recorded at Guy's Hospital and elsewhere. Clinically, they are distinguishable from tuberculous meningitis of the base by characters which we owe to the observations of Dr Gee, Dr Thomas Barlow, Dr Lees, Dr Carr, and Dr Still. They are admirably set forth in a monograph on the subject, based on nearly a hundred cases, in the seventh volume of Allbutt's 'System of Medicine' (pp. 492—559).

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* *Synonyms.*—Simple basal meningitis of infants—Posterior basic meningitis—Leptomeningitis infantum (Huguenin).

Anatomically, these cases do not differ from those of tuberculous neningitis except in two points, the cardinal one of absence of tubercles, nd the secondary point of the meningitis beginning in the posterior fossa f the skull, most often between the cerebellum and the bulb. Like the nore common and better-known disease, this form of meningitis seldom preads to the vault of the hemispheres, but almost always to the ventricles, nd very frequently to the membranes of the cord; like it, also, the exudaion is serous, fibrinous, and purulent in varying proportions.

Non-tuberculous basal meningitis attacks younger children—about half nder six months old, and only seldom after completion of the first year; hile only rarely have cases been observed in children between two and six ears old. It is not more common in boys than girls. Convulsions are not ery common, but tonic spasms are almost always present, particularly arly retraction of the head, which sometimes extends to well-marked connuous opisthotonos with stiffness of the limbs. Vomiting is as common as n other acute cerebral diseases. Strabismus is often seen, and nystagmus s not infrequent. Champing movements of the lips and tongue are seen, s in the tuberculous disease. There is often amaurosis, temporary or ermanent, but optic neuritis appears to be remarkably rare.

The course of the disease is decidedly longer than that of tuberculous, nd still more than that of septic meningitis; and the prognosis is much etter than in either. In cases which recover, there is not infrequently, owever, permanent hydrocephalus, owing to adhesions of the inflamed nembrane occluding the exit of blood from the choroid plexuses. Hence the isease is sometimes described as “ occlusive meningitis,” and the result as “ acquired,” in distinction from congenital hydrocephalus.

Some cases appear as sequels of measles or whooping-cough, others as omplications of bronchitis or broncho-pneumonia. In the exudation the Diplococcus of Fränkel (Pneumococcus or *Micrococcus lanceolatus*) has been ound, as well as *Streptococcus pyogenes* and *Staphylococcus pyogenes aureus*. These cases, however, we may surmise would belong to the “ pneumonic ” nd to the “ septic ” form of meningitis respectively. What is of much reater interest is that the microbe which we shall presently see to be haracteristic of epidemic cerebro-spinal meningitis—the *Diplococcus intrallularis* of Weichselbaum—has been observed in the sporadic cases just escribed. Dr Still found it in almost every example; and, while noting rtain differences between the microbe of the sporadic and the epidemic ses, believes that these differences point to a modification of characters ther than to a distinction of kind.*

Voltolini's disease.—In 1867 Voltolini, and afterwards Brunner, of ürich, described cases in which a child falls ill with fever and vomiting; is head is hot, he becomes delirious, tossing about in bed and screaming olently; within the first twenty-four hours he loses consciousness, and fter two to four days passes into a state of coma. At the end of another eriod of two to four days, however, consciousness returns; but when the hild first tries to walk he is found to stagger. He quickly becomes deaf, nd, as a consequence, remains dumb for the rest of his life. Voltolini's heory is that the disease in such cases is an acute inflammation of the labyrinth; the cerebral symptoms, as in Ménière's disease, being caused by affection of the portio mollis. Direct evidence from dissection after death seems to be wanting; and as Brunner remarks, the fact that the deafness is constantly bilateral is opposed to Voltolini's interpretation, and makes some affection of the floor of the fourth ventricle more probable.

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* The chief difference seems to be the much greater vitality of the diplococcus in sporadic ompared with epidemic cases. The distinction between it and the so-called pneumococcus (*D. anceolatus*) is its not staining by Gram's method; but they agree in living for only a few days.

Before going on to the epidemic form of meningitis, some other ætiological groups must be mentioned, in which sporadic non-tuberculous meningitis is met with—usually in adults—as a complication of other diseases, namely, pneumonia, syphilis, and Bright's disease, or as the result of drink, or exposure to the sun.

Meningitis is sometimes found secondary to acute *pneumonia*, and the presence of the specific microbe of the latter disease shows that the coincidence is not accidental.

Dr Fagge recorded the following cases:—(1) A man aged twenty-two, who had been in Guy's Hospital six weeks for pleuro-pneumonia, and was convalescent, was again attacked with dyspnœa and high fever, and died in three days; the spinal membranes, as well as those of the brain, were inflamed. (2) A man, aged twenty, had acute meningitis involving both hemispheres of the brain, and pleuro-pneumonia of the lower lobe of the left lung. (3) In the remaining case the patient was a temperate man, aged fifty-seven, who died, after sixteen days' illness, of acute pleuro-pneumonia affecting the left lower lobe; no cerebral symptoms appear to have been observed beyond delirium before death. There was much recent lymph, both at the base of the brain and on the vertex. The only other morbid conditions that were discovered were chronic renal disease (apparently in moderate degree) and hypertrophy of the heart.

The present writer, among 434 cases of acute lobar pneumonia, has only met with meningitis twice as a direct complication of pneumonia. In both cases there was abundant effusion of serum at the base and in the ventricles, with a little fibrin and no pus, and the *Diplococcus lanceolatus* was found on staining. In three other cases there was also ulcerative endocarditis.

Huguenin says that at Zürich meningitis is an exceedingly frequent complication of pneumonia. He quotes Chvostek as having found it four times in 220 cases in Vienna, and adds that in Zürich the percentage is still higher. Dr Osler also found it comparatively common at Montreal, 8 cases in 100.

The late Dr Moxon taught that *syphilis* sometimes is a cause of acute meningitis, and Dr Fagge found five cases of this kind in our *post-mortem* records, in addition to those set up by caries of the skull, and one in which a gumma was discovered in the brain-substance. In no instance was any disease noticed in the cerebral arteries. The present writer has seen two or three similar cases.

Occasionally in an autopsy on a case of Bright's disease, meningitis is discovered; and when this is associated, as it sometimes is, with pericarditis and pleurisy, we may accept it as secondary to the renal disease. There is no evidence of "rheumatic" meningitis.

Alcoholic poisoning has in two or three cases appeared to be the occasion of diffuse meningitis (see Dr Pitt's paper, 'Path. Trans.,' 1889, p. 353).

In hotter weather than we often meet with in England, meningitis is attributed to direct exposure to the rays of the sun. But few cases authenticated by an autopsy reach us from New York or Madras.

Huguenin speaks of labourers who have been attacked while working bareheaded in the fields, and he gives full details of the case of a sailor who fell ill the day after he had been rowing without anything upon his head in the hottest weather of July, and who died in five days. Another case related by the same writer is that of a student who was foolish enough to spend three hours, from 1 to 4 p.m., during a hot summer's afternoon, paddling about the Lake of Zürich on a floating board. All the exposed parts of his body were severely sunburnt. At 6 p.m. he was seized with headache, and an hour later with shivering, and all the symptoms of meningitis rapidly developed themselves; his illness, however, terminated in recovery. Guersant met with a similar instance, which ended fatally, and was verified by an autopsy, in an infant aged six months, whose cradle had been left in full sunshine in a garden. Rilliet and Barthez mention the case of a child who was attacked after reading a book in strong sunlight (!).

Epidemic Meningitis.*—During the present century there have been recorded from time to time epidemics of a disease characterised anatomically by inflammation of the membranes of the brain and cord, and clinically by fever, various eruptions, and cerebral and spinal symptoms, especially rigidity of the neck, or of the whole vertebral column.

History.—The first epidemic of this disease seems to have been in 1805 at Geneva. In 1806 it appeared in the United States, and continued to prevail there for ten years. From time to time, down to 1850, it was observed in several towns of France and Italy, in Algeria, Spain, and Denmark. In 1854 and for seven years afterwards it raged in Sweden, and destroyed more than 4000 persons. From 1861 to 1864 it showed itself in various parts of the United States. In 1863 it broke out in Germany; the north-eastern provinces of Prussia were the first to suffer; but within the next year or two it appeared in Erlangen, in Nuremberg, and in the country districts of Franconia. From that time it has never ceased to show itself at intervals of a few months or longer, now in one part of the German Empire, now in another. In New York in 1863, and in Boston from 1890 to 1898, it has been prevalent.

The British islands have hitherto been remarkably free from this disease. In 1846 it appeared in many of the workhouses of Ireland; and in 1866—1868 a very fatal type of it prevailed in Dublin, and to some extent in other parts of the country. Scotland has only once (1884) been visited by this disorder; and in England only a few isolated epidemics have been observed, in Lambeth (1867), Rochester and Birmingham (1876), and one or two villages in the country.

A few cases have from time to time been recorded as sporadic examples by writers who seem to have thought that the fact that a meningitis was cerebro-spinal was of itself sufficient to justify a presumption that it was related to the epidemic disease. But we have seen that all forms of inflammation at the base of the brain are apt to extend to the cord. The present writer has seen sporadic cases of cerebro-spinal meningitis in children, which have been proved after death to be due neither to tubercle, injury, nor septicæmia; but whether they ought to be classed with the epidemic diseases was impossible to decide before the present knowledge of the bacteriology of meningitis.

Anatomy.—In the deadhouse a case of epidemic meningitis has little to distinguish it from non-tuberculous leptomeningitis of the base. Pus and lymph are found both at the base and on the convexity of the brain, especially between the pons and the chiasma, along the large vessels, and in the various depressions and furrows on its surface. In the spinal canal the exudation is generally most abundant at the lower part, and on the posterior surface of the cord, having probably accumulated there by gravitation. The ventricles of the brain contain turbid serum; and the choroid plexuses and ependyma are coated with puriform lymph. Punctiform hæmorrhage or small spots of softening may be seen in the cerebral substance, and occasionally minute abscesses. The cord presents similar changes but less marked.

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* *Synonyms.*—Cerebro-spinal meningitis—Cerebro-spinal fever—Meningitis cerebrospinalis epidemica—Purpuric fever.—*Germ.* Nackenstarre—Genickkrampf.

In the most rapid "fulminant" cases, meningitis may only be discoverable with the aid of a microscope. But in one of Dr Gordon's cases (to be mentioned below) within a few hours greenish lymph had already been poured out on the brain and along the cord.

Among the appearances presented by other organs are congestion of the lungs, liver, and spleen, and kidneys. *Rigor mortis* is said to be of long duration. The cadaveric stainings of the surface appear early, and there may be the remains of purpuric eruptions during life.

The pericardium and the pleuræ are sometimes found ecchymosed or coated with coagulated lymph. The joints may contain pus, and sometimes there are scattered abscesses among the muscles. All these complications appear to be due to secondary pyæmia.

Course and symptoms.—As a rule cerebro-spinal fever sets in suddenly; the patient is seized with shivering and violent pain in the head, and feels so ill that he is obliged to go to bed at once. But sometimes—among Ziemssen's cases in five out of forty-three—there are slight premonitory symptoms, as headache, nausea, loss of appetite, and wandering pains.

Vomiting is almost always an early symptom, and is repeated whenever an attempt is made to sit up. After a day or two it generally ceases, but the headache mostly continues throughout the whole course of the disease, although it may sometimes subside for a time. It varies in character and in seat, being sometimes frontal, sometimes occipital, sometimes diffused over the whole of the head. Giddiness is often present with it. In cases of moderate severity the patient lies in a state of stupor, tossing restlessly about, but rousing when spoken to and trying to answer. He is often very irritable to light and sound. The pupils may at first be normal or contracted; ultimately they become dilated. In the more severe cases the patient becomes delirious, with or without convulsions, or passes into a condition like coma, though he still seems to feel the pain in the head, and groans or grasps his temples between his hands.

The *muscular rigidity* of the neck already referred to is scarcely ever absent, but it is not often marked during the first day or two. It varies in degree from a slight stiffness, noticed only when an attempt is made to bend the head forwards, up to a forcible retraction, bringing the occiput almost to a right angle with the spine. Burdon Sanderson explained it as due to a half-voluntary effort on the part of the patient for the relief of pain in the muscles; but it may be present when there is not pain at all, either in the neck or in the back. In about half the cases this symptom is accompanied by contraction of the extensor muscles of the dorsal and lumbar vertebræ. Sometimes the back is arched so as to be in a state of opisthotonos, but more often it is straightened into what Ziemssen calls *orthotonus*. If an attempt is made to raise the patient, he either slips down to the foot of the bed without bending his back at all, or allows his body to be lifted a little way, at the cost of much pain. He usually prefers to lie on one side with his knees drawn up, which relieves the pain in the back. Aching in the limbs, and especially in the legs, is often complained of; and the joints may become hot, red, and painful. An extreme cutaneous hyperæsthesia is another common symptom; the patient, though he may be comatose, will scream out at the slightest touch, or even if his bed is shaken. Rigidity seldom affects the limbs, nor is trismus of frequent occurrence; but strabismus, according to Osler, is a common symptom.

The degree of *fever* in epidemic meningitis is variable, and its course is very irregular. The temperature usually ranges from 100° to 103°, but it may fall and remain normal or nearly so for a day or two at a time; sometimes it rises to 105° or 107°, especially towards the last. The rate of the pulse may be natural or slightly increased, but it is liable to frequent fluctuations and is occasionally very slow. The spleen is sometimes, but rarely, felt enlarged. As a rule the abdomen is retracted, but it may be greatly distended. An abundant secretion of urine has been noticed by several German physicians, notwithstanding high fever; in exceptional cases a small quantity of albumen or of sugar has been present. There is decided leucocytosis, even in the so-called typhoid cases.

An important symptom in this, as compared with other forms of meningitis, is the occurrence of *cutaneous eruptions*. The most frequent is herpes of the face. This generally begins on the lips and spreads to the cheek, nose, ear, and eyelids; it is often bilateral, and more extensive than in pneumonia or ague. It first appears between the third and the sixth day, but fresh outbreaks may take place afterwards. It is said to have been noted on the trunk or on the limbs; but these parts are more often the seat of a roseola, urticaria, or purpura, like the symptomatic erythema of acute rheumatism. Sometimes patches coalesce so as to form large ecchymoses on the legs and trunk; and in bad cases at Dublin Dr Gordon noticed hæmaturia, epistaxis, and hæmatemesis.

The organs of *sight and hearing* are affected in many cases of cerebrospinal fever. There is often intense conjunctivitis, attended with chemosis. Ulceration of the cornea, irido-choroiditis, or optic neuritis, may develop. Vision may be suddenly lost at an early period of the disease; and is often not regained if the patient recover.

The present writer met with a similar case many years ago. A child between two and three years old had symptoms of meningitis with double optic neuritis; she recovered, but with loss of sight, complete optic atrophy coming on; and she continued blind until her death at the age of about sixteen, probably of some tuberculous disease.

The hearing was found by von Ziemssen to suffer in eight cases out of forty-two. Pain, tinnitus, impairment of hearing were generally experienced soon after the patient fell ill; they either passed off or ended in a partial or total deafness. Such symptoms sometimes depend upon suppuration of the tympanum, leading to perforation and discharge through the meatus. It has been suggested that in other instances they may be direct results of inflammation of the floor of the fourth ventricle, involving the *striæ acusticæ*, or that they may be due to suppuration around the seventh pair of nerves. In certain cases in which deafness had been present, Heller discovered after death a suppurative process in the labyrinth, besides an infiltration of the portio mollis with pus. It then became a question whether these morbid changes were caused by an extension of mischief rom the pia mater, or whether they began simultaneously with the meningitis. Heller was disposed to adopt the former opinion. It is urious that in cases of this kind the portio dura constantly escapes, so hat facial paralysis is not observed. Severe inflammation of the labyrinth sually leads to an absolute loss of hearing; and in most cases both ears are affected. The consequence is that the patient, if very young, never learns to speak. Even children two or three years old, who were able to talk before they fell ill with meningitis, and whose articulation after their recovery was at first tolerably distinct, soon begin to lose the power of speech, and ultimately become unintelligible. How important a part epidemic meningitis sometimes takes in the production of deaf-mutism is shown by the fact that in 1874 every one of the inmates of an asylum at Bamberg owed the defect to this disease.

The course of infective meningitis differs greatly in different epidemics, and is far from constant during a single outbreak.

Varieties.—The most severe cases of cerebro-spinal fever have been described as *foudroyant* or fulminant. The patient dies within a few hours. Thus Dr Gordon recorded one instance in Ireland in which the disease ran a fatal course within five hours, accompanied by a dark purplish eruption of spots of various sizes and shapes. Ziemssen says that among forty-three cases he met with four in which the duration was from twelve to thirty hours. This form of the disease is seen chiefly at the beginning of an epidemic.

Very mild and brief cases are termed *abortive*. The patient is only confined to bed for a day or two, or may even go on with his work as usual from the beginning to the end of his illness. Ziemssen has recorded in detail three examples of this form of the disease; in each of them headache, a painful stiffness of the neck, and vomiting (or at least nausea) were present; in one there was also rigidity of the upper dorsal vertebræ, and in another herpes and partial deafness were observed. In every instance recovery took place within four or five days. Abortive cases are said to be most numerous when an epidemic is declining. Their proportionate frequency is very variable. According to Hirsch it sometimes happens that the greater part of the population where cerebro-spinal fever is prevalent only suffer from this mild variety of the disease. Or it may be seen in the adults and the old people, while the severe form is raging among the children. Dr Ormerod aptly compares these cases with those of diarrhœa during the decline of cholera, and those of sore-throat in an epidemic of scarlatina.

Another modification of the disease is an *intermittent* form, in which there are regular paroxysms of fever, recurring with aggravation of all the other symptoms, the intervals being more or less completely apyretic. These cases are neither partly nor wholly of malarial origin, for they have been observed in districts where ague does not occur. Moreover, Ziemssen has shown that when measured by the thermometer the fever is far from exhibiting the regular course of that disease: the remissions or intermissions often last over several days, the temperature is irregular, and it is unaffected by quinine.

Yet another point of distinction is its tendency to attack children rather than adults.

Another so-called variety is the *typhoid*. This arises in protracted cases, and is marked by muttering delirium, a dry, brown tongue, sordes on the lips, involuntary evacuations, and bedsores.

Several observers have found that during or just after an epidemic of cerebro-spinal fever, meningitis has presented itself with unusual frequency as a complication of other acute diseases. This is especially apt to be the case with acute lobar pneumonia; no less than fourteen instances of it are recorded as having occurred in Erlangen between 1866 and 1872. It has also been noticed with pleurisy, acute tonsillitis, and scarlatinal nephritis. Many of the patients recover perfectly well, the headache and stiffness of the neck passing off as the symptoms of the primary malady subside; it may then be said that the meningitis is of the "abortive" variety. But not a few such cases terminate fatally; and the usual morbid changes in the membranes are then found at the autopsy.

Relapses are not very uncommon, even in cases of moderate severity, after a week or two of illness; and even when the patient's recovery is uninterrupted, it is often very slow. The headache sometimes continues throughout his convalescence, and may persist for years afterwards, undergoing aggravation when he stoops or makes any mental or bodily effort.

Sequelæ.—The most important result of the disease is *chronic hydrocephalus* due to cicatricial thickening of the pia mater. The quantity of fluid in the ventricles is often very great; in a case of Ziemssen's which proved fatal at the end of thirty weeks—that of a boy two years old—the thickness of the hemisphere (white and grey matter together) was only about an inch. A sufficient explanation of the occurrence of hydrocephalus under such circumstances might be found in the persistence of the inflammation of the ependyma which exists during the acute stage of the disease. But the fact that between the meningitis and the symptoms of the ventricular effusion there is often a clear interval of time, seems to show that, at least in many cases, the secondary affection depends on the cicatricial changes in the membranes. Dr Collins, of Dublin, in a case which he examined on the sixty-sixth day, found the opening between the cerebellum and the bulb occluded, and attributed to this the hydrocephalus, in accordance with Hilton's well-known views. As in another of Hilton's cases, the aqueduct may be occluded; or, again, the veins of Galen may be obliterated by adhesions. In other cases we may admit that the effusion is due to active inflammatory changes in the ependyma lining the ventricles, and this explanation would, as Dr Fagge remarked, particularly apply to the series of cases recorded by Dr Merkel, of Nuremberg, where, beside hydrocephalus, there was a large quantity of fluid beneath the arachnoid round the *cauda equina.*

The symptoms which indicate the supervention of hydrocephalus are said by Ziemssen to be severe headache, paroxysms of pain in the back and limbs, attended with vomiting, convulsions, and involuntary discharge of fæces and urine. During the intervals, which may last for weeks at a time, the patient may appear to be in good health; but often he is dull and stupid, or he is affected with a general cutaneous hyperæsthesia, or with paralysis or contraction of one or more of the limbs. Progressive emaciation appears to be another symptom.

Ætiology.—Those who are attacked by epidemic meningitis are seldom over forty, and generally less than twenty, years of age; in some epidemics almost all the cases have been in children under fifteen. But two of Ziemssen's patients were old people, aged seventy and seventy-seven respectively. Males appear to be more often affected than females. When it occurred in France at the beginning of the century it was often entirely limited to the soldiers in barracks, the civil populations of the same towns escaping entirely. In Ireland, in 1846, it affected principally the inmates of the workhouses; in the United States it fell with especial severity upon the negroes.

It is no doubt an infective disease, but of its being contagious in a narrow sense there is no evidence whatever; indeed, all observers are agreed that it does not pass from the sick to the healthy. Hirsch, however, has collected a series of cases which seem to show that a man going from an infected to a healthy place may sometimes carry with him the germs of the disease, so that not only he himself afterwards falls ill with it, but others are attacked in their turn. One suggestion is that a contagious principle is given off by the sick, but that it has to undergo some transformation or intermediate stage of its development, possibly in another animal, before it can infect a human being. It is stated, on the authority of Mr Ferguson, Veterinary Officer to the Privy Council in Ireland, that on each occasion when the disease has prevailed in that country there has been an epizootic of the same kind among pigs and dogs.

The transmission of a contagion, if there be one, is unusually rapid and mysterious. Burdon Sanderson reported that in 1865 meningitis broke out on or about January 15th in two districts of the department of Dantzic, distant at least thirty miles from each other. So again Stillé insists on its having repeatedly prevailed in Europe and in America in the same years, and on the way in which it has made its appearance within the United States at places hundreds of miles apart. In this respect he compares it with influenza.

From its natural history we should expect that one of the bacterial microbes would be present in this disease, and it seems clear that this was discovered in 1887, by Weichselbaum, who named it *Diplococcus intracellularis*, from its occurring within the exudation cells of the effused lymph or pus. The recent prevalence of the disease in the United States has enabled Stillé, Flexnor, and Barker, and other American pathologists to ascertain its approximately constant presence. If so, the epidemic disease is pathologically identical with sporadic basal meningitis (cf. *supra*, p. 813).

Diagnosis.—This is seldom difficult. At the commencement of an outbreak, however, it would seem that enteric fever may sometimes be mistaken for it. Leyden says that among the German troops before Paris in 1870 a series of cases occurred in which marked rigidity of the neck, severe headache, and hyperæsthesia were present, while the abdomen was flat, the temperature was low, and the bowels were confined. As first it was doubtful whether the disease was not meningitis, but the autopsies showed that it was really typhoid fever, with but slight implication of the intestine. Epidemics of the two diseases may also prevail simultaneously. Leyden has seen this several times; he insists on the facial herpes as distinctive, since it is never observed in enteric fever.

When occurring as a complication of acute pneumonia, epidemic meningitis may be far from easy of diagnosis. Ziemssen remarks that rigidity of the neck is often absent in these cases. Maurer maintains that a tense projecting fontanelle is only seen in pneumonia when accompanied by meningitis. Possibly, however, the two diseases are in their origin identical.

Again, if an isolated case of meningitis should occur, it is often impossible to say whether it belongs to the epidemic, the simple, or to the tubercular form. It would seem that the spinal symptoms—the painful stiffness of the neck, the rigidity of the vertebræ, the hyperæsthesia, and pains in the limbs—are generally more marked in the epidemic disease. Tuberculous meningitis may often be distinguished by its prodroma, by its gradual onset, by its slow and interrupted course. The presence of a roseolous or purpuric eruption would probably be a conclusive proof that the case was one of cerebro-spinal fever.

Tapping the subdural space in the upper lumbar region enables the conclusive bacteriological test to be applied. The tubercle-bacillus, the micrococci of suppuration, and the diplococcus of sporadic and epidemic basal meningitis, have each been discovered by this method.

Prognosis.—In epidemic meningitis recovery is not infrequent, but the disease often leaves deafness or blindness behind as a consequence; in several cases the remains of inflammatory exudation have been discovered when the disease has run into chronic hydrocephalus, which has proved fatal a few months later.

In giving a prognosis it is important to bear in mind the treacherous character of the malady. A case which at first appears to be of but little severity may afterwards develop dangerous symptoms and prove rapidly fatal; while, on the other hand, patients whose condition had seemed hopeless sometimes recover.

The mortality appears to vary in different epidemics from 30 to 70 per cent.; the mean mortality is estimated at 40 per cent.

Treatment of meningitis generally.—At an early stage one important object it to relieve the headache and to diminish the cerebral excitement. The patient should be placed in a cool, dark, well-ventilated room, and should be kept perfectly quiet. The hair should be cut short or shaved. Cold should be applied to the head, and for this purpose a large bladder containing small pieces of ice and a little water is more serviceable than anything else; evaporating lotions are far less effective.

In epidemic meningitis Ziemssen advises icebags to the head, back, and neck for weeks together, and finds that they relieve the patient's sufferings, and enable him to sleep.

In 1878 I saw a case which appeared to demonstrate the usefulness of the local application of ice. A man aged forty-three, a baker, was taken with a kind of fit on the evening of the 26th June, and was seen the same night by Dr Churchward with a temperature of 104·6°, a pulse of 160, vomiting, and great pain in the head and neck. During the night of the 28th he became collapsed, and remained sleepless and delirious, with contracted pupils and constant twitching of the hands. On the 30th I saw him and found the optic discs normal. We prescribed a mixture containing the iodide and the bromide of potassium, and a draught of chloral and morphia. Next day (July 1st) he appeared to be rather worse than better; he had not slept for more than five minutes; he had been sick again; and was constantly talking and picking the bedclothes. Dr Churchward then ordered a towel to be wrung out of iced water, and kept it applied all over his head and neck with lumps of ice between the folds. In a few hours he became quiet and fell asleep; the sickness ceased, and during the night he slept for six hours. On the following day (July 2nd) he was rational, and told Dr Churchward that he remembered nothing since the 29th June, except that another doctor had examined his eyes. The ice was continued until July 4th, and his recovery was uninterrupted. This patient died in the spring of 1880 of acute pneumonia. Dr Churchward obtained for me permission to examine his brain. There was no conspicuous change,—nothing, I think, that would have attracted my notice if I had not known of his former illness. But the arachnoid and pia mater at the base of the brain appeared to me to be thicker than natural, specially over the right Sylvian fissure; and there was more connective tissue on the under rface of the pons. The velum interpositum also seemed to be increased in density, though the lateral ventricles were not dilated.—C. H. F.

Most physicians abstain from anodynes in cases of meningitis; but ristowe frequently gave opium with manifest relief; Huguenin recommends he subcutaneous injection of morphia in small doses, and the use of enemata ontaining fifteen to forty-five grains of chloral; and Ziemssen says that in pidemic meningitis anodynes are indispensable.

To check vomiting the patient may have little pieces of ice to suck, or, what is often better, hot water in teaspoonfuls; of drugs, bismuth, hydrocyanic acid, and small doses of tincture of iodine, are the most useful.

There is perhaps no reason to suppose that leeches, or venesection, or the most active purging could cut short a meningitis which has gone on to the effusion of lymph and pus. But is it certain that such measures are useless in cases less severe? In epidemic meningitis German authorities now recommend all those "antiphlogistic" measures which were in vogue in this country thirty years ago. They not only apply leeches behind the ears and cupping-glasses to the spine; but in the most acute cases, employ venesection; and they administer calomel, or rub in blue ointment.

The older English physicians believed that they had seen successful results from similar energetic treatment, and some of Abercrombie's cases will perhaps bear quotation.

(Case 69) A girl, aged eleven, had violent headache and vomiting, followed by dilated pupils and stupor; pulse 130. She had been ill five or six days; purgatives, blistering, and mercury to salivation had been employed without benefit. One bleeding from the arm gave an immediate turn to this case; the headache was relieved; the pulse came down; the vomiting ceased; the bowels were freely acted upon by the medicines which they had previously resisted; and in a few days she was well. (Case 72) A gentleman, aged twenty-one, complained of headache, and after a day or two had double vision. The pulse was at first frequent, but fell gradually below the natural standard; and he became more and more oppressed, until he sank into a state of stupor. The case went on in this manner for eight or ten days, during which time he was treated by repeated venesection, cold applications, blistering, etc. The bowels were very obstinate, and the case was considered desperate, when he began to take croton oil in full doses, repeated every two or three hours. In a few hours he was purged nine or ten times; the same evening he was relieved from every alarming symptom, and in a few days he was well. (Case 75) A girl, aged seven, had severe headache, impatience of light, fever and slight delirium, followed by stupor, squinting, and great obstinacy of the bowels. She was considered in a hopeless state of hydrocephalus. At the end of a week, after strong purging, she recovered rapidly, and in a few days was free from complaint.—'Diseases of the Brain and Spinal Cord,' 3rd ed., 1834, pp. 155, 157, 158.

It is, no doubt, possible that the favourable issue of such cases was spontaneous, and would have occurred independently of treatment. But, on the other hand, it may be that under the less active practice of the present day they would have ended fatally.

In adult patients it is important not to overlook the possibility that syphilis may be the cause of the meningitis. The most careful search should be made for evidence of past lues, such as the presence of nodes or gummata, pigmented scars, swollen testes, or adhesions of the iris. If there is any reason to suppose that this cause may be in operation, antisyphilitic treatment may save the patient's life.

In cases of traumatic and septic meningitis the first point is to ascertain the origin and source of mischief, and sometimes this may be remedied by surgical interference. When otorrhœa is present, an exploratory operation is always advisable and is frequently successful. In too many cases, however, the endocarditis or other source of sepsis cannot be reached, and we must fall back on treatment by anti-streptococcic serum. In one or two cases the writer has seen this apparently successful, although in most it has failed.

In cases of tuberculous meningitis iodides in full doses are recommended by most authors, and may be of service when the effusion of serum is unusually large. But the only drug which the writer has seen useful is mercury, introduced as rapidly as possible by inunction and afterwards continued in small doses of the perchloride. This treatment was adopted in the only five cases in which he has seen recovery after apparently unequivocal symptoms of basal and presumably tuberculous meningitis. In one of the earliest of these he had taken charge of the child during the absence of his senior physician and supposed on its recovery that it had not really suffered from meningitis, to which the other replied: "You examine a case as authorities direct, you diagnose as they tell you, you treat the patient as they advise, and when the treatment succeeds you throw up your diagnosis."

In cases of non-tuberculous basal meningitis, the less rapid course and less fatal tendency of the disease enables us to use the above described method of treatment with more hope, and success has followed it. If, as seems probable, the epidemic cases are essentially of the same pathology as those which occur sporadically, the treatment which we believe may save life in the latter cases, ought sometimes to succeed in the former. But here the evidence seems to be strong that leeches and blisters, purging, mercury, and potassium iodide are all useless, if not injurious, and that the most promising treatment is by assiduous feeding (by the nostrils or by the rectum if needful), by administering brandy, and by giving opium in proportion to the patient's age. This drug was long ago recommended by Burdon Sanderson in England, by Ziemssen in Germany, and more recently by Stillé in America.

In cases of meningitis with symptoms of effusion, the attempt has been made to diminish the pressure in the skull by paracentesis between the first and second lumbar vertebræ. Unfortunately the success of this measure has been very slight; but being apparently free from danger, it seems likely to be of more service for the purpose of bacterial diagnosis between the tuberculous bacillus and the *Diplococcus intracellularis*, *D. lanceolatus*, and *Streptococcus pyogenes*. We may hope that such a diagnosis may some day give reasonable hope of effectual treatment by the appropriate antitoxic serum.

The yet bolder plan of trephining the skull and tapping, draining, or irrigating the subarachnoid spaces has been repeatedly tried, but in every case—excepting that of Dr Wallis Ord and Mr Waterhouse in a child between five and six years old, which was published in the 'Lancet' in 1894 (i, p. 597)—the result has been unsatisfactory.

A less severe method of relieving pressure, recommended by Barlow and Lees, is paracentesis of the tympanic membrane on both sides, and they record cases in which puriform exudation was thus evacuated and the patient recovered.

HYDROCEPHALUS.*—Among the diseases to which infants and young children are liable is distension of the cerebral ventricles with fluid.

Origin and pathology.—Hydrocephalus is sometimes *congenital*. At birth the fœtal head may be so large as to prevent its passage until it has burst or has been perforated by the instruments of the accoucheur. If the enlargement is less considerable, expulsion may at length take place, and the child may live for a shorter or longer time. In still slighter cases the head of the new-born infant is noticed to be softer and more pulpy than natural, but there is no obvious increase of size until a few weeks have passed. Among twenty-six cases collected by Dr Dickinson ('Lancet,' ii, 1870) there were four in which the disease was said to have been present from birth, sixteen in which it was discovered within the first six months, and six in which it was not noticed until between the sixth and the twenty-sixth month. No doubt these figures express, not the relative frequency of the congenital and acquired forms of the disease, but the number of congenital cases among them seen later in childhood. Acquired hydrocephalus arises later than in any of Dr Dickinson's cases. He insisted on the frequency with which water on the brain occurs in cases of *rickets*, and Huguenin says it is very common for rachitic children, if they are attacked with whooping-cough and bronchitis, to acquire hydrocephalus. In like manner the occasional association of hydrocephalus with congenital *syphilis* is supposed to depend on a defective growth of the cranial bones.

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* *Synonyms.*—Chronic Hydrocephalus—Hydrocephalus internus—Water on the brain—Dropsy of the head.

A few striking instances of its occurrence in several children of the same father and mother have been recorded. Frank saw in one family six, in another seven cases. Gölis reported the case of a woman who aborted six times in succession with dead hydrocephalic fœtuses at the sixth month, and bore three living children, two of whom died of the same disease when eighteen months and three years old respectively. These cases, which seem all to be congenital, were probably due to intra-uterine lues.

Acquired hydrocephalus has sometimes been ascribed to an injury, but this is very doubtful. In many cases the membranes at the base of the brain are found opaque and adherent; but it can only be seldom that hydrocephalus arises out of acute meningitis. In the epidemic form of that affection such a termination has been recorded, and also in the simple basal form; but there is no evidence of its following tuberculous meningitis, nor from the rarity of recovery could we expect it.

Huguenin speaks positively of having seen hydrocephalus begin with acute symptoms exactly like those of (non-tubercular) infantile leptomeningitis; but instead of ending fatally, they subsided and passed into those of chronic enlargement of the head. He says that in such instances he has found the pia mater at the base of the brain thickened and opaque, and the choroid plexuses also showing traces of a former inflammatory change. In a case of this kind related in detail by Rilliet and Barthez, where the early active symptoms lasted six days, it is expressly stated that the ventricular fluid contained only a trace of albumen, that the ependyma was normal, and that there were no adhesions of the pia mater at the base.

It is doubtful whether the physical and chemical properties of the hydrocephalic fluid enable one to determine whether the morbid process was of a passive or of an inflammatory origin. The normal cerebro-spinal fluid is well known to be of very low specific gravity, and to contain scarcely a trace of albumen; and in some cases of hydrocephalus the fluid has possessed similar properties, with the same proportion of potass to soda salts, and of phosphates to chlorides. In other cases the fluid has been of higher specific gravity, and has contained from 3 to 11·5 parts of albumen in 1000; and this is supposed to be a proof of the inflammatory nature of the exudation. The presence of leucocytes or of minute flakes of lymph is probably safer evidence, for Dr Fagge observed no less than four instances of the fluid being almost free from albumen in cases which were of inflammatory origin. The patients were all adults, and there was no reason to believe that they had had hydrocephalus for more than a few months.

Hilton and subsequent pathologists have shown that there is sometimes closure of the aqueduct of Sylvius, and, more frequently, occlusion by adhesions of the pia mater at the lower angle of the fourth ventricle, or by pressure on the veins of Galen obstructing the return of blood from the choroid plexus.* Hilton believed that such mechanical obstruction is often the cause of hydrocephalus, by preventing the outflow of the normal intra-ventricular fluid into the subarachnoid space of the cord, which ought to occur whenever increase in the physiological activity of the brain leads to an increase of its supply of blood.† The origin of the obstruction would in most cases be inflammatory, but the result would be passive exudation.

Other cases are probably due to intra-uterine disease or errors of development, and others again are exclusively and directly inflammatory, or rather, irritative exudation. That the decision is difficult is not surprising when we remember that in the far more frequent and accessible cases of ascites and hydrothorax, it is often difficult to decide how far an exudation is active and due to irritation, or passive and due to obstruction.

Anatomy.—The quantity of water found in the ventricles in cases of long standing is sometimes very great; six, eight, and even twenty pints have been measured after death. In extreme cases the hemispheres are transformed into a thin shell, perhaps not more than a line or two in thickness, so that it is difficult to understand how they retained any of their functions. The distinction between the white and the grey matter is lost; and the sulci, if visible at all, appear only as shallow grooves beneath the pia mater.

The nerve cells are said to be more or less completely atrophied; but accurate histological details seem to be still needed. The corpora striata and the thalami are flattened out, as are the crura cerebri and the pons. The weight of the brain is in most cases much reduced. Some of the more delicate structures, such as the septum lucidum and the soft commissure, are defective or absent, probably in consequence of the stretching to which they have been subjected. The foramen of Monro and the aqueduct between the third and fourth ventricles when not occluded are widely dilated. The ependyma is generally thick, tougher than natural, and of an opaque white or grey colour, while its surface is covered with translucent beads which give it an appearance aptly compared by Moxon to the leaf of an ice-plant.

Under the name of *Hydrocephalus externus* a condition has been described in which the seat of the effusion is said to be the subdural (arachnoid) space. No doubt this may be the case where there is an extreme degree of malformation of the brain, as in some microcephalic or anencephalous fœtuses, but otherwise the occurrence of external hydrocephalus is very dubious. The classical example is that of James Cardinal, recorded by Dr Bright, who died in 1824, at the age of twenty-nine, in Guy's Hospital.

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* See Cruveilhier, tom. iii, p. 385; and Hilton on 'Rest and Pain,' 3rd ed., p. 23, and figs. 1, 2, 8, and 9, with Mr. Jacobson's note, p. 39. Also a case of occlusion of the aqueduct with hydrocephalus, reported by Dr Fawcett ('Path. Trans.,' 1897).

† It is difficult to see why the fluid should continue to be poured out under the increased pressure which must necessarily result, and which certainly is present in most cases of hydrocephalus. Moreover, since the adhesions themselves are the results of a more or less widely diffused meningitis, such as is often attended with an inflammatory change in the ependyma, it would seem more reasonable to regard this as the cause of the effusion than to adopt Hilton's theory. It is well known that in one and the same case the various serous membranes may pour out fluids of very different specific gravity; and it is not mprobable that the ventricles of the brain may continue to secrete a fluid containing scarcely any albumen, even when the process is inflammatory. But the question can only be settled by careful observations as to the state of the openings into the fourth ventricle n a series of cases of hydrocephalus in which the physical and chemical properties of the luid are also accurately determined.—C. H. F.

The total quantity of fluid in this case was seven or eight pints, and all of it, with the exception of one pint, lay beneath the dura mater at the time of the autopsy. There was, however, a hole in the corpus callosum, and Bright himself supposed that the ventricles were the original seat of the effusion. Wilks always believed that the rupture through the corpus callosum did not take place till just before death; and the case cannot be fairly cited as one of external hydrocephalus (see Catalogue of Guy's Museum, No. 1000).

Congenital hydrocephalus is often associated with malformation of other parts as well as the brain. It is found in combination with spina bifida, and in one of these cases at Guy's Hospital the central canal of the cord was greatly dilated.

A certain degree of secondary dilatation of the ventricles is common in cases of cerebellar tumour; and sometimes, when the cranium becomes enlarged, the disease has been mistaken for primary hydrocephalus, until a new growth is discovered at the autopsy. In these cases the tumour probably presses on the veins of Galen and thus produces passive effusion into the ventricles.

Enlargement of the head is usually the first, as well as the principal symptom of hydrocephalus. Among forty-five cases collected by the late Dr Charles West there were twelve in which the frequent repetition of fits first drew attention to the disease, four in which it began with some other indication of cerebral disturbance, and six in which it arose out of an acute attack; in the remaining twenty-three cases no definite cerebral symptom preceded the discovery that the head was increasing in size.

The effect of the disease upon the cranial bones is to separate them more and more widely except at the base. Trousseau compared the change to the falling back of the petals of an opening flower. The frontal bone rises vertically above the eyebrows, or even overhangs them; the temporal and parietal bones arch outwards, so as to hide the ears when the scalp is viewed from above; the occipital bone extends backwards almost horizontally. Thus the head may acquire an enormous circumference. Dr Dickinson had under his care a child nine months old, in whom it measured thirty-one inches, and instances in which it ultimately reached forty and even fifty-two inches are cited by Trousseau. A strange contrast is afforded by the shape of the face, which appears unnaturally small, with angular features and a sharp chin. The base of the skull is generally narrow, with shallow fossæ. The presence of the fluid in the anterior cornua of the ventricles affects the orbital plates, so that they become convex downwards and thus arises a curious symptom of the disease; part of the iris, and even of the pupil, is hidden by the lower lid, while the sclerotic above the cornea is exposed to view.

The vertex of the head forms an open area, which corresponds with the natural fontanelles, expanded so as to meet together between the parietal bones and dividing the two halves of the frontal bone by a cleft that reaches nearly to the root of the nose. There is always, however, a work of ossification going on which tends to cover the brain in, in spite of the process of expansion which is going on. In examining the crania of hydrocephalic children whose death occurred while the disease was in progress, Dr Fagge found that the original outlines of the bones were still plainly visible, but that they were surrounded by broad zones of new osseous material, marked by radiating lines which showed the direction of their growth. The closure of the skull is often hastened by the formation of *ossa triquetra*, which may be felt loose in the membrane. The date at which it is completed varies widely in different individuals; in Dr Bright's patient, Cardinal, the anterior fontanelle is said to have been finally ossified at about the twenty-seventh year. As a rule the shape of a hydrocephalic skull approaches that of a sphere. It often, however, appears somewhat quadrilateral, from the projection of the frontal and parietal eminences. The bones have no diploë; they are generally very thin, and are sometimes translucent; but where life had been prolonged past middle age they have been found greatly thickened. The museum of Guy's Hospital contains a specimen of extensive ossification of the dura mater, which was taken from a case of this kind.

The separation of the cranial bones by accumulation of fluid within the skull is not altogether confined to infants. There is a classical case, recorded by Matthew Baillie, of a boy, aged seven, whose skull had appeared to be firmly united, but in whom at the time of death there was an interval of three quarters of an inch at the sagittal, and one of half an inch at the coronal suture. Dr Dickinson says that the same thing has been known to occur in adults; but even in children it is exceedingly infrequent when the bones have once become interlocked.

Physical signs.—In well-marked cases of hydrocephalus it is easy to transmit a wave of fluctuation from one hand to the other across the distended scalp. The skin and the subcutaneous tissues are exceedingly thin, and the ramifications of large veins are often plainly visible through the scanty hair. The head may be translucent, like a hydrocele, when a candle is held behind it; and in Cardinal's case this was observed, if the sun was shining behind him, until he was fourteen years old.

One may sometimes obtain a *bruit de pot fêlé* on percussing a hydrocephalic head, especially when the mouth is open.

Rilliet and Barthez maintained that in cases of hydrocephalus the systolic murmur, which is audible on listening over the fontanelle of a healthy child, is not to be discovered; but more recent observers have plainly heard it, although they admit that it is sometimes absent. Even under normal conditions this cranial murmur is only audible until the fifth month.

Symptoms.—A hydrocephalic infant is often unable to raise its head from the pillow, and an older child sits with the head resting on the table, or walks with it carried between his hands, "just as a milkmaid steadies her pail."

In those rare instances in which a skull already ossified expands under the influence of the disease (as in Baillie's patient already referred to), headache, stupor, and paralysis of all the limbs may be present long before any enlargement is noticed. Such cases are transitional between the hydrocephalus of early childhood and that of adult life. But in young children in whom the cranial cavity readily yields, it is often surprising how few symptoms of cerebral disturbance can be made out, even when the quantity of fluid is already large. Headache is often present; and the children sometimes show that they are in pain by restlessness and a sad whining cry. Vomiting is of frequent occurrence, and, like the headache, it is apt to be excited by movement, especially when the child sits up.

The *sight* sometimes remains good throughout the whole course of the sease; but in most cases there is blindness from an early stage, and the optic discs are found white and atrophied. Perhaps this sometimes occurs as the direct result of pressure on the optic tracts; but in many instances it is secondary to "choking" or neuritis. Huguenin examined three infants with congenital hydrocephalus between the twentieth and the thirty-fifth day after birth, and in each case found the discs reddened and swollen *(Stauungspapille).*

The *hearing* is seldom impaired; as Dr Dickinson remarks, a young child who is perfectly blind may at once recognise its mother by her voice. The smell is said to be sometimes absent, and the olfactory lobes atrophied, but probably in such cases there would be other malformations of the nervous centres as well. The *taste* seems often to be perverted; for the appetite is voracious and indiscriminate.

Impairment of the muscular power of the limbs may be present in all degrees, from a slight failure of co-ordination in standing or walking up to a total paralysis. Spasmodic affections of various kinds occur, nystagmus especially, but also partial clonic or tonic spasms of the limbs, and sometimes general eclampsia. The legs and arms are often stunted in their growth and their muscles undeveloped.

In some cases of congenital hydrocephalus there is no advance of intelligence from the time of birth onwards. In others the brain develops to some extent, but very slowly, so that during childhood such patients would generally be classed as imbeciles.

Cardinal, whose head grew from a fortnight after birth until he was five years old, learnt to walk soon after, went to school, and soon learnt to read well and to write a little; but he was obliged to give up writing from the headache caused by stooping over the desk. His health was tolerable until the age of twenty-three, when epileptic fits began to occur. When he was twenty-nine years old his memory was weak, and it did not retain dates and periods of time; it was said that he had never been known to dream. There was something childish and irritable in his manner, and he was easily provoked. He was stated not to have sexual desire, but he was fond of society and affectionate to his mother. His voice was childish, weak, and somewhat hoarse. He died in his thirtieth year, in 1824, having become exceedingly feeble, with symptoms of phthisis.

In the Fulbourn Asylum the late Dr Bacon had a female patient, aged fifty-three, whose head measured twenty-seven inches in circumference; yet she could sing and talk, and was not without memory or intelligence.

In some rare cases, in which hydrocephalus is thus arrested, the intellectual faculties are said to reach normal, and the name of the author of 'Vanity Fair' has often been cited as an illustrious example of the fact. But probably the difficulty of distinguishing a rachitic enlargement of the head from "water on the brain" has not been sufficiently taken into account.

For, strange as it must appear, it is a fact that errors have not infrequently been committed, even by skilled observers, as to the presence of water on the brain, and it has sometimes turned out that the disease was only rickets. This particular blunder may be avoided by measuring the circumference of the skull, and comparing it with what it should be, according to the age of the child, in a table of standard measurements, such as that which is given by Huguenin. The same writer says that even where the yielding of the softened bones leads to an accumulation of ventricular fluid in a rachitic infant, the ophthalmoscope shows the true character of the case, since the optic discs are unaffected. It is, however, doubtful whether hese tests apply to the cases which there is really most danger of confusing ith hydrocephalus, namely, the rare condition which will be described as ypertrophy of the brain* (p. 850).

Course.—The course and termination of hydrocephalus vary widely in ifferent cases. Sometimes the disease undergoes a rapid advance, and estroys life in a few months by coma or by a succession of epileptiform eizures. Very often the child dies through some intercurrent malady, uch as measles or whooping-cough. In certain very rare cases the fluid akes its way through the skull, and may even be discharged externally. okitansky once saw it poured out through the open sutures and diffused eneath the tissues outside the cranium. Several writers have recorded nstances in which it has escaped through the nose, or even through the pper eyelid (see a remarkable case by Dr StClair Thomson, with analyses of the cerebro-spinal fluid by Dr Halliburton, 'Proc. R. Soc.,' 1900). When the discharge takes place into the nasal fossæ it is supposed that the ethmoid bone must have been loosened from its attachments by the pressure to which it is subjected, just as a skull is separated into constituent parts by being filled with pus and then with water.

In 1884–5 the writer had a child under his care in Guy's Hospital with extreme ydrocephalus, whose case is worthy of note from its having been under continuous obser-ation from the beginning of the disease till its end. The patient was a remarkably fine oy, six years old, and free from family proclivity to disease of the brain. He first, while ving in the country, began to lose power over his bladder, then he had occasional fits, omiting, headache, and staggering, with slight loss of power in the limbs, and then dim-ess of sight. We found optic neuritis. It was at first thought that there was a tumour the superior vermiform process pressing on the veins of Galen and causing secondary ydrocephalus; but the subsequent course of the disease did not confirm this supposition. he child went through a slight attack of diphtheria. The head gradually enlarged, the it became feeble, complete blindness with double optic atrophy followed, and at last he y passive and almost motionless in bed with incontinence of fæces and urine, but almost ways free from pain and with no convulsions or paralysis. During the last few weeks ere was tonic contraction of the arms with ankle clonus, and for the first time rapid loss flesh. He died at the age of eight, having lived nearly two years in the hospital (Dec. 3th, 1885). *Post mortem* all the organs were healthy, except the nervous system. The rain was distended by serous effusion in both ventricles. The skull was completely sified. The central canal of the cord was wider than usual in the cervical region, and ere was early sclerosis of one lateral column. There were no adhesions about the fourth entricle nor signs of meningitis anywhere, except some adhesions and thickened dura ater over the orbital plates.

Prognosis.—Sometimes the disease may undergo arrest after a time, and s the child grows older the increased size of the head ceases to be con-picuous. Some years ago, in examining the body of a man aged forty-hree, who had been killed by an accident, Dr Fagge found well-marked ydrocephalus, probably a residue from childhood; unfortunately no information as to the man's mental capacity or attainments could be obtained. Such persons have been known to live to advanced age. It is, however, elieved that cases of this kind have a marked tendency to relapse, and that here is special risk of it at the time when the ossification of the skull is inally completed. In most cases death closes a melancholy scene before the hild has reached three years.

Treatment.—The treatment of hydrocephalus is unsatisfactory. As ight be expected, diuretic drugs are those chiefly prescribed, such as mall quantities of grey powder or of calomel, pills containing mercury and squill in doses adapted to the age of the patient, the liquor hydrargyri perchloridi, and iodide of potassium. It is doubtful whether benefit is obtained by such remedies, notwithstanding the two remarkable cases cited by Sir Thomas Watson. In Lord Herbert's Life (c. 1625) he relates the following marvel: "My cosen, Athelstan Owen, having an hydrocephale in that extremity that his eyes began to start out of his head, and his tongue to come out of his mouth, . . . I prescribed for him the decoction of two diuretic roots, which after he had drank four or five days, he urined in that abundance that his head by degrees returned to its ancient figure, and all other signs of health appeared."

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* I suppose a case of this kind occurred to me at the Evelina Hospital of which I ave unfortunately preserved no notes. During the child's life the presence of hydro-phalus was never doubted by me, nor by anyone who saw it; but when the skull was ened it was found to contain nothing but a large solid brain.—C. H. F.

The frequent presence of rickets in hydrocephalic children suggests the administration of cod-liver oil and of the preparations of iron; and these do not exclude such more direct treatment as is afforded by digitalis, iodide, or acetate of potass, or the perchloride of mercury, if it should be deemed advisable.

Of local modes of treatment, one consists in the application of pressure to the head. Formerly it was usual to cover the whole of the scalp with strips of adhesive plaster. But it has since been found sufficient to surround the head with a fillet of elastic webbing, two or three inches wide, and carefully adjusted, so as not to cause redness, or to impress the pattern of the material upon the skin. Care must also be taken to shift it from time to time, so that it may not irritate the frontal eminences. The necessity for such precautions is shown by the fact that sloughing of the integuments, leading to the death of the patient, has occurred when they have been neglected. Dr Dickinson speaks in high terms of this procedure, and says that it may be expected to succeed in arresting the disease in the majority of cases, provided that the child is young and that the enlargement of the head is of recent development.

One instance, which he relates in detail, is that of a boy, aged thirteen months, whose skull had been growing out of proportion to the rest of the body for nine months, and measured twenty-two inches round. The eyes were depressed. There were occasional convulsive attacks. An elastic bandage was put on, and it was worn for three years, being renewed as often as was necessary. Diuretics and cod-liver oil were given during the same period. Within four months the circumference of the head became reduced to 20½ inches. At the end of the treatment the bones were fully ossified; and although the head, which was then completely covered with hair, measured 21¼ or 21¾ inches, its disproportionate size was much less conspicuous. The position of the eyes was natural. There had been no convulsions for eighteen months. The child, at this time more than four years old, was sensible, and could make use of simple words for talking; but his memory was deficient.

Paracentesis of the head with a small trocar, at the outer angle of the great fontanelle, has been recommended by some physicians, and it has been performed on several occasions at Guy's Hospital. Perhaps the best thing that can be said in its favour is still the fact that in a case recorded many years ago by Mr Greatwood, a hydrocephalic child, having accidentally fallen upon a nail which penetrated its skull, recovered after three pints of fluid had slowly escaped through the wound. Cases are also on record of recovery after evacuation of fluid through the nose. Scarcely less successful results have been reported after paracentesis; but most of the cases do not stand criticism.*

Only a small quantity of fluid (not more than two or three ounces) should be withdrawn at a time, on account of the danger of setting up convulsions.

* See, for example, the late Dr West's remarks in 1842 on fifty-six cases of hydrocephalus treated by tapping the skull ('Lectures on Diseases of Children,' p. 130); and those of Sir Samuel Wilks ('Diseases of the Nervous System,' p. 175).

e result appears to be good, the tapping may be repeated again and , a bandage being applied in the intervals. The injection of iodine the distended ventricles has occasionally been practised, and has not followed by death; but there is no ground for supposing that it does . After having several times practised paracentesis, Dr Eustace Smith seen no decided benefit result, and sometimes decided evil, and Dr hart speaks but little more favourably of the operation. In the one in which the writer advised it the results were disastrous. Mr Watson ne has reported three cases in which "drainage" of the ventricle into subdural space was accomplished by means of catgut threads. The ts were not satisfactory (Brit. Med. Assoc., August, 1898).

f late years paracentesis has been performed in the lumbar region in of hydrocephalus as well as in tuberculous meningitis. It is a mode moving the fluid from the ventricles more gradually and more safely by puncturing the skull. Some cases seem to have benefited by the tion, and it deserves further trial.

ydrocephalus of adult life.—This is a very rare condition. Dean is said to have died of it in 1745, when seventy-seven years old, after ness of three years' duration; but one cannot speak confidently of the Watson records the case of a young and successful barrister, who, one or two sudden attacks of unconsciousness, became dull, stupid, nsensible, and at length died; the only alteration found in the brain he presence of a large quantity of serous fluid in the ventricles.

natomically there is a close correspondence between cases of this and those of hydrocephalus occurring in childhood. Similar changes ound in the ependyma of the ventricles; it is tough and thick, and be granular to the touch, or may have a pitted aspect like that of the le of the liver in some cases of perihepatitis; the floor of the third icle bulges; and the membranes at the base are thickened, opaque, natted together. The quantity of the fluid varies; it has not exceeded een ounces in the cases observed at Guy's Hospital. Whenever its cters have been recorded, it was clear, and contained very little albu- The cranial bones are generally thin, and there is deficiency of the e; the interior of the skull is marked by sharp ridges, with furrows en them, which correspond to the sulci and gyri of the hemispheres re obviously caused by the outward pressure of the brain. There is ed flattening of the hemispheres.

linically, the relation between the hydrocephalus of adult life and that ildhood is one of contrast rather than resemblance. The enlarge- of the head, which renders the diagnosis of the latter so easy, want- n the former; and the symptoms present scarcely distinguish it from chronic affections of the brain.

he following are the most remarkable among fifteen cases extracted the records at Guy's Hospital by Dr Fagge. In all of them the ven- s were dilated with effusion. The additional facts added in brackets observed in the cases which came under the present writer's view.

A man aged twenty-three, a patient of Dr Wilks, said that he had been well until a efore, when he noticed a numbness in his feet and legs, which gradually extended up face. He had kept his bed for three months. A fortnight back he once found f unable to micturate, so that a catheter had to be used. Within the last weeks he d two fits, in which he was insensible. On admission there was a slight convergent ; the pupils were dilated; the optic discs were ill-defined and red, with some

plugging of the veins and retinal hæmorrhages. He had numbness of the feet and legs, and of the face about the mouth. He did not chew his food properly; portions of it would fall out of his mouth while he was eating. His mind seemed not to be clear; and his statements varied. He passed his urine into the bed. After a few days he died suddenly.

2. A man, aged thirty-two, under the care of Dr Pavy, had been obliged to give up work eighteen months before his admission into the hospital, on account of a pain in the head. Nine months ago he lost his speech, had right hemiplegia, and was insensible for three weeks. From that time his memory was imperfect. Eight days before his death he again became affected with partial hemiplegia and inability to speak. He passed his urine and fæces under him. During the last few hours of his life he had a series of fits, which began in the left side of the face, and in which the eyes were turned to the right.

3. A man, aged fifty-seven, a cooper, was taken in with partial right hemiplegia, which had come on suddenly four days previously, while he was in the act of striking a piece of iron; he managed, however, to get home afterwards, the distance being half a mile. He did not seem very ill on admission, and looked a strong, healthy man; but he died rather suddenly, nine days after the attack. [In addition to immense distension of the ventricles, with roughening of the ependyma, there was in this case softening of the superficial parts of the corpora striata, and granule-masses were discovered with the microscope.]

4. A man, aged fifty-five, was admitted for chronic dilatation of the stomach, in order that the treatment with the stomach-pump might be carried out. But he became light-headed, and two days later he was convulsed and fell into a semi-comatose condition, in which he lay groaning and muttering until at the end of a few hours he died. He had complained of severe pains in the head three months before.

5. A boy, aged fifteen, who had two sisters in an asylum, was taken in with very obscure symptoms. He was anæmic; he vomited occasionally; there was slight fever; some of the superficial glands were swollen; he complained of pains in the head and in the back of the neck. After a time his mind began to wander; he lay on his side, with his legs drawn up; he answered questions slowly and unwillingly; his eyes were half closed and pupils dilated; the temperature was now below normal, sometimes not above 97°. He died very gradually.

6. A man, aged twenty-two, was admitted under Dr Habershon in 1871 for severe cerebral symptoms. He had once fallen from a scaffold, striking the left side of his head; and was insensible for a fortnight, with bleeding from the mouth, the nose, and the left ear. At the end of three months he resumed his work, but was noticed to be strange in manner. Seven months before his death he was attacked with violent headache and shivering. He gradually became unable to stand, and passed his urine and fæces under him. While in the hospital, he lay all day in a drowsy condition. He would answer questions, but soon began to ramble in his talk. He was occasionally sick, his pupils were dilated, and his head was drawn back. Two months before his death he had a fit; and a month later a second one. After this he lay perfectly still, saying nothing, and eating no solid food; towards the last he became extremely emaciated. [Besides well-marked indications of chronic meningitis, and a greatly dilated state of the ventricles, the anterior and middle lobes of the brain were adherent to the dura mater at the base, especially on the left side. There was a little ochrey-yellow discoloration, extending into the brain-substance, no doubt the result of effusion of blood at the time of the injury. The foramen of Magendie at the apex of the fourth ventricle appeared to be closed, so that the case might be cited in support of Hilton's theory of hydrocephalus (p. 825). The ventricular fluid contained scarcely any albumen.]

7. A man, aged thirty, a patient of Dr Wilks, died in the hospital in 1876 from disease of the aortic valves and bronchitis. Towards the last he seems to have had no marked cerebral symptoms, but when admitted he was comatose, passing urine and fæces under him, and remained so for several days before he gradually recovered consciousness. His coma began with a succession of fits, and while in the ward he had one or two attacks affecting the left side. He seemed intelligent, and answered questions readily. Eight years before, when he was a healthy young man, he had fallen from a ladder, and cut his head. He was brought home insensible, was delirious for two days, and was laid up for eleven weeks. Ever after he was unable to do any hard work. His memory failed him, he had headache, and feeling on the left side of the body gradually became defective. His first fit occurred five years before his admission. [Besides distension of the ventricles and the aqueduct, which held fourteen ounces of fluid, there was a repaired fracture of the base of the skull, and the brain on its under surface was discoloured.]

It is clear, from the cases just recorded, that in the adult diagnosis between hydrocephalus and other chronic cerebral diseases is exceedingly difficult, if not impossible. Hemiplegia is sometimes present, and, with eclampsia and vomiting, might easily lead to the diagnosis of a cerebral tumour.

Huguenin attempted to give a clinical account of the hydrocephalus of adults; but his description of the symptoms by no means corresponds with our experience. He speaks of the affection as bearing the closest resemblance to general paralysis of the insane, and reports two cases in which the resemblance undoubtedly existed, although there was no *délire des grandeurs.* His cases only afford additional proof of the variety of aspects that the disease may assume, and of the difficulty of its diagnosis.

In the last two of our cases acquired hydrocephalus was attributed to a severe fall on the head, and in one of Huguenin's patients it was traced to a railway collision. He refers to cases in which persons who had experienced blows or falls on the head, after suffering for several months, or even for years, have at length recovered; and remarks that the chief symptoms are headache, giddiness, and other distressing sensations, which often cause the patient to withdraw himself from society.

A patient of my own, a bank porter, had seven years before received a blow on the head from a heavy door of an iron safe; ever since he had been liable to pain in the occiput, vertigo, and a peculiar light feeling in the head. Sometimes he was free from these symptoms for weeks, but the least thing affected his head, so that he could take scarcely any stimulant. Reading often made him feel giddy; and he had been obliged to go out of church on account of indescribable discomfort. Once he complained of numbness in the left side of his head, and again of feeling as if his collar were too tight. The optic discs were normal. The perchloride of mercury was more useful than any other medicine to this patient; on one occasion it entirely kept off his symptoms for about a year. He also took bromide of potassium and the ammoniated tincture of valerian; and a blister was once applied to the back of the neck.—C. H. F.

DISEASES OF THE DURA MATER.—These are far less important than those of the pia: the most common is pachymeningitis secondary to injury or disease of the bone. The primary inflammatory thickening of the cervical dura described above (p. 675), has no analogue in the skull.

Plastic thrombosis of the sinuses of the dura mater. This is rare as a primary affection, though frequent as a result of injury. It consists in plugging of one or more of the sinuses with a firm clot, which becomes closely adherent, sometimes laminated, and finally undergoes organisation into permanent tissue. To von Dusch and Gerhardt we are indebted for the best account of it. It has also been described as "marasmic" and as "simple" thrombosis.

In the first edition of this work Dr Fagge could only refer to a single case from the records of Guy's Hospital. Dr Pitt, in his 'Gulstonian Lectures' for 1890, quotes eight additional cases from the same source. Three were in infants, two in patients between 20 and 30 years of age, and three between 40 and 46; two of the adult patients were men, and three women. The following account is chiefly taken from these lectures.

The most usual seat of the thrombus is the longitudinal sinus; sometimes it extends into the tributary veins, so that the hemispheres are covered with a coiling worm-like pattern; sometimes it is prolonged into one of the lateral sinuses. In some instances hæmorrhage in the pia seems to have occurred as a consequence of this affection, in others the substance of the brain has been found much ecchymosed or in a state of red softening.

There are certain conditions which favour this form of thrombosis, and may, therefore, suggest a suspicion of its presence. It has been observed in ill-nourished infants, six months or a year old, who have suffered severely from diarrhœa for some weeks before their death. Such cases generally resemble those of the "spurious hydrocephalus" described at p. 806, but with the addition of some more definite cerebral symptoms, such as nystagmus, squinting, ptosis, facial paralysis, and, most frequently, rigidity of the neck, back, or limbs. The fontanelle is generally sunken, but in one of Gerhardt's cases it became tense towards the last.

In adults, adhesive thrombosis of the cerebral sinuses and veins may occur like clotting of the femoral vein, after enteric fever, or parturition, especially when much blood has been lost. Such cases have been described as "thrombosis from marasmus," distinct from infective thrombosis. It sometimes also occurs in cases of pneumonia, and in the course of phthisis or chronic Bright's disease, in any condition of feeble cardiac power with liability to venous stagnation.

Dr James Taylor, in 'Allbutt's System' (vol. vii, p. 574), refers to cases of thrombosis of the longitudinal or cavernous sinuses which occur in chlorotic girls, apparently as the result of anæmia.

The recognition of plastic or adhesive thrombosis of a cerebral sinus is rarely possible during life. In its clinical features it resembles the more common affections of meningitis or tumour of the vault of the brain. Engorgement of veins running from the anterior fontanelle of an infant to the neighbourhood of the temples and ears, and the occurrence of epistaxis have been described, as signs of plugging of the longitudinal sinus; and œdema over the mastoid process as pointing to obstruction in the corresponding lateral sinus. In the chlorotic cases severe headache and vomiting have been observed, followed in some cases by hemiplegia and septic neuritis.

A curious case was reported by Dr Hyslop from the West Riding Asylum, of a woman suffering from acute mania, who lost all her symptoms for a period coincident with the appearance of hæmatoma of the ears and œdema of the mastoid region and of the orbits, which was believed to depend on temporary plugging of the sinuses ('Brain,' April, 1886, p. 90).

The duration of the thrombosis is variable; it may last several weeks, or death may occur a few days after its commencement, so far as this can be determined from the clinical history of the case. The lumen of the obstructed vein or sinus may, however, be restored and recovery ensue.

It is doubtful whether we have any direct means of preventing coagulation in the sinuses. In one remarkable case, the late Dr Moxon believed that salicylate of soda was of great benefit.

Infective thrombosis of the sinuses.—Far more common than the last-described condition is one of septic thrombosis of the sinuses.

This is always secondary; the thrombosis is soft and granular, and the endothelium is no longer smooth and thin. Anatomically it has the same relation to the last lesion as septic phlebitis has to adhesive inflammation of a vein, and it is as dangerous as the same process elsewhere; bacteria are always present, commonly streptococci.

It is most often the result of otorrhœa, with caries of the petrosal bone, but sometimes follows compound fractures of the skull, or caries of the bones of the nose or other parts, or facial carbuncle. In fact, its causes are identical with those of cerebral abscess (cf. p. 789).

It often leads to abscess, or to meningitis; or the septic phlebitis may spread from the lateral sinus down the internal jugular vein and produce purulent lobular pneumonia on that side, or general pyæmia.

Dr Pitt found 36 cases recorded at Guy's Hospital in the twenty years, 1870 to 1889. They were all secondary: 22 were due to septic suppuration of the ear; 7 followed fracture of the skull, and 3 carbuncle of the face; only one (a case of pyæmia) was caused by a distant lesion.

In 15 of these cases there was also lobular pneumonia; in 4 general pyæmia; in three abscess of cerebrum (1) or cerebellum (2); and in 5 meningitis.

He found the most frequent seat of intra-cranial thrombosis (whether simple or septic) to be the lateral sinus (36 cases out of 44), next the longitudinal (12), and next the cerebral veins (7). The other sinuses affected were the cavernous (4), circular, and superior and inferior petrosal. The greater liability of the labial sinus depends, no doubt, on the frequency of septic thrombosis from caries of the middle ear and mastoid cells.

This condition can only be recognised when local symptoms of the primary disorder, such as otorrhœa or tenderness over the mastoid process, or enophthalmos, give a local interpretation to the general signs of intracranial disease. The temperature is often very high, seldom normal as in cases of cerebral abscess. Rigors are usually present, with headache, vomiting, and sometimes diarrhœa.

Treatment.—In cases with local symptoms, the mastoid bone should be trephined, and a suppurating lateral sinus may thus be reached and opened. With the happy audacity of modern surgery, this has been repeatedly performed, and the patient's life saved. Moreover, to prevent extension of the septic phlebitis down the internal jugular vein, that vessel has next been ligatured, opened, and washed out; and, lastly, the upper segment has been fastened to the surface of the neck, so as to give free external exit to the products of inflammation in the skull. At Guy's Hospital, Mr Lane had a successful case of this kind in a boy ten years old, in August, 1888; and Mr Ballance, of St Thomas's Hospital, brought before the Medical Society of London (March 31st, 1890), four similar cases, two of which were brilliantly successful. Earlier cases are recorded in Macewen's classical work on 'Pyogenic Diseases of the Brain.'

*Hæmatoma of the dura mater.**—There is a condition which was for many years a puzzle in pathology, and is not without clinical interest. It consists in the presence of one or more membranous layers on the inner surface of the dura mater, within the so-called arachnoid cavity. When recent they are soft and vascular, but in course of time they may become tough, white, and fibrous, so as to resemble in appearance the dura itself. Virchow in one instance counted six or seven layers, and the membrane often extends over the greater part of one hemisphere or symmetrically over both. The layers adhere at their margins so as to form a closed sac, or a series of sacs, and are generally attached more firmly to the inner face of the dura mater than to the outer surface of the pia; but the cyst is sometimes perfectly unattached, so that it falls out as soon as the subdural space is opened. A considerable quantity of blood, either recent or tawny-brown with age, is found between the layers, which are often deeply stained with hæmatoidin; or the spaces may be filled with a thin serous fluid, sometimes containing cholesterine.

In 1845, Prescott Hewett, in a paper read before the Royal Medical and Chirurgical Society, maintained the opinion (which had before been advocated by Houssard and Baillarger) that the starting-point of this disease is an effusion of blood. But a few years afterwards Virchow gave powerful support to the doctrine (previously upheld by Bayle and by

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* *Synonym.*—Pachymeningitis interna hæmorrhagica.

Hodgkin), that the earliest morbid change is an inflammation of the dura mater; this, he supposed, becomes hyperæmic, and exudes upon its under surface a delicate material, richly supplied with wide thin-walled vessels, which rupture and yield the blood that is so commonly found extravasated. Virchow's view has been adopted by most of those who have since written on the subject; and his name, "pachymeningitis hæmorrhagica," has met with general acceptance.

Huguenin still maintains that the disease begins as a hæmorrhage; and if his observations are correct, one can hardly escape from the conclusion which he draws; in any case the best designation seems to be the old one of "hæmatoma of the dura mater," which leaves the question of its origin open. In a majority of instances the affection is merely a complication of cerebral atrophy, whether senile, or due to intemperance, or associated with general paralysis of the insane. Huguenin believes that in many cases of general paralysis he has been able to trace the earliest stage of a hæmatoma in the presence of a soft layer of blood-clot, spread out over the convolutions, having its greatest thickness (2 mm.) opposite the parietal eminence, and gradually thinning off towards its margins. This substance comes away in shreds from the dura mater, with which it has no vascular connection. Moreover, the microscope shows that the clots contain at this period nothing but a network of coagulated fibrin, blood-discs, and leucocytes. Afterwards the clot undergoes organisation, vessels are developed in it, and become continuous with those belonging to the dura mater, which now looks more or less injected. Subsequent changes lead to the formation of thick membranes, with blood or serum between the layers. So that apparently the same anatomical result may have its origin in either hæmorrhage or inflammation.

Admitting that some cases are primarily hæmorrhagic, Osler has described others in which delicate "false membranes" have been seen on the inner surface of the dura, vascular, but without any trace of hæmorrhage.

The most usual seat of this lesion is over the upper surface of the hemispheres, corresponding to the dura lining the parietal bones. In about half the recorded cases it has been bilateral.

Hæmatoma of the dura mater occurs more often in men than in women. It is met with chiefly in those who are advanced in life, but occasionally it may be seen much earlier; in 1864 Wilks showed to the Pathological Society a specimen taken from a young adult. It is often found in asylums and workhouse infirmaries, though very rarely in the deadhouse of a general hospital; the specimen just mentioned came from the dissecting-room, the subject, a "half-witted" young man, having died in a workhouse.

Among the causes of hæmatoma are enumerated chronic affections of the lungs or kidneys, anæmia, scurvy, and hæmophilia. Injuries to the head seem occasionally to produce it. In 1885 Dr Quain showed to the Pathological Society a specimen taken from a farmer, aged fifty-eight, who had for three years suffered from cerebral symptoms, following a fall from his cart, when he received a large scalp wound, and was stunned.

Excessive anæmia, particularly Addison's anæmia gravis, and chronic alcoholism, appear to be best established as predisposing causes.

Sometimes the only symptom of hæmatoma of the dura is a fatal apoplectic seizure, from outpouring of fresh blood between the membranes of an old coagulum; and often the lesion is found in those who have died of general paralysis of the insane, without unusual symptoms.

Huguenin relates the case of an intemperate man, aged thirty-one, who about two years before his death began to complain of severe headache, and who one day fell down unconscious, with a slow, full pulse, contracted pupils, transitory convulsive movements of the right side, and partial paralysis of the right side of the face. After twenty-four hours he gradually recovered his senses and got up; but he continued to suffer from pain in the head, slowly lost his memory and intelligence, and finally had another seizure which proved fatal in four days. The brain was found atrophied, with dilated ventricles, and there was a hæmatoma on each side consisting of a membranous sac divided into compartments.

That it is possible for hæmatoma to subside after giving rise to well-marked symptoms appears from the following cases of Griesinger and Bouillon Lagrange. So little is positively known of recoveries from any organic disease of the brain, that they are well worthy of record.

The first case was recorded by Bouillon Lagrange, and quoted by Huguenin:—A man, aged seventy-five, who was suffering from drowsiness and failing in memory, had a fall from his horse. Though not injured outwardly, he became deeply unconscious, with right hemiplegia. At the end of two months he was comatose and almost totally paralysed; he was unable to speak, and passed his motions under him. After this, however, he gradually began to improve, he regained his senses and his memory, he recovered the use of his limbs, and when two more months had elapsed he was considered to be cured. He remained well for six months, and then he was murdered. On a *post-mortem* examination, beside a recent injury to the skull, the back part of the right hemisphere was found to be flattened by a cyst, which adhered to the visceral arachnoid and contained three or four spoonfuls of a bloody liquid.

Griesinger ventured to infer the presence of dural hæmatoma in a man, aged fifty-seven, who recovered and was in good health at the time when his case was published. He had been a spirit-drinker, and for about five months had suffered from severe headache, extreme drowsiness, and confusion of ideas. His gait then became unsteady, his pupils were contracted, his pulse was rather irregular, and there seemed to be slight paralysis of the left facial nerve. He would sleep all day long, and passed his urine in his bed; but at the end of a month he began to improve, and in about ten days from that time he was perfectly well. Griesinger confesses not only that he anticipated a fatal issue, but also that he expected to find a rapidly growing cerebral tumour.

Meningeal hæmorrhage.—When blood is found effused into and under the pia mater, the symptoms are those of cerebral hæmorrhage producing apoplexy or hemiplegia or both. The effusion is usually a large one, the symptoms severe, and the fatal issue not long delayed. The cases referred to on p. 754 of eclampsia in cases of cerebral hæmorrhage, are most often, possibly always, cases of rupture of a vessel in the meninges covering the hemispheres in the neighbourhood of the motor tract.

In a woman who was more than once under the writer's care with symptoms of mitral stenosis, complicated by embolism of the femoral artery, and subsequently of the radial, death followed about eight hours after a sudden attack of apoplexy, and meningeal as well as cerebral hæmorrhage was found after death (April, 1890).

Congenital meningeal hæmorrhage sometimes occurs during protracted labour from interference with the circulation. It may prove rapidly fatal; but when the infant survives, atrophy of the cortex corresponding with the hæmorrhage follows, and produces a remarkable condition marked by spasmodic movements, and described as congenital choreiform spasms.

GENERAL PARALYSIS

AND OTHER CHRONIC DISORDERS DUE TO DIFFUSED LESIONS OF THE BRAIN

> "Quemne ego servavi in campis Gurgustidoniis,
> Ubi Bombomachides Cluninstaridysarchides
> Erat imperator summus, Neptuni nepos!"
>
> PLAUTUS, *Miles Gloriosus.*

General Paralysis of the Insane—History, distinctive characters, and nomenclature—Ætiology—Symptoms, stages and course—Exceptional cases—Anatomy and pathology—Diagnosis—Prognosis—Treatment.
Other forms of cerebral atrophy—Senile—Alcoholic—Saturnine.
Chronic diffused sclerosis of the brain—Hypertrophy of the brain.

GENERAL PARALYSIS OF THE INSANE.*—By far the most frequent and important of diffused as distinct from local cerebral lesions, is a disease which has not received a very distinctive or appropriate name, but is characteristic in its anatomy, its symptoms, and its course. With the exception of a slight reference in the works of Thomas Willis, the celebrated anatomist and physician, who died in 1675, the earliest mention of this disease seems to have been made by John Haslam, in 1798. In the second decade of the nineteenth century its study was taken up in France, and the first complete account was published by Calmeil in 1826. It has since attracted much attention both in England and in Germany, and has every claim to be considered a separate disease. Its symptoms and course are remarkably definite; its anatomy is constant, and brings it into close relation with its pathological allies, tabes and insular sclerosis. It rarely supervenes in the course of other forms of insanity; and the impairment of muscular power is of a peculiar kind, so that the mere association of hemiplegia or paraplegia with unsoundness of mind does not constitute paralytic insanity. Lastly, it differs from all other forms of mental disorder in having a constant and peculiar anatomical lesion as its basis.

Perhaps the best name is "Dementia paralytica," but in Germany the one most used is "progressive paralysis of the insane," and in this country "General Paralysis of the Insane," or more briefly "General Paralysis." It forms a large proportion of the cases which in popular language are called "Softening of the Brain;" and its importance may be estimated from the fact that in some lunatic asylums as many as one in four of all the male patients are said to suffer from it.

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* *Synonyms.*—Dementia paralytica—Softening of the brain (in part)—Cerebral atrophy with delusions—General paresis—Chronic meningo-encephalitis—Degeneration of the cerebral cortex.—*Fr.* Folie paralytique—Polyparésie—Délire des grandeurs.—*Germ.* Progressive allgemeine Paralyse der Irren.

Ætiology.—The persons most apt to be attacked by general paralysis are *men* in the prime of life. The proportion of males to females is variously stated; some writers give it as eight or ten to one, others as somewhat lower. The few women who suffer from this disease almost all belong to the lower classes, whereas among men it is most common in those who are well made and well educated, who have enjoyed life and lived hard.

The *age* at which it usually occurs is between thirty and fifty, particularly about thirty-eight and forty. In persons over sixty it is scarcely ever seen, although Austin refers to one instance in a man aged seventy-six. Those who live in towns and cities furnish a far larger proportion of cases than does a rural population. Thus the disease is rare in Ireland, in the Highlands of Scotland, and in Wales.

Paralytic dementia differs from other forms of insanity in being almost independent of inheritance or congenital predisposition. It is an acquired disease.

Until lately paralytic dementia was generally attributed to sexual excesses. Dr Sankey found that at Hanwell a large proportion of the women affected with general paralysis had led irregular lives. Griesinger attributed the disease to excessive "emotional agitation." Austin, Mickle, and Savage agree that it often follows anxiety, and the mental strain undergone in securing a fortune, particularly when combined with gambling and drink, late hours and dissipation; and in like manner Hitzig believes that it results most frequently from the combination of hard work with venereal excesses and indulgence in drink. It sometimes follows injuries to the head, or an acute febrile disease. Dr Savage finds it rare in epileptics, and frequent in those who are suffering from tabes. He believes that it is not caused by masturbation or sexual excesses, nor by excess in drink any more than by over-eating. When we are told that general paralysis is most often seen in fair persons with grey eyes, we must remember that most Englishmen are xanthocroic; and if most patients are married men, this is true of most healthy men also between the ages of thirty and fifty.

Whatever exciting condition may determine its outbreak, there is increasing evidence that the most important and almost necessary antecedent of general paralysis, as of tabes, is syphilis. In fact, Fournier classes tabes and general paralysis together as *parasyphilides*. The irregular lives, the sexual excesses, the late hours, dissipation and drink probably cause General paralysis only by favouring the infection of syphilis. The undoubtedly frequent co-existence with tabes is explained by both forms of chronic sclerosis being sequelæ of syphilis. It is true that dementia paralytica also follows insular sclerosis, and that no evidence of lues can be found in a certain minority of cases among men, and a still smaller minority among women. It is also true that most persons who suffer from lues never become general paralytics. Hence, as so often is the ætiology of disease, we must be satisfied with the discovery of a predisposing condition, and regard the efficient cause as the coincidence of this predisposition with some almost accidental determining occasion.

Symptoms.—The course of the disease is usually divided into three stages.

(*a*) During the *premonitory period* an alteration in the character is the chief symptom. Perhaps the man is extravagant in his expenditure; or he may be dull and sulky in his demeanour, or depressed and melancholy; and with this there is some impairment of memory. As Dr Blandford says, such a patient is regardless of appointments, forgetful of the time of meals, of the hour of going to bed, and the like. He comes and goes, scarcely noticing those about him, giving absurd and conflicting orders to his servants, and falling into a passion if they are not instantly executed. He neglects his business, and is careless and indifferent about what formerly interested him. He may commit indecent actions; but if he exposes his person he seems not to know what he is doing; or perhaps he assaults women, without regard to opportunity, place, or consequences. He sleeps ill, and drinks to excess from inattention. He eats hastily, and is apt to spill his food on his dress. He is neglectful of his appearance, and his dress is incongruous or unsuitable for the occasion. All this time there is no physical change detectable.

Dr Savage notes, among these early or premonitory symptoms, change of expression and of temper, perversions of the sense of smell, muscular fatigue, temporary aphasia, *petit mal*, and fits of unconsciousness or of convulsions. He also has frequently observed that a man has begun to fail in a handicraft in which he formerly excelled.

It is of the utmost importance that the distinctive characters of incipient general paralysis should be well known to the general practitioner, since he alone is likely to see the patient at this period of his disease.

(*b*) After a few weeks, or at the end of a month or two, the *second stage* develops itself, and the patient becomes manifestly insane. He now exhibits delusions which are connected with ideas of colossal size, or magnificent wealth, or extravagant numbers. He says that he can walk 100 miles in a day, or write 10 tragedies and 100 poems in the same space of time, or that he is going to make his fortune by buying up all the joint-stock banks, or that he is about to marry a princess. French writers give to this form of insanity the name *délire des grandeurs*; it answers to Mickle's "expansive" form. These "large delusions" are not maintained from day to day, nor are they consistent with one another. In fact the patient's mental condition is one of progressive decay. He is very self-satisfied, full of ideas of greatness, importance, and riches; but he takes no notice of the fact that the palace in which he resides is really a madhouse, and that the great men who surround him are lunatics like himself. Thus he cares much less about being placed under restraint than other insane patients.

In some instances, however, the delusions are not of this kind. Blandford mentions that one of his patients thought he was going to be arrested, that people were about to injure him, that they were maligning, or would rob him. Yet, although his symptoms so far resembled those of melancholia, this man was often cheerful and talkative; he was very vain of his appearance, and enjoyed his meals.

Again, cases of general paralysis sometimes begin with hypochondriacal fancies. Such patients imagine that their bowels are obstructed; or that their body has become very small; and the term "micromania" has been invented to contrast with "megalomania," the delusion of greatness and power. But a practical aid to diagnosis is the fact that, however depressed such patients may be in the earlier stage of the disease, they eat well and become fat and easy in mind afterwards.

:his second stage of general paralysis is further characterised by certain ical symptoms, which must be carefully looked for. The earliest of ı is usually a defect of articulation, an indistinctness or *thickness of :h*, or a hesitation in the middle of a sentence, or a tendency to sub- te for the proper word another of which some letters are the same, with ility to pronounce a difficult word or phrase clearly, leading to a ed and slovenly pronunciation, like that which marks a certain stage runkenness. The tongue, when protruded, is *tremulous;* that is, it merely shows a fibrillary tremor—which is often observed in persons are in perfect health, if they are nervous or anxious—but it oscillates ;ularly to and fro, and cannot be held quiet. Sometimes the lips are to quiver, as they do in persons about to burst into tears. Austin that the mouth is generally closed, that the lips are compressed, and the upper one is straight, its natural curves being obliterated. The ; loses its tone and becomes harsh in quality.

:he *pupils* are almost always unequal; and they are sluggish in their :ion to light and to accommodation. As a rule there is no optic itis to be found.

n some cases the *gait* becomes tottering, the patient walks stiffly and not lift his feet; he straddles, or shambles along, and stumbles over obstacle in his path. The movements of the hands may also be im- :d; Griesinger speaks of them as being "stiff;" objects are grasped ulsively, and suddenly allowed to fall. An important indication of disease at this stage is an alteration in the character of the *hand- :ng*—it becomes tremulous and uncertain, words are omitted, or the ı sentence is written over and over again, or the whole becomes an ıerent jumble.

:he *knee-jerk* is sometimes found to be absent: more often it is exag- :ed. Irregularity in this function is often a valuable early symptom.

n most cases one or more "fits" occur during this period of the dis- These may be of various kinds; the patient may become comatose, complete paralysis, anæsthesia, and abolition of reflex movements, in such an attack he may die. Or he may be less deeply insensible, loss of power affecting one side only, which may disappear in a few s. In many cases the fits are epileptiform, and these are often directly . Lastly, the patient may be attacked with convulsions without be- ng insensible, or falling.

'ersons affected with general paralysis in its second stage are ex- ingly liable to *paroxysms* of rage and fury, surpassing in violence ; that occur in any other form of insanity except paroxysmal mania. ı in an asylum such patients cause great anxiety and trouble to those ɔnsible for their safety, and they should never be allowed to remain in hands of their friends at this period of the disease. Blandford remarks these are the persons who sometimes get their ribs broken by atten- s before they can be mastered. They make the most desperate efforts scape; or tear up their clothes and go about naked; and they are often y in their habits.

After a variable period, a week, or a month, or a longer time, these ent symptoms commonly pass off. Blandford remarks that persons :ted with general paralysis are always more feeble in cold weather; ng the heat of summer they may regain strength to a surprising extent, with the first frosts they fall back. The patient's condition may even improve so much that he is able to leave the asylum, and the disease is perhaps thought to be cured; but all writers are agreed that permanent recovery is the rarest possible event, if it ever occurs. Blandford has known some persons affected with general paralysis who were able to live with their families, to spend their money without extravagance, and to write long letters without mistakes, detailing their travels and amusements; but he adds that he has not met with one case in which the patient was capable of work or business. In such persons a childishness and slowness of intelligence is noticeable, and if they attempt to resume their former occupations they break down, and have again to be placed in confinement.*

In many cases no improvement takes place at any stage of the disease, and its progress is uninterrupted.

(*c*) In the *third stage* the patient's mental condition gradually passes into one of dementia, and his bodily state into one of complete paralysis. His notions of magnificence may continue a little longer, but his understanding and memory undergo gradual dissolution. His speech cannot now be understood; his power of swallowing is impaired; his hands tremble so that he can scarcely hold anything; and it as much as he can do to shuffle about the garden with the aid of an attendant.

This stage leads on to a fatal termination, which is seldom long delayed, for the average course of the disease is a short one. According to Hitzig the majority of patients die in from fifteen to thirty months after their admission into an asylum. Calmeil and Griesinger speak of the ordinary course of general paralysis as lasting from several months to three years. But the last-named writer admits exceptional cases, in which life was sustained for as long as ten years; and Blandford mentions particularly the case of a baronet of large fortune, on whom a commission of lunacy was held in 1858, when he was suffering from general paralysis, and who was alive in 1870. In his experience the average duration of the disease is considerably longer than that stated by Griesinger.

The immediate cause of death is sometimes almost accidental. Thus a frequent mishap is choking; the patient goes on filling his mouth without swallowing the food, until it gets into the larynx and trachea, or at least fills up the pharynx so as to obstruct the entrance of air. Persons suffering under general paralysis should always have their meat minced, and an attendant should be present at meal-times. Another way in which death occurs is by the supervention of bronchitis or pneumonia, which may destroy life in a few hours.† If not thus cut short, the course of the disease is towards a stage in which the patient is bedridden, imbecile, and wasted, with bedsores and vesical paresis—the last scene to which so many diseases of the nervous system converge.

Exceptional cases.—The above account of the symptoms and course of the disease is, as in other cases, only generally true.

One exception of which the writer has seen more than one example is that there may be no period of confidence and boasting: the patient may be melancholy and depressed from the first. Several friends conversant ith mental disease have told him that such cases are more common than ey used to be. Probably in this, as in so many other instances, we form general, and on the whole, a true, conception of the natural history d course of a disease, and with increasing experience find that all general atements are liable to individual exceptions. For instance, we find cancer an earlier and tubercle at a later age than we supposed, we see young abetic patients improve and old men die quickly of the disease, and we em more often to see enteric fever begin abruptly and rheumatic fever adually.

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* The only exception to this rule that I have met with in my reading is a case related by Schüle ('Allg. Zeitschrift f. Psychiatrie,' xxxii).—C. H. F.

† I know of one case of general paralysis in which the patient had been for his usual drive in the afternoon, but in the evening was noticed to be out of sorts; on auscultation it was found that he had pneumonia, and next morning he died.—C. H. F.

Dr Savage remarks that not unfrequently we may be misled into think- g a case of general paralysis is one of melancholia until the patient, ough always complaining, gets fat on his diet; and then unequal pupils, r defects in his speech or handwriting, or increased or diminished knee- rks confirm the hint thus given; or an epileptiform fit may at once clear p the diagnosis.

Another exception is when the physical signs of general paralysis pre- de any mental symptoms. Thus a patient long under Dr Savage's obser- tion, a man thirty-eight years old, had hesitating speech, shaky hand- iting, tremulous tongue, unequal pupils, and increased knee-jerks, but ere was no insanity or exaltation of ideas until the paralytic symptoms d lasted for a long period.

While most cases are clear and unmistakeable, not a few are extremely scure, and it may be impossible for even a skilful and experienced server to determine whether they are really dementia paralytica or some her lesion of the nervous centres, or functional disturbance from alcohol, an unusual form of purely mental disorder.

As melancholia, so mania or dementia from drink or other causes may simulated by general paralysis. Moreover, instead of the uninterrupted ogress from bad to worse which marks many cases, there may be not only parent recovery after going away for rest and quiet in the prodromal riod, or transfer to an asylum in the later stages; but such decided im- ovement that the patient is sent back to his family, and the diagnosis is anged. Such apparent cure may take place more than once, so that an termittent type of the disease results which is like the *folie circulaire* mania.

In women general paralysis, according to Dr Savage, begins rather ear- er than in men, lasts longer, and has less severe symptoms; and while astic symptoms are common, those of ataxia are very rare. Unfortu- tely they are not barren till the later stages are reached, and their ildren are often imbecile.

Dr Clouston describes in 'Allbutt's System' (vol. viii), cases of "ado- cent general paralysis" occurring in boys between twelve and fifteen, the bjects of hereditary lues. They begin with symptoms of insular sclerosis d speedily pass into the stage of dementia.

Anatomy.—General paralysis differs from all other forms of insanity in ing constantly attended with morbid changes in the nervous centres. e condition is one of atrophy of the ganglion-cells of the cerebral cortex, th increase and hardening of the neuroglia—a sclerosis or grey degenera- n analogous to the sclerosis of tabes in the cord.

The dura mater is occasionally found much thickened, and it may be the at of hæmatoma (p. 837).

The pia mater is often adherent to the convolutions, so that the cortical substance is torn when one attempts to strip off the membrane; but in other cases there is an excess of fluid beneath the arachnoid, and the pia mater can then be removed more easily than from the healthy brain. Thickening of the upper surface of the pia over the hemispheres is perhaps the most constant condition—present according to Dr Goodall in more than 80 per cent. of the cases.

The weight of the brain is, as a rule, below the average and the ventricles are often moderately enlarged, with thickened ependyma.

In the earliest (and most instructive) cases, Dr Edwin Goodall has found the minute blood-vessels of the cortex thickened and their sheath swollen, and the pia mater infiltrated with small exudation-cells. Hæmatoidin-crystals are met with, and Deiters' spider-cells become numerous in the grey layers of the convolutions. The ganglion-cells become shrunken and distorted, with destruction of their axons and protoplasmic processes, and sometimes vacuolation of the body and disappearance of the nucleus. Sometimes, however, they are swollen and sclerosed, a change which Dr Major ('West Riding Asylum Rep.,' vols. iii and iv) described as hypertrophy.

In the white substance a diffused firmness is often observed, and patches of sclerosis have been seen.

In a well-known case Lockhart Clarke found the white substance of the convolutions full of cavities, varying in size from that of a small pea or barleycorn to that of a grain of sand, so that the cut surface looked like Gruyère cheese, or crumb of bread. Most of them were empty, but some contained the remains of vessels mixed with granules of hæmatoidin. Probably they all were originally perivascular canals, like those which have been discovered by Dr Dickinson in other diseases. They seem, however, to be distinct from the polycystic condition of the brain which was described by Drs Savage and Hale White, in the 'Pathological Transactions' for 1883 (vol. xxxiv).

Changes in the blood-vessels have been described in the morbid histology of general paralysis. The small arteries become dilated and tortuous, while their nuclei undergo proliferation. The arterial walls may be affected with fatty degeneration or calcification, and Dr Savage has often seen the perivascular sheaths crowded with leucocytes.

Other parts of the nervous system, as well as the brain, present morbid changes. Westphal found that the posterior or the lateral columns of the spinal cord are often affected with grey degeneration, exactly like that of tabes or lateral sclerosis. It is not supposed that such changes arise by the extension downwards of a morbid process which had commenced in the encephalon, for Westphal could not trace the affection of the posterior columns above the commencement of the fourth ventricle, nor that of the lateral columns beyond the lower end of the crura cerebri. They must, therefore, be regarded as of independent origin, corresponding to the clinical association of tabes with general paralysis. The whole cord is often, like the brain, shrunken and atrophied, with thickened and adherent arachnoid. The peripheral nerves have also been found altered. Thus the sciatic nerves were found by Dr Bevan Lewis ('West Riding Reports,' vol. v) to be smaller and softer than natural, with atrophy of the nerve-tubules, and overgrowth of the connective-tissue elements. This parenchymatous degeneration is confirmed by Dr Goodall.

Bonnet and Poincarré stated that the sympathetic ganglia, especially in the cervical region, present constant morbid changes; their nerve-cells sclerosed and pigmented or destroyed. They believed that these changes are the starting-point of the disease, and that its phenomena depend upon the resulting vaso-motor disturbances. But this conclusion is unjustified. The appearances are probably not morbid in the strict sense of the word, and after childhood are almost constant degenerative changes (see Dr W. H. White's paper, 'Guy's Hosp. Rep.,' 1890). So Dr Savage remarks, "I have carefully examined sections and can only say that I have found absolutely nothing characteristically pathological. That many cells are granular and contain pigment is nothing unusual."

The question which has already met us in the pathology of the cord, and will again in that of the abdominal viscera—whether the primary morbid change is in the essential elements of the affected organ or in its blood-vessels and interstitial tissue—is still undecided in the case of the brain. Some pathologists believe the primary process to be the effect of a hypothetical toxine upon the blood-vessels, leading to irritative changes in their coats and exudation into the perivascular sheaths, blocking of lymph-channels, passage of leucocytes into the neuroglia, and subsequent contraction and hardening (sclerosis) which destroys the ganglion-cells by starvation and strangulation.

But the view held by most neurologists at present is that ably expounded by Dr Mott in his 'Croonian Lectures' on the "Degeneration of the Neuron." According to this hypothesis, the first process is a decay of the neurons belonging to the "higher plane" of the nervous system—the ganglion-cells of the hemispheres, beginning in those of the frontal lobes and spreading over the entire cortex; while the chronic inflammation of the pia mater and the sclerosis of the neuroglia are secondary results of "the decay of the neurons and the irritation of their products of degeneration."

Diagnosis.—This must be considered from two points of view. In some cases it is a question between general paralysis and functional psychoses; in other cases between it and paralysis dependent upon chronic poisoning by alcohol, wasting of the brain, or other diffused changes in the cortex.

It is not generally difficult to say whether a patient is suffering from general paralysis or from *mania*, even at an early stage. But mistakes have sometimes been committed, from its being supposed that the presence of exalted ideas is constant.

Dr Blandford mentions the case of a gentleman who wanted to make a tunnel through the earth to the antipodes, and who thought that people might live a thousand years if they would bathe in beef-tea and beer, and that he should be able to pay off all the mortgages on his estate by assembling 10,000 persons in his park, having them photographed, and selling the photographs at five pounds apiece. But it was clear that he was not labouring under general paralysis, for he did not stutter, his memory was perfect, and he had had a similar attack some years before. The opinion was given that the case was one of mania, and that it might pass off; and the sequel proved the correctness of the judgment.

The exceptional cases of general paralysis which are occasionally seen in advanced age may be difficult to distinguish from those of *senile dementia*, and the childish delusions in those who are suffering from the latter disease lends aid to the diagnosis. But senile dementia seldom befalls persons under sixty; it is slower and more uniform in its course; it is less apt to be complicated with epileptiform fits; and any apoplectic attacks that may occur are much more likely to be followed by permanent hemiplegia.

Chronic *alcoholism* sometimes gives rise to a chain of symptoms so like those of the less marked forms of general paralysis that it may be impossible to make a diagnosis between the two diseases until one has watched the case for a time. Cases of what is called *syphilitic insanity* are quite distinct from dementia paralytica. They occur much earlier in the course of lues, not as a late sequel; they are marked by the wasting and earthy pallor of specific cachexia, and they show bullet-glands, ptosis and myosis, and suffer from nocturnal pains. Lead poisoning may also simulate paralytic dementia, but the presence of a blue line is distinctive.

Again, there are cases in which an impairment of muscular power shows itself some months before any physical symptoms develop themselves. It has even been a question whether the disease may not sometimes run its course without being attended at any time with impairment of the mental powers. This question is answered in the negative by alienist physicians; but one may fairly reply that they would not be likely to see such cases, supposing them to occur. It is in the wards of a general hospital that they must be looked for. Wilks published in the 'Guy's Hospital Reports' (xvi, p. 194) an instance in which the patient's mind was unaffected while paralytic symptoms were well marked; but this patient had only been ill nine or ten months, and mental symptoms may have afterwards developed.

There is sometimes great difficulty in distinguishing general paralysis in its early stage from *tabes dorsalis*. Wilks relates how two physicians —one an authority with regard to the former disease, the other with regard to the latter—had the same case shown to them, and that each of them pronounced it to be an example of the malady with which he was the more familiar. The explanation lies no doubt in the fact that the two diseases are frequently present at the same time. In Westphal's cases of grey degeneration of the posterior columns of the cord with general paralysis, the patients had presented the ordinary symptoms of tabes first; and as far back as 1862 Baillarger published clinical reports of five cases of a similar kind. General paralysis is thus a sequel of tabes.

In like manner there may also sometimes be difficulties of diagnosis between General Paralysis and *Insular Sclerosis*, of which it is not infrequently a complication or sequela (see Dr Bristowe's lecture, 'Brit. Med. Journ.,' Jan. 1st, 1887).

Prognosis is uniformly unfavourable. Although periods of arrest and even of improvement may intervene, yet, as a rule, the course is not prolonged, varying from a few months to two or three years, as much more rapid than cerebro-spinal sclerosis as that is more rapid than tabes. Prolongation beyond three years is not frequent, but authentic cases have lasted much longer (cf. p. 842).

In a series of statistics published by Dr James Adam in the 'Lancet' (Nov. 12th, 1898), he finds that the average duration of cases in public asylums is under two years, in military and naval asylums about two years, and in private asylums two years and a half.

Treatment.—In the early stage of general paralysis confinement in an asylum is absolutely necessary. Possibly it might sometimes run a more favourable course if the patient were secluded at an earlier period than is usually the case. His friends are too apt to take him to the sea-side, or to make him travel from place to place for change; whereas what his brain really needs is absolute rest. Among medicinal agents Blandford speaks highly of tincture of digitalis in doses of ♏xv to ♏ xxx every four hours; he says that it often soothes such patients wonderfully, and restores them from a state of noisy turbulence to one of comparative reason. According to

Hitzig the application of galvanism to the nape of the neck is sometimes of marked, though temporary, utility. The bichloride of mercury has been extensively used, but without any good result, even when there is a clear history of syphilis. Blandford lays stress upon the importance of withholding stimulants, such as brandy, while there is excitement; but he says that opium, morphia and chloral are often useful both in the early, and still more in the later stages of the disease. Crichton Browne published two cases in which the extract of Calabar bean in doses of a quarter to a third of a grain, continued for nine or twelve months, appeared to cure the disease ('Journ. of Mental Science,' 1875). Batty has seen benefit in the early stages from blistering the head and neck. Trephining the skull and drawing off the fluid subarachnoid has been repeatedly performed, but with only temporary results.

Other forms of atrophy of the brain.—In sharp contrast with the definite clinical course of General Paralysis, as seen in asylums for the insane, we sometimes meet with cases of a somewhat similar anatomy, but clinically distinct, and often marked by entire absence of symptoms. The fact was stated by Wilks in the 'Journal of Mental Science' for 1864; and this form of cerebral atrophy is not infrequent as is evident from notes of no less than fifty cases which Dr Fagge found in the *post-mortem* records of Guy's Hospital, and selected as the more striking examples.

The wasting of the cerebral substance is shown, not only by loss of weight, but also by an obvious shrinking. When the dura mater is turned aside, after the calvaria has been removed, the surface of the hemispheres may appear to be covered with a gelatinous substance, which, however, is really an accumulation of serous fluid in the meshes of the pia mater, and runs out as soon as the "visceral arachnoid" is punctured, leaving the membranes collapsed and wrinkled. The convolutions are small, and they are separated by deep, broad sulci; the ventricles are often large, the ependyma granular, and the choroid plexuses have undergone more or less cystic degeneration. The cerebral substance itself is in some cases firm and natural-looking; but in others it presents patches of softening, either thickly scattered throughout the white matter beneath the convolutions, or limited to the basal ganglia. In an old man of seventy-six, whose body was examined in 1876, the cut surface of the hemispheres showed smooth-walled cavities round the vessels, especially in the neighbourhood of the grey matter, so that it had an appearance like that of Gruyère cheese—exactly such as described above (p. 844). The minute arteries not infrequently are so thickened that their cut ends look like so many bristles embedded in the brain.

Sometimes this atrophy of the brain seems to be a *senile change*, and it may then give rise to no obvious symptoms; the old man whose hemispheres resembled Gruyère cheese was said to have been perfectly clear in his mind. In other persons of advanced years, however, there is a more or less marked failure of both mind and body, amounting in the gravest cases to decided dementia. One patient, a woman, aged sixty-eight, who was brought to the hospital for a fractured thigh, got out of bed, splint and all, nd tried to walk about, with the result that the limb became gangrenous. nother, a man aged seventy-four, who was taken in for bronchitis, showed o special cerebral symptoms, except that he passed his evacuations under im. Others again have been admitted for injuries due to attempts at suicide: a woman had thrown herself out of the window and fractured her spine, and a man had cut his throat. Probably many cases of this kind are indistinguishable during life from those of ordinary insanity, in which the brain shows no change which at present we can detect.

Cerebral atrophy is not confined to old people: cases occur in persons between forty and sixty years of age. Thus in 1867 there died in Guy's Hospital a man, aged forty-nine, who had been an accountant, but who for some time had been unable to do his work. His memory had failed, his speech had become uncertain, he wrote badly, and he misspelt his entries in a cheque-book. Afterwards his writing improved, but he formed his words very slowly. When speaking he would lose the thread of his discourse, and vainly clasp his head with his hands in search of what he wanted to say. For two or three days before his death he lay in a doze. At the autopsy patches of brown discoloration, breaking down in the centre, were found in each corpus striatum; the cerebral arteries were rigid and calcareous, and the kidneys were granular.

In some cases of this kind the disease proves fatal within a few months of its commencement, but more often it is prolonged over several years. There is often more or less complete paralysis of the limbs; sometimes convulsive movements or jactitations occur, and sometimes epileptiform seizures. Headache does not appear to be often a marked symptom; but in one patient it was exceedingly severe for the last six years of his life.

Cerebral atrophy is often the result of chronic *alcoholic poisoning*. A large proportion of those who die of delirium tremens have wasted brains; although in others the brain is found apparently healthy.

Another cause of cerebral atrophy is *poisoning by lead*. In 1863 a compositor, aged thirty-four, died in Guy's Hospital, who had been admitted for colic, and who afterwards became almost totally paralysed; the convolutions of the brain were shrunken, the sulci were deep and contained an excess of fluid, and the ventricles were unduly large. Chronic plumbism, as is well known, sometimes gives rise to epileptiform convulsions, which may be repeated at intervals, and followed by delirium and by a fatal coma. In cases of this kind also the brain is mostly found wasted.

In a patient of the writer's, however, a woman, aged thirty-five, who died after eclampsia, obstinate vomiting, and other symptoms ascribed to plumbism, the brain and its membranes after death were found perfectly normal and weighed forty-two ounces. No trace of lead could be found here, although it was abundantly present in the liver, spleen, and intestines. The patient had worked in a white-lead factory, and showed the characteristic blue line; she also suffered from colic and extensor paralysis of the hand.

General sclerosis of the brain.—If we consider how important a part in the pathology of the spinal cord is played by diffused chronic "inflammation" or induration, it is remarkable that similar lesions so seldom occur in the brain. We may compare the fact with the rarity or non-existence of acute idiopathic softening in the brain, and the frequency of acute myelitis. The various systemic diseases of the cord have also no counterpart in cerebral pathology. Dr Fagge, however, made two autopsies in which general cerebral sclerosis was found.

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(1) One of them was recorded by Wilks in the 'Guy's Hospital Reports' (Series iii, vol. xxii, p. 22). A girl, aged fourteen, was admitted in 1876 for general failure of mind

and body, which had been coming on for about a year. The earliest symptom was loss of sight, which came on one day while she was out on an errand. Soon afterwards she had two epileptiform seizures, which began with a scream. She generally lay quiet, with a vacant stare, making no complaints, but smiling when spoken to. She could evidently see, and the optic discs were normal, but the pupils were dilated. If asked what her name was, or any other simple question, she answered sensibly, but very slowly. When food was put into her mouth she would cease to chew without swallowing it, so that it lay there until removed by the nurse. She often vomited. She was unable to stand, and had very little power over her legs; even her arms were moved but slowly and feebly. Her skin appeared to be sensitive. A month before her death she tried to strangle herself, and was found with a towel tied round her neck. Towards the last she lay with her eyes open, making no attempt to speak beyond a slight motion of the lips. Her temperature was a little below or a little above normal. Before death her extremities became remarkably livid; and large bullæ made their appearance on the feet, which looked as though gangrene were beginning.

At the autopsy a deep purple discoloration was seen over the feet and ankles, the cuticle was detached from the rete mucosum, and the deeper tissues were all infiltrated with blood. The calvaria was thick and heavy and very unsymmetrical. The membranes were a little opaque, and there was an excess of fluid at the base. Over the convolutions the pia mater was everywhere adherent, so that in attempting to strip it off one peeled away a thin granular-looking layer of the cortex, leaving a roughened surface. The brain was small, weighing only thirty-four ounces, and its substance was tough. The cortex was not obviously wasted, and the boundary line between the grey and the white matter was everywhere well defined. The ventricles were much dilated, but their ependyma was not granular, except in the fourth ventricle.

(2) The other case occurred in 1878, in a child only eighteen months old, a patient of Dr Pavy's, in Guy's Hospital. Its illness was said to have begun with a fit at the age of four months. From that time it had been subject, especially at night, to seizures, in which it would fight for its breath, and appear to be choking, and would afterwards lie insensible for three days at a time, taking very little food, and twitching at the corners of the mouth. Fifteen days before its death it had a fit more severe than any before. Eleven days later it was again attacked, and on the following day it was brought into the hospital. It then lay comatose on its left side, with the corners of the mouth drawn down and constantly twitching: when it was moved over, its whole body would become rigid, and the twitching at its mouth was more marked. It appeared to have lost sensation on the left side. The temperature, at first 102·4°, rose to 103·8° before death, and the pulse became scarcely perceptible.

At the autopsy the child was wasted, with a pigeon-shaped chest and rickety enlargement of the ends of the ribs and of other bones. The brain was pale on the surface. The meninges looked healthy; they were not at all thickened or opaque, but very thin and delicate, as is usual at an early age. On attempting to strip the pia mater from the convolutions, however, it was at once found to be abnormally adherent, so that a thin, uniform, superficial layer of the cortex peeled off with it, leaving a rough granular surface behind. The substance of the hemisphere also was indurated, cutting firmly, and giving sections with sharp resistent edges; but the most striking appearance was a discoloration of the white matter of the hemispheres, which had a yellowish tint. This was more obvious on the right side than on the left, and in the anterior and lateral parts of the hemispheres than in the occipital lobe. It was also more marked in the prolongations of the white matter into the convolutions than towards the centre of the brain, and in some of the convolutions the limit between the white and the grey substances was ill defined. The grey matter itself looked natural. The ventricles were not dilated. The basal ganglia, the pons, the bulb, and the cerebellum were all of their natural soft consistence.

Portions of the affected tissues from each of these two cases were examined microscopically by Dr Savage and also by Dr Frederick Taylor, but no histological change could be detected, except possibly a slight excess of corpuscles in the interstitial neuroglia.

Dr Gee has reported two cases of general cerebral sclerosis occurring in children: in one, a girl of eleven, there was spastic paralysis, and after death the brain was found to be small and firm, with excess of leucocytes in the lymph-sheaths of the cerebral arteries; in the other, a girl of ten, there was right hemiplegia with indistinctness of speech, and at the autopsy there was found atrophy of the left hemisphere with general induration of the brain ('St Barth. Hosp. Reports,' vol. xvi).

In many recorded cases this diffused sclerosis has not affected the whole brain, but only certain parts (*sclérose lobaire*); and some of these have been attended with hemiplegia. Frequently the children affected have been deficient in intellect, and more cases are found in asylums and homes for imbeciles than in hospital wards.

HYPERTROPHY OF THE BRAIN.—It has long been known that in certain cases in which the head appears too large, so that the presence of hydrocephalus is suspected, a solid brain really fills the skull. Instances of this kind were recorded by Morgagni; and others were observed in the early part of the nineteenth century by several French observers, among whom were Laennec and Andral. The condition has generally received the name of hypertrophy of the brain; but its nature and its relations to other diseases still remain among the most obscure questions in pathology. It is certainly not increase in size or number of the cerebral neurons, like hypertrophy of the biceps or of the heart. Virchow surmised that it consisted in an overgrowth of the neuroglia. Sir William Jenner, not long afterwards, maintained that it was due to an "albuminoid infiltration" of the brain, like that which he believed affected the liver and spleen in rickets; he regarded it, in fact, as one of the minor effects of that disorder (cf. *supra*, p. 535). But it appears to be certain that the subjects of cerebral hypertrophy are not always rachitic.

Magnan is said to have minutely examined the structure of a child's brain so affected in 1874, and to have found it perfectly normal, and D'Espine and Picot accordingly described the disease as a true overgrowth of all the elements of the cerebral substance. This conclusion, however, we can scarcely accept, for such hypertrophy, or, rather, hyperplasia, always affects the interstitial, not the essential, elements of an organ. The "diffused chronic cerebritis," or sclerosis just described, is easily recognised by the naked eye, although the microscope fails to reveal marked or constant histological changes; and it is worthy of notice that in each of the two cases of "hypertrophy" which Andral described, the grey substance of the convolutions is said to have been indistinguishable from the white, while the bulb resembled a hard-boiled egg, and in some parts was harder still. How can such a condition be distinguished from sclerosis?

Dr Fletcher Beach, of the Darenth Asylum, furnished Dr Fagge with the following notes of two among six cases of cerebral hypertrophy that have come under his notice. He found that the white matter showed "a uniform granular appearance under the microscope, with nerve-cells scattered sparsely throughout; and there were numerous leucocytes present. The increase in size was evidently due to the large amount of granular matter"—not to hypertrophy of the nervous elements.

In each instance the size of the head was a conspicuous feature during life. In one, a boy aged sixteen, it measured twenty inches in circumference; in the other, a boy aged ten, the measurement was twenty-two inches. Its form was, in each instance, square, not, as in hydrocephalus, round.

Other points of distinction from hydrocephalus are that the enlargement is more marked just above the superciliary ridges than at the temples, the eyes are not so wide apart, and the fontanelle is often depressed instead of being full and elastic. This last character seems hardly to consist with the accounts which have been given of the dura mater bulging as soon as the skull was opened, and of the convolutions being flattened. The brain the older of the above two patients weighed no less than sixty-two ounces, the subarachnoid fluid was increased, and the ventricles were somewhat dilated.

None of Dr Beach's patients presented any signs of rickets, but, as he himself remarks, it is possible that they may have been rachitic at an earlier period, since they were, with one exception, above the age of ten years when they came under his observation.

Clinically it would seem that hypertrophy of the brain is characterised by a more or less marked deficiency of intelligence, drowsiness, headache, and liability to epileptiform convulsions; the gait is sometimes slow and tottering, and the weight of the head may cause it to hang forwards, or may throw the patient down upon his face when he is walking.

Under favourable circumstances life may be prolonged for years. The disease often ends by some intercurrent pulmonary affection, or by the supervention of acute symptoms and coma which, in one of Dr Beach's cases, were due to acute suppurative meningitis.

With regard to treatment nothing is as yet known.

FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM

THE SPASMODIC NEUROSES

> *Ποιεῦσιν δὲ αὐτῶν* (sc. *Λιβύων*) *συχνοὶ τοιάδε· τῶν παιδίων τῶν σφετέρων, ἐπεὰν τετραέτεα γένηται, οἴσπῃ προβάτων καίουσι τὰς ἐν τῇσι κορυφῇσι φλέβας . . . ἢν δὲ καίουσι τὰ παιδία σπασμὸς ἐπιγένηται, ἐξεύρηταί σφι ἄκος· τράγου οὖρον σπείσαντες ῥύονταί σφεα.*—Herod., iv, 187.[*]

> "Una senum facies, cum voce trementia membra."—Juvenal.

Arrangement of the Neuroses and particularly of Spasmodic Neuroses.

Local Spasmodic Neuroses—*Facial or Histrionic Spasm—Spasmodic Wry-neck or Torticollis—Course and treatment.*

Reflex and Fatigue Spasms: saltatorial and salaam palsy—Spasmus nictitans—singultus—athetosis, etc.—Tic convulsif de Gilles de la Tourette—Scrivener's Palsy, Hammer-Palsy, and other handicraft-spasms—Ætiology, diagnosis, prognosis and treatment of these affections.

Tetanilla—Description—Relation to Trismus neonatorum, Laryngismus, and Carpo-pedal contractions, and of all to Rickets—Ætiology—Treatment.

Thomsen's Disease—*Its history, characters, and symptoms.*

Paralysis Agitans—*History—Symptoms—Pathology—Diagnosis—Ætiology—Treatment.*

We have now completed the survey of those diseases which can be more or less certainly associated with organic lesions of the nerves, the spinal cord, or the brain. We have said nothing of diseases of the "sympathetic system," because, in the first place, the nerves and ganglia so named are not independent, but form part of the spinal system; and secondly, because there is no evidence of any clinically recognised disorder depending on derangement of the sympathetic nerves and ganglia.

There remain the functional disorders of motion, sensation, and intelligence, for some of which perhaps a special morbid anatomy is yet to be discovered; while others in all likelihood depend, not on lesions which can ever be distinguished by the scalpel or the microscope, but on nutritive or toxic disorders, which pervert action without perceptibly altering structure, or even on hereditary or acquired habits, which are only "morbid" when they are inconvenient or ungraceful.

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[*] Many of the Lybians have this custom: when their children complete their fourth year, they burn the veins on the crown of their heads with sheep's wool; and if this causes spasms in the child, they have discovered a remedy; for by sprinkling the place with goat's urine, they cure the disease.

We found it desirable to have regard to clinical characters even in the arrangement of organic diseases; and, in our ignorance of the causes and nature of almost all functional disorders, we have no choice but provisionally to classify them by their symptoms and course.

The first group of Neuroses may be termed Spasmodic, for all of them are marked by spasms or cramps in the voluntary muscles.

PAINLESS TIC.*—This disorder has no more distinctive title in English. Dr Sieveking, in translating Romberg, introduced the term *histrionic spasm*, but it does not carry its meaning with it, and has not been generally accepted. The term *facial spasm* is too limited.

Its essential feature is the occurrence, at longer or shorter intervals, of a sudden and rapid involuntary contraction in a single muscle or group of muscles. One eye may be instantaneously closed and opened again, or the forehead may be wrinkled on one side, and as quickly made smooth; or the angle of the mouth may be twitched, so as to cause a meaningless grin, or the nostril drawn up with an audible sniff, or the head is suddenly jerked round, or one shoulder shrugged.

The patient himself is often unaware of the complaint, which is, indeed, scarcely more than a morbid habit or trick. Trousseau once was consulted by a lady who came up from Burgundy with three daughters; they had all spasmodic tic in different parts of the body, and their mother complained of their unladylike twitches without knowing that she herself had intermittent spasms in her face.

The movements sometimes shift from one part and attack another. Sir Thomas Watson relates the case of a gentleman who when young used to give an involuntary shake of his head: a blister, applied to his throat for some affection of the air-passages, made this movement painful, and the movement ceased; but, to use his expression, it broke out in his nose, so that he was ever afterwards wrinkling it. A young Englishman consulted Trousseau for violent spasms of the head and one shoulder: methodical gymnastic exercises were prescribed; and after some time the affection disappeared from the affected shoulder, but presently showed itself in the other. A boy, aged twelve, four years before Dr Fagge saw him, had acquired a trick of sniffing and coughing, as though he would be choked; this was followed after two years by a habit of "making faces;" and a month before he was examined he began to toss his head over to the right side at intervals of a few minutes, whereupon the sniffing and coughing stopped. In adults, however, an affection of this kind often lasts for a lifetime unchanged. In a patient, whom the writer saw ill with pneumonia, the tic was a slight elevation of the angle of the mouth and the nostril of one side, followed by a sudden shake of the whole head. This was repeated at intervals in conversation, before the illness, during the attack, and after recovery. Trousseau narrates how, after the lapse of twenty years, he recognised a former schoolfellow, who happened to be walking behind him, by a barking noise that he made.

The involuntary clonic spasms just described are not confined to the muscles supplied by the facial nerve. Sometimes they affect one arm or one leg. The writer knew of a man who, in walking along the street, lifted one leg at regular intervals and slapped it with his hand, a trick of which he seemed to be unaware.

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* *Synonyms.*—Facial spasm—Spasmodic tic—Habit-spasm—Histrionic or pantomimic sm—Clonic spasm in the area of the portio dura—Habit-chorea (a misleading name).—Tic convulsif—Tic non-douloureux faciale—Tic simple.—*Germ.* Mimischer Gesichtsmpf.

These spasmodic movements almost always begin in childhood. They seem often to depend upon bad nutrition, anæmia, and depressed health generally, leading to want of stability and control of reflex, imitative, or emotional movements by the higher nervous centres. They have nothing to do with chorea or neuralgia or insanity, and under suitable treatment they usually disappear. But if neglected they become ingrained habits, and the nervous system is so modified by numberless repetitions of the action, whatever it be, that no change is possible. If a child "sniffs" or "makes faces" or "blinks" its eyelids, the habit may be broken; but if it persists after puberty it is probably incurable.

The best treatment seems to be, first, fresh air and good food, to which cod-liver oil, steel, and arsenic may be added with great benefit; and, secondly, helping the control of the patient over his movements by regular muscular drill, by sufficient sleep, and by removal of causes of imitation or of irritation. In cases where sleep is interfered with, chloral or bromide is the medicine indicated. Such irritative occasions for the movements as adenoids in the naso-pharynx, or hypermetropia, phymosis or masturbation, should be also looked for and treated.

SPASMODIC WRYNECK.*—A more serious form of spasmodic tic is a clonic spasm of the cervical muscles, which has been called "wryneck" or spasmodic torticollis." This is characterised by a rapid succession of jerking movements, which draw the head with great force towards one shoulder, and give rise to extreme discomfort. When one sterno-mastoid is chiefly involved, the corresponding ear is pulled down towards the clavicle, while the chin is pushed upwards, and the whole face is turned to the opposite side. When the trapezius is the muscle affected, the head is drawn backwards, without rotation of the chin, and the shoulder at the same time is raised. One or more muscles in a state of powerful contraction can be felt, or even seen, through the skin.

Ætiology.—Spasmodic wryneck occurs chiefly in adults. Reynolds noted that all but one of his patients have been more than thirty years of age, and the majority more than forty. However, one well-marked case appeared some years ago at Guy's Hospital in a girl aged eleven, who had had it ever since infancy. Men and women seem to be affected in about equal numbers.

This disease has been directly traced to cold. Thus the late Dr Golding Bird ('Guy's Hosp. Rep.,' vol. vi) had a patient who was attacked shortly after having been thrown into a state of partial stupor by driving across an open country in a gig on a severe winter's night. And Bright relates the case of a woman who was sitting exposed to a draught of cold air, when she suddenly felt what she thought was "a nerve giving way" on the left side of her neck, whereupon her head was drawn to the right. We had two instances in which the complaint appeared to be the result of a fall upon the head. Very often, however, no exciting cause can be discovered. The patient, if a woman, may have been pregnant when the complaint began, or it may have followed a severe mental shock or an attack of fever; but none of these conditions precede it often enough to be regarded as even predisposing causes. Dr John Harley reported, in vol. lvii of the 'Med.-Chir. Trans.,' the case of an engraver, whose head was habitually kept raised and slightly rotated to the right, so that he might have an oblique view of the plate on which he was engaged; but at last this became a clonic spasm. He also mentions the case of a governess, who spent much of her time in ruling the pupils' copy-books, and who used to rotate her head rhythmically and with emphasis from left to right; she, in her declining years, lost her control over this movement; and her face was twisted every second to the right shoulder, unless her attention was strongly engaged in some other matter.

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* *Synonyms.*—Torticollis clonica—Tic rotatoire—Nickkrampf—Clonic spasm in the area of the nervus accessorius.

Irritation from disease of the teeth is another reputed cause of wryneck. In two such instances, however, the spasm seems to have been *tonic* instead of clonic.

One case, which occurred in 1813, is related by Mr Mitchell in the fourth volume of the 'Med.-Chir. Transactions.' The spasms began in the tongue and side of the face, and only affected the neck after the lapse of some days; but precisely similar changes, in the extent to which different muscles are involved, occur in the ordinary clonic form of wryneck. The patient was a woman, aged fifty. During the attacks her neck was drawn round to the left shoulder, her arm was rigidly extended, her eyelids were widely opened, and the two eyes directed to the left; her mouth was opened and distorted to the left side, showing the clenched teeth; her tongue felt as hard as a board, and was curved to the left side; her forehead was wrinkled, and all the muscles of her face were thrown into a state of rigid distortion. After three months the tonic spasm was succeeded by a rapid tremulous motion of the affected parts, and the attack passed off, to be repeated at intervals which rapidly grew shorter, so that a fortnight from the commencement of the disease the spasms became almost continuous. Extraction of some carious teeth and fangs in the left upper jaw was quickly followed by a cure.

The other is Hancock's case ('Lancet,' 1859, i, p. 80) of a girl, whose head for more than six months had been drawn down nearly to the left shoulder; extraction of a stump and of a decayed tooth in the left side of the lower jaw completely cured her in a few days.

Course.—As a rule, the development of the complaint is gradual. Reynolds describes the patient at first feeling uneasy in his neck, and thinking that something is wrong with his cravat or with the pillow, until after some months he discovers, or is told by a friend, that his head is not straight. During the early progress of the case an ordinary observer might suppose that the man's shirt-collar was uncomfortable, and that he was easing it by twitching his neck; or that he was trying to look over his shoulder. After a time he complains of a dull, aching sensation, which extends to the shoulder or down the arm. At first, by an exercise of the will, he can overcome the spasm, so as to look straight before him, or towards the affected side; and even at an advanced stage he can sometimes by a great effort restore the equilibrium of the two sides for a moment, but at the cost of much distress, and with the risk of aggravating the severity of the spasms afterwards. As the disease goes on, he generally learns how to bring his head into the proper position with his hands; thus a patient used to walk about with his arm raised, and ready to seize hold of his nose, which he employed as a kind of lever to control the movements.

The spasms are always arrested during sleep, and they often cease when the patient is in the recumbent posture, or when he supports his head with his hands. They are at once brought on by any excitement, or by talking, by attempting to walk. In the more severe cases the head is jerked out for hours without any interval. Sleep is almost impossible, and the tient's state is one of the utmost misery.

The disease may remain stationary for years, neither advancing nor receding. But sometimes it extends from the parts first affected to the side of the face and the eye, or to the shoulder and arm. In two cases at Guy's Hospital the movements began in the upper limb and afterwards spread to the neck. This, and the fact that the deep cervical muscles are sometimes the ones mainly concerned, prove that the spasm is not always confined to the sterno-mastoid muscle, nor to the distribution of the spinal accessory nerve, as would appear from the description given by Erb and others. In some cases it passes off within a few months under treatment, and long afterwards returns with its characters unaltered. Thus in the case of Dr Golding Bird's, already referred to, there was an interval of about seven years between the first and second attack; and in another case the patient got well, returned to his work as a gasfitter for thirteen years, and at the end of that time was again seized with the disease, but on the opposite side.

Wryneck seems very seldom, like facial spasm, to desert one part of the body and attack another afterwards.

In the advanced stages of the disease, the muscles which are the seat of the spasms sometimes fall into a state of *tonic* contraction, so that for hours together the head remains obliquely drawn down to the shoulder, but perfectly motionless. Even then, however, any kind of emotional excitement, or the slightest attempt at voluntary movement, generally leads to a recurrence of clonic spasms.

There are, however, cases of primary *tonic wryneck* (*caput obstipum spasticum*), in which no jerking movements have preceded the continuous contraction. A girl's head was drawn down so that the chin rested on the second left costal cartilage, and the right side of the lower jaw was so close to the sternum that it was difficult to introduce a finger between them. Very slight twitching movements were all that had been noticed, but the affection was said to have developed itself while the patient lay paralysed in bed, unable to move a limb; she was the subject of well-marked hysteria, and no doubt the wryneck was of the same nature as hysterical contraction of the limbs. This tonic contraction of the sterno-mastoid is to be distinguished from the, for the most part, congenital contraction of the sterno-mastoid, associated with hemiatrophia of the face (see Dr Taylor's case in the 'Path. Trans.,' vol. xxvi).

Results.—One curious effect of wryneck when it occurs in childhood is the permanent deformity in the bones of the face to which it gives rise. In the case just cited the left side of the face was considerably larger than the right. When the girl's head was erect, the right eye lay at a higher level than the left one, the median line of the face, instead of being upright, formed an obvious curve, with its concavity towards the left, and the left side of the upper jaw projected beyond that of the lower jaw, while on the right side the relation between them was reversed. The vertebræ of the neck were also distorted, there being a projection on the left side, apparently caused by the transverse process of the atlas. Precisely similar deformities were noted in a little girl of eleven, who had been in the hospital a few years before. They are of importance, because they might be regarded as indications of primary disease of the vertebræ, such as often produces a prominence of the cervical spine on one side, and also causes impairment of the movements of the head.

Treatment.—This is very unsatisfactory: on the one hand many cases are altogether intractable and are not benefited by any curative measures; on the other hand, those in which relief is afforded, or which get perfectly well, yield to the most diverse remedies, of which each in turn fails more often than it succeeds. Reynolds (in his 'System of Medicine,' vol. ii, p. 796) found that a continuous galvanic current of moderate strength, passed through the muscles which are the seat of spasm, causes them to relax for a time. But he adds that even when by this means the head has been maintained in equilibrium for many minutes, he has often observed that, as soon as the poles are withdrawn, the spasms return; and sometimes the ultimate effect of the treatment has been rather to increase than diminish the movements. In a patient who some years ago came under the late Dr Habershon's care at Guy's Hospital, a considerable amount of relief was afforded by fastening the sponges connected with a weak, portable galvanic cell upon different parts of the neck, and leaving them for some hours at a time. Faradisation of the muscles on the opposite side of the neck is sometimes useful, by increasing their power of resistance. On the affected side there is increased sensibility to interrupted currents; the patient may be unable to bear one which is so weak as to cause no pain whatever on the healthy side.

Various mechanical supports have been invented to keep the head in its proper position, and they may be useful in recent cases, by controlling the movements for an hour or two; as, for instance, when a physician wants to get through a lecture. Reynolds speaks highly of the hypodermic injection of morphia as a sedative. But this is a cure which may easily prove worse than the disease.

In one case recorded by Dr Radcliffe ('System of Med.,' ii, p. 133) arsenic was injected subcutaneously, five to fourteen minims of Fowler's solution over the affected muscles at intervals. The local inflammation was diminished by diluting the solution with water; and after nine weeks the patient left the hospital almost well. This method of treatment was, however, tried at Guy's Hospital by Wilks and also by Moxon without success.

Dr John Harley ('Med.-Chir. Trans.,' lvii) related two cases in which the administration of large doses of succus conii proved useful. Beginning with an ounce of the liquid, he rapidly pushed the dose in each case until one patient took three and a half ounces daily. In the first case the spasm became greatly diminished; but the drug had ultimately to be discontinued on account of the mental depression it caused. In the second case the affection was almost cured, and the patient resumed his occupation, working half-time, with only an occasional tendency to a twist of the head if he became over-tired. Moxon followed this plan with a decided measure of success.

In a case of the writer's the constant galvanic current failed to do good, but the patient gradually improved under a mixture of bromide of potassium and arsenic, and went out apparently cured. Others, however, have seemed incurable.*

Division of the spinal accessory nerve is not justified by experience; still less resection of the sterno-mastoid muscle, an operation often successful in cases of neither clonic nor tonic contraction, but congenital shortening of the muscle.

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* A severe plan of treatment was recommended by Busch,—the application of the actual cautery to the neck on both sides of the spine, in lines five or six inches long. In me inveterate case ('Med.-Chir. Rev.,' 1866) Campbell de Morgan excised an inch of the trunk of the spinal accessory nerve, above the origin of the branches to the sterno-mastoid muscle, with permanent relief to the spasm, but with equally permanent paralysis of the affected muscles.

Reflex and co-ordinated tics.—In more complicated cases of spasmodic tic, the attempt to walk is accompanied by a spasm of certain muscles, so that a kind of stringhalt is produced. One patient, a journeyman tailor, had an extraordinary seizure whenever he jumped up suddenly from the sitting posture; the right leg was bent at the knee, the left was thrown over it in violent flexion, the body and head were twisted to the right, the right arm was extended and raised, and the left thrown into extreme pronation. Then there was writhing of the whole frame, the facial muscles twitching here and there; and the attack passed off with a groan of relief. This man could make every possible movement, as slowly or abruptly as he pleased, so long as he remained seated or lying down. He was even able to get up, and after cautiously standing still for a moment, could walk away as well as anyone else. Such cases resemble the saltatorial spasm and the hereditary cases of Thomsen, to be presently discussed (p. 871).

In other cases, again, spasms accompany the act of laughing or talking, or, above all, chewing. One of Weir Mitchell's patients, as soon as he began to masticate his food, felt his mouth jerked open, so that he had to keep shutting it with his hand. When he wanted to swallow, his face assumed an aspect of terror. "He would suddenly muster courage, and swallow the contents of his mouth at a gulp. Then instantly the jaw flew open, the head was drawn back and down upon the left shoulder, the face was convulsed, and sweat ran from the forehead." In another case of the same kind the gastrocnemii were involved in the spasm, so that the patient was jerked into a squatting position. In an instance recorded by Dr Fagge, the chewing of food brought on a kind of wryneck, the head being twisted over to one shoulder; in another, the effort of speaking was attended with an extraordinary series of grimaces. Such cases seem to be allied to the spasms brought on by handicrafts (p. 860).

Saltatorial spasms.—Of all this group of spasmodic neuroses called forth by voluntary or reflex movements, the strangest are those in which, as soon as he stands up, the patient's legs pass into a state of clonic spasm, and he begins to execute a succession of rapid jumping and skipping movements. So great are his efforts that his face flushes, his pulse is quickened, and he breaks out into a profuse sweat. Yet when he is lying or sitting he remains quiet, and can move his legs perfectly well in all directions. For this affection a special name was invented in 1859 by Bamberger, who recorded in the Vienna 'Med. Wochenschrift' two cases of what he called "Saltatorial reflex-spasm, a remarkable form of Spinal Irritation." Dr Gowers described two cases of the affection ('Lancet,' July and August, 1877), with an analysis of the five previously recorded. In their details these cases differ somewhat from each other, but we include them all under "reflex fatigue-spasms."

Sometimes the patient can excite the movements by pressing his feet against the foot-board of the bed while he is lying down; in one instance, tickling the skin of the soles would elicit them; in another, pressure upon a tender part of the spine had the same effect. They are often augmented by emotional excitement; and this has sometimes led to doubts as to their genuineness, since they are much less violent when no one is looking on; but the same thing may be said of many other nervous affections which are certainly not simulated.

There is sometimes pain in the contracting muscles, more often none; and there is no loss of power or of sensation. The reflex arc does not appear to begin in the skin, but in deeper structures, probably the tendons or joints of the feet.

Erb believes that saltatorial spasms will ultimately be found to be a kind of exaggerated tendon-reflex; but Gowers found no ankle-clonus in either of his cases. No doubt the movements are due to the combined and simultaneous action of peripheral and voluntary stimuli upon irritable centres in the cord; but the same stimuli are in action in all healthy persons whenever they stand upright. In Bamberger's first case, that of a youth of nineteen, the affection came on during convalescence from acute pneumonia; the spasms disappeared in about a month under the internal administration of morphia. Other cases have been much more protracted; in one instance the spasm had not subsided when the patient died of fever at the end of five years. Dr Fagge saw two cases in women, both of whom were decidedly hysterical; one had several attacks of the spasm, each lasting some months, and her recovery from one of these was sudden, exactly as happens in hysterical contractures.

The muscles of the trunk and head are sometimes affected, as well as those of the legs, but the arms seem always to escape.

This curious affection has been observed in both sexes, and at all periods of life, from childhood to old age. It has been found associated with spastic paraplegia as a sequel of myelitis; but most cases seem to be "functional," and occur with hysterical symptoms. What was called Chorea Sancti Viti Major, or dancing madness in the fifteenth century, would probably, if it appeared in a sporadic form, approach some of the cases of saltatorial spasm now seen in hysterical women (p. 880).

Saltatorial spasms, once begun, may continue for months and years, or may return after disappearing; but they do not appear to lead to further symptoms or to theaten life. No special treatment is known.

Other varieties.—Many other rhythmical spasms may be observed, chiefly in patients not suffering from organic disease. One has been called "Salaam palsy" (*Eclampsia nutans*), a habit of continually bowing the head; it has been frequently observed in children. Similar spasms may affect the arms, as in a boy once in Guy's Hospital, under Sir William Gull, who was continually moving one arm up and down (*Malleatio*). Not very unlike is the case above mentioned, in which a man used, without knowing it, to lift up one leg at intervals and strike it with the hand (p. 854); and these curious tricks have, again, relations with such scarcely morbid and quite unconscious habits as Dr Johnson's of counting the posts, and the late Prof. De Morgan's of touching the railings one by one as he walked down to his lectures at University College.

Clonic spasms may affect individual muscles of the face or limbs. When the eyelid of one side is the seat of the disorder, it has been named *pasmus nictitans* or *blepharospasmus*. Hiccough or *singultus* is a clonic pasmodic affection of the diaphragm.

Athetosis was a name given by Hammond of New York to slow rhythical clonic spasms of the hands. It has been already mentioned (p. 737), s an occasional sequel of hemiplegia.

Stammering and stuttering (*balbuties*) is due to laryngeal or labiongual spasm, or to both, and exhibits the same union of irregular and ineffectual contractions, with explosive completion of a co-ordinated function, which we have had occasion to notice in the whole group now under discussion. Some of these spasms are clearly reflex. Some resemble histrionic spasm, some saltatorial cramp, some hysterical movements, chorea major, and "ugly tricks" in children. Many are curable by discipline, or by a shock to the imagination, or by growing out of them.

In some of his cases of function-spasm Dr Mitchell found benefit from the injection of solution of atropine into the affected muscles.

Mental tic.—Charcot and his followers at the Salpêtrière, to whom is due much of our present knowledge of spasmodic tics, both simple and co-ordinated, include under the same head, two affections which appear to the writer to be far too widely separated from the cases just described to be classed with them.

The former of these they call *tic convulsif*—an inadequate name; for all tics, and certainly all co-ordinated tics, may be called convulsive. The real difference which distinguishes the group in question is that mental affections come in beside the involuntary movements. The patient accompanies the muscular spasm by an explosive utterance of a word or sentence, and this is under the influence of an imperative idea. This condition is known as *la maladie de Gilles de la Tourette*, after the physician who drew attention to it and defined its characters. He distinguishes repetition of words heard (*echolalia*), obscene exclamations (*coprolalia*), and other forms. Such cases, however, are on the borders of so-called "moral insanity," and seem to have their natural allies not with the harmless tics, but with the Dancing Mania of the Middle Ages, the convulsions of dervishes, the "jumping disease of Maine" in America, and the "Myriachit" of Siberia.

The latter group of cases, which Charcot admitted among the tics, is purely psychical. There is no muscular spasm at all, only a strong delusion, for that is what "the imperative idea" means. *Il y a des tics dans la pensee comme dans la corps* may be true; but in the body they are real, in the mind they are metaphorical.

FATIGUE-SPASMS.*—Spasmodic contractions, either clonic or tonic, may be brought about by voluntary movements. The exciting cause in each instance is limited to some one particular action, so that the patient remains quiet when at rest or when using his muscles for other purposes. One of the early papers on the subject was by Dr Weir Mitchell, in the 'American Journal of Medical Sciences' for 1876. His name for this group of affections is that of "functional spasms;" but this might be taken as meaning "spasms which are functional and not organic," in which sense it would include all the diseases described in this chapter. "Function-spasms," "Movement-spasms," and "Fatigue-spasms" (Dr Poore's term), are terms which, if less euphonious, are also less misleading.

*Spasms caused by handicrafts.**—In many cases the muscular spasms are only just sufficient to disturb the due execution of some highly specialised action, involving numerous and complicated muscular movements; and over-exercise of this manœuvre appears to produce the spasms.

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* *Synonyms.*—Functional spasms (Weir Mitchell)—Movement-spasms. Business-spasms, Occupation-spasms, and Professional-spasms are uncouth translations of the German title. This group includes most Reflex spasmodic neuroses, Saltatorial spasms, and the Beschäftigungsneurosen of German writers, viz. writers' palsy, fiddlers', tailors', cigar-makers' cramp, lock-spasm, milkmaids' cramp, etc. There is no name which would conveniently include this group of spasmodic affections: that of "co-ordinated business-neuroses," proposed by Benedikt, is somewhat unwieldy, and "occupation-spasms" does not seem quite right. "Handicraft-spasms," perhaps, might serve.

A musician becomes unable to play the piano or the violin; a telegraph clerk cannot work Morse's machine; a tailor or shoemaker can no longer sew; or a milkmaid press the milk from the cow's udder. In one case a man, whose whole occupation was to clean knives and boots and shoes for a large house, became completely incapacitated for his work, although he could still do anything else.

Two of Mitchell's cases were what he terms "lock-spasm," and these were tonic not clonic. A watchmaker, who often had to pick up and adjust very small screws, would find that his thumb and forefinger suddenly became locked upon one of them, so that he had to release it with a loop of twine, not always without wounding the finger; even then the cramp would last for some time longer. At a later period, when he was turning over the pages of a book, the finger and thumb would sometimes close with violence, so as to tear the leaf. Another man, a sawyer, was liable once or even twice a day to have the arm arrested and fixed in a state of flexion at the moment when his saw was drawn back to prepare for the downward movement. By no effort that Mitchell could exert was he able to overcome the spasm; on one occasion he made the patient bend over, so as to bring the forearm into a horizontal position, and he then found that for five minutes the biceps supported a weight of eighty pounds suspended from the wrist.

Writer's cramp.[*]—The exact kind of impairment of the power of writing varies widely in different cases of this disorder, so that hardly any two are alike. Sometimes the patient complains that after he has been writing for a few minutes his hand and forearm feel wearied; sometimes that the fingers become suddenly extended, so that the pen drops from his grasp; sometimes that the index finger is straightened and drawn off the pen-handle; or that the fingers are pressed too tightly upon the pen, so as to impede its movements. In many cases there is no visible cramp or spasm—all that a looker-on can see is that the motions of the hand are arrested in the act of writing; but in other cases there is obvious shaking of the pen.

The handwriting is often altered in character; in some instances the change consists in a reduction of the size of the letters, the power to make free strokes failing, and the writing becoming "cramped;" other patients can no longer accomplish a "running hand," and are obliged to confine themselves to a slow "round text." Dr Fagge had one patient who could write anything but shorthand perfectly well, but unfortunately he was a law reporter. In most cases, however, the peculiarity consists in an uncertain tremulous formation of the strokes which make up the letters; and in the worst cases the handwriting becomes illegible—a meaningless succession of shaky lines and curves.

Persons affected with writers' cramp adopt all sorts of odd devices. Some will write only with a quill, and so far as it goes this is undoubtedly a good plan, for the muscular effort required is much less than with a steel pen. Others employ a large, thick penholder, or fix a nib in a broad piece of cork. Dr Poore ('Practitioner,' xi) mentions the case of a man who used a rounded mass of wood of the shape of a boy's top, which he held tightly with the hollow of the palm of his hand. Others grasp a penholder with the closed fist, or fasten it to one finger by means of a ring.

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[*] *Synonyms.*—Scriveners' Palsy.—*Fr.* Crampe des écrivains.—*Germ* Schreibekrampf.—enman's spasm—Mogigraphia (μόγίς, with difficulty; γράφω, write)—Graphospasmus.

Sometimes, however, the fault seems to be not so much in the hand as in the wrist and forearm. A patient of Dr Poore's said that the first thing he noticed was a difficulty in bringing down the hand upon the paper, and for three days he forced himself to write by holding down the right wrist with the left hand, but at last that resource failed him. Indeed, one of the most distinctive and unfortunate characters of "writer's cramp" is that it is a progressive disease. For a time a person affected with it may succeed in accomplishing the art of writing by bringing different sets of muscles into exercise. Thus, instead of employing the weak muscles of the thumb and fingers, he may use the forearm; or he may place the arm in some extraordinary attitude as soon as he takes up the pen, resting, perhaps, his wrist upon the table, and raising his elbow at an angle; or pressing the limb close to the side of his body; or, again, straightening the elbow, and holding the instrument at arm's length. But if he persists in writing much all these resources soon fail him, and the muscles which he has called in to his assistance "give out" in their turn. Even if he take the pains to learn to use the pen with the other hand, that too after a time may be attacked. At advanced periods of the disease the muscles are often affected with spasmodic contractions independently of all efforts at writing. Dr Poore's patient complained that his hand would sometimes bounce suddenly out of the side-pocket of his coat while he was walking in the street, and he once broke a jug in consequence of an unexpected spasm while he was pouring out water.

In some cases the spasm extends to the muscles of the neck and body. Reynolds mentions an instance in which the effort to write caused the head to be drawn downwards to the right shoulder and the trunk to be contorted, so as to be concave on the right side. The same writer also states that he has seen, in association with writer's cramp, torticollis, occasional strabismus, stammering, and palpitation of the heart.

There is not generally any impairment of sensation. Sometimes the patient complains of "numbness," or "tightness," or "coldness," or of some peculiar sensation which he cannot define in one particular finger, or running from some part of the hand up towards the arm. In some rare cases anæsthesia has been observed. Very commonly the act of writing, if continued for any length of time, causes the hand and wrist to ache, or even the shoulder or the spine. These abnormal feelings distress the patient to an extent which seems altogether disproportionate to that of the actual pain which he has to endure. Indeed, a peculiarity is often observable in the psychical state of those who are affected with writer's cramp in its more advanced stages; they are highly irritable and excitable, and very desponding about their complaint. Dr Poore speaks of one man as having been apparently on the verge of suicide, and of another as having actually taken up a knife with his left hand and stabbed himself on the wrist. Such patients are generally "nervous," and they are least able to write when others are looking on, or when the subject-matter is of special importance. Dr Poore was told by one person that he got on better when copying than when composing what he wrote; and another patient had to watch the point of his pen, for if he looked away from it his handwriting at once became unsteady.

Ætiology.—The immediate cause of writer's cramp, and of the affections allied to it, is obviously over-use of the muscles, or working too hard and too long at that particular kind of labour which is accompanied by the spasm; and it is undoubtedly true that most of those who are attacked are clerks, or accountants, or in some way constantly engaged in writing. Moreover, as Dr Poore has remarked, the commencement of the disease often dates from the time at which some heavy task has been accomplished, as when an architect has worked against time to complete the details of a plan, or a lady to finish an etching for a bazaar. Yet some persons strain their powers to the utmost, and get through extraordinary amounts of writing, without suffering from scrivener's cramp; and others are attacked who have never written more than is safely accomplished by most persons. Dr Fagge met with two such cases: one in a boy, aged fifteen, whose education seemed to have been rather neglected than otherwise; and one in a lady of fashion, who had at most to write a few notes to her friends. Dr Poore met with one case in which three generations in a direct line were affected.

Pathology.—The seat of this neurosis, as of the other allied spasms, is doubtful. One theory, which is supported by Reynolds and by Erb, is that it lies in the cortical neurons which effect the association and co-ordination of muscular movements for the more complex actions. The nutrition of these structures is supposed to be impaired as the result of over-exertion, and the consequence is a perversion of their functions, which expresses itself in irregular spasmodic movements. A point in favour of this view is the fact that when one arm has been affected the other is often attacked afterwards; and the relations which writer's cramp bears to torticollis and other spasmodic neuroses afford a further argument.

The other theory, which was first proposed by Zaradelli (1857) and is ably advocated by Dr Poore, is that the starting-point of the morbid change is exhaustion of some particular muscle or set of muscles—generally one of the small intrinsic muscles of the thumb or fingers—which are kept constantly in action throughout the whole time that a pen is held in the hand. One or more of these, it is supposed, begins after a time to respond sluggishly to the stimulus of the will. The patient then unconsciously calls into play other muscles, generally those of the forearm. In their turn these too become worn out, and thus the process of substitution is carried on, always with the same result. Dr Poore has in a large number of cases of writer's cramp tested the electric reactions of the muscles which are specially engaged in the act of holding a pen, and has found that often one or more of them showed a marked impairment of irritability as compared with those of the opposite limb.* It might be thought that a strong argument against the theory in question is afforded by the well-known and remarkable fact that a patient who is severely affected with the disease is often perfectly able to perform other actions with his hands. But Dr Poore argues that the utility of the affected muscles for other purposes is only apparent; the hand and forearm, he says, are so bountifully furnished that for most movements there are more muscles than are required. If a man finds some of them to be weak, he unconsciously substitutes others in their place to do the work required. This writer maintains that certain among the more delicate actions of the hand are generally interfered with beside those which give to the case its principal character. Thus one of his patients could no longer hold a knitting-needle; another was unable to take up her dress between her thumb and her forefinger; and a third could not toss over a letter into a letter-box. So sufferers from writer's cramp may be unable to button their clothes, to work embroidery, or to play upon the piano, or may find great difficulty in feeding and dressing themselves.

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* Electrical irritability is generally deficient, as Dr Frank Smith found to be the case in hammer-palsy; but Dr Dubois, of Berne, has stated that in early cases of telegraph-worker's cramp faradic and galvanic irritability of the affected muscles is exalted, although afterwards depressed or lost ('Brit. Med. Journ.,' 1887, vol. i, p. 1302).

Benedikt proposed to distinguish a spasmodic, a tremulous, and a paralytic form of scrivener's cramp, but these are rather varieties in the course of the disease than separate forms.

Diagnosis.—The diagnosis of writer's cramp, or of the allied forms of handicraft- and reflex-spasm, is tolerably easy when they have reached an advanced stage. But one must always recollect that impairment of the power of writing may result from many gross lesions of the nerves and muscles of the upper limbs—progressive muscular atrophy, neuritis of the ulnar, the median, or the radial nerve, compression of one of these nerves by a tumour. Dr Poore mentions a case of subclavio-axillary aneurysm in which the first thing noticed by the patient was that he could write only with great effort, and then illegibly.

As a rule, a person affected with writers' cramp goes on for a long time without medical advice, struggling against what he deems a tiresome incapacity to carry on his duties. But if one should be consulted at a very early period, there may be great difficulty in determining whether the disease is really present or not. This is particularly the case if the patient is a nervous professional man, whose mind has for some time been dwelling on the subject; or if he is a bank-clerk, who has heard all about the complaint, and has perhaps been reading of it in medical books. Such persons come to one complaining that the act of writing causes unpleasant sensations; and one may be in great doubts whether to laugh at their complaints or to take them seriously. Perhaps the best indication is the effects of rest. If the supposed symptoms show themselves only at the end of a hard day's work, and are entirely gone the next morning or after the interval of a Sunday, they are not likely to be of serious consequence. Thus Dr Poore speaks of an eminent pianist who, after practising for a long time, found that he could not always strike the right note; but a few hours later he could play as well as ever. In such a case it is evident that nothing is seriously wrong.

Prognosis and treatment.—In most cases an early diagnosis is of the utmost importance, for on it may depend the whole future of the patient. At this period the discontinuance of all writing—or of whatever other action may be the exciting cause of the complaint—may completely cure it in the course of a month or two. Even then there is always a risk that the disease may return if the muscles should be again overworked, and as a rule the prognosis that must be given is unfavourable. At an advanced stage of the disease there is scarcely any chance of recovery, unless the patient can altogether give up his occupation for six months or for a whole year; and in many cases treatment fails entirely, the disease advancing steadily in spite of all that can be done.

The first point in treatment is giving up ordinary writing at once and altogether, letting the left hand take the place of the right, or replacing the work of either by the typewriter.

Next to rest of the overworked muscles and nerves, the best remedy appears to be galvanism. Of this Erb speaks highly, recommending the employment of ascending currents along the cervical spine, as well as peripheral galvanisation of the nerves and muscles specially concerned. The best results seem to have been obtained by Dr Poore, whose method consists in making the patient perform rhythmical movements with the affected muscles, while a continuous current of moderate intensity is passed through them. His first case was that of a man, aged thirty-two, who had suffered from writer's cramp for more than nine years, and in whom it presented its severest form. Dr Poore exercised separately the deltoid, the biceps, the triceps, and the other muscles, as well as those of the hands and fingers. When the treatment was commenced the patient could not finish writing the word George; at the end of five months he was able to write all his letters well. He had been unable to feed himself; he could now do all that he wanted. Another of Dr Poore's cases is little less remarkable. A man, aged forty, had for four years suffered from the disease, and had for six months been unable to write, the attempt to do so giving him great pain. After the first application of the current this pain disappeared, within a week he could sign his name with tolerable ease, and at the end of ten months his handwriting looked as good as it had ever been. Unfortunately, similar successes are exceptional.

Wolff's combination of galvanic treatment with massage is commended by Charcot, by Ross, and by de Watteville. Gowers strongly advocates free writing from the shoulder as a preventive and a remedy, and says that the disease is unknown among shorthand writers.

Poore also uses careful and moderate shampooing of the hand and forearm, in conjunction with a continuous weak galvanic current, applied to the affected muscles with a large moist rheophore as the negative pole. Neither massage nor galvanism should ever cause pain. Faradism appears to be useless or injurious.

No drug has an established claim to value in writer's palsy. Bromide of potassium may be occasionally beneficial by relieving the mental irritability. Stimulants and strychnia are injurious; and rubefacient liniments useless.

*Hammer palsy.**—This curious variety of handicraft spasm was described by the late Dr Frank Smith, of Sheffield ('Lancet,' March 27th, 1869; and 'Brit. Med. Journ.,' Oct. 31st, 1874). He observed eight cases among the pen, blade, and file forgers, who use a hammer of three to eight pounds weight sometimes for twelve or thirteen hours, delivering as many as 28,000 carefully adjusted strokes every day.

The patient finds that his hand has lost its cunning; he is awkward, and hits awry. Spasms of the flexors and pronators of the forearm appear, then gradual weakness of the whole limb, and finally muscular atrophy.

The writer had a typical case of hammer spasm in Guy's Hospital (Philip Ward, No. 31, 1887) in a healthy, well-nourished man of twenty-seven. He worked at nail-making with a hammer of two pounds weight, and worked by the piece. His arm, on his attempting to move it, or on the hand being grasped, was at once thrown into clonic spasms, without ain, which affected the pronators and flexors of the forearm, and also the flexors and dductors of the arm. Sensation was perfect. There were no signs of affection of the ranial or other nerves, no wasting of muscles, and no cephalic symptoms.†

In Dr Smith's cases the right leg was frequently weak; there was neuralgia or numbness, and painful spasm in the affected arm; sometimes ptosis or thickness of speech, while in one instance (No. 7) there was aphasia. For such cases he proposed the name Hephæstic hemiplegia.

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* *Synonyms.*—Hammer spasm—Hephæstic hemiplegia.

† This case was the same as that which formed the subject of a lecture by Dr Poore n the 'Lancet' for August 21st, 1886.

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Of one case he writes: Neuralgia and an indescribable centripetal feeling frequently affected the right arm and forearm. The tactile sensibility of the forearm was almost lost; electro-sensibility and electro-contractility were almost extinct. Thermal sensibility remained. The temperature in the right axilla was 1° Fahrenheit below that of the left. There were no mental symptoms; sight, smell, hearing, taste, and speech were unaffected.

Gowers regards most of these cases as ordinary hemiplegia from hæmorrhage or other gross lesion in the motor tract. But, as Poore justly remarks, fatigue certainly affects the motor nerves and motor centres in the brain as well as the muscles, and such handicrafts as striking a nail, are always volitional, like shooting at a mark or threading a needle.

The facts of spasms being followed by paralysis, of extension of motor symptoms to the leg and face, and of the affection of speech, all appear to point to the motor region of the cortex as the seat of the lesion. No autopsy has yet been recorded, and it is quite possible that the local lesion is, at least in its earlier stages, either vascular or otherwise unrecognisable after death. A "discharging," becoming afterwards a "destroying" lesion, would probably best explain the symptoms.

In any case it seems that hammer palsy is a true "fatigue" spasm or handicraft palsy, and thus an ally of writer's cramp.

Some of the Sheffield cases improved under sedatives and rest. In the case above mentioned in Guy's Hospital, Dr Poore's treatment for scriveners' cramp was tried without benefit; also Calabar bean, bromides, and succus conii raised to a two-ounce dose, and continued three times a day for nearly a fortnight. Rest in bed seemed to be most useful, but the patient thought chloral hydrate at night not only gave him refreshing sleep, but made the spasms less severe during the day.

Miner's nystagmus is also a kind of handicraft spasm. It, like hammer spasm, has been observed at Sheffield, and described by Mr Simeon Snell there, and also by Dr Dransart among the coal miners in the North of France. Working in low galleries and narrow spaces, the miner's eyes are constantly strained in a certain direction, because he cannot move his head and body as he otherwise would. The strain is soon felt, and often results in a "fatigue-spasm"—oscillation of the eyeball (like the lateral nystagmus from tumours of the cerebellum, *supra*, pp. 777-8), accompanied in severe cases by headache and vertigo, but cured at once when the patient is able to leave the mine and adopt any pursuit which does not involve the straining of the ocular muscles just described.

Beside the other handicraft spasms enumerated above (p. 861), we may mention fatigue-spasms in sawyers, bricklayers, and opera dancers.

TETANILLA.*—In the disorders hitherto described, the spasms are of a clonic kind. But in another member of the group tonic spasm is the essential symptom. This is the disease to which Lucien Corvisart in 1852 gave the name of *tetanie:* previously it had been described by Dance in 1831 as "a kind of intermittent tetanus;" and Steinberg had called it "idiopathic" or "rheumatic contraction of the extremities." Trousseau's lecture on this curious affection drew attention to it in this country, and Dr Moxon recorded case in the ‘Guy's Hospital Reports' for 1870. It differs pathologically nd clinically from the terrible disease always known as tetanus, and now nown to depend on infection by a microbe (p. 373).

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* *Synonyms.*—Tetany—Tonic cramps of the fingers and toes in children—Rheumatic contraction of the extremities—Remittent tetanus.—*Fr.* Tetanie—Tetanille—Contracture rhumatismale des nourrices (Trousseau).—*Germ.* Tetanie.

Tetany, or tetanilla, is generally confined to the limbs, and affects hiefly their distal parts. Sometimes it is limited to the forearms and ands; more rarely to the legs and feet. As a rule it is bilateral, and may ivolve all four extremities at the same time or alternately. Occasionally ie muscles of the face, the trunk, and even those of the eyes, may be fected.

The patient first has a sensation of tingling in the parts which are to affected, and then begins to find that their movements are no longer free. on the thumbs become forcibly adducted; the fingers of each hand are sely pressed together, and are half flexed at the metacarpo-phalangeal ints, but extended beyond; the palms are hollowed by the approximation their inner and outer surfaces. Hence Trousseau's comparison to the nical form of the accoucheur's hand when introduced into the vagina to rform turning. The thumbs may be driven into the skin so violently as leave marks, or even (it is said) to give rise to sloughs. The wrists are lf flexed, the forearms pronated, and the arms drawn close to the sides. the lower limbs the toes are bent down and adducted, the soles of the et are hollowed, and the heels are pulled up by spasm of the muscles of ie calves into the position of talipes equino-varus.

The contracted muscles feel hard; their resistance may be overcome by ie employment of some force, which generally gives the patient pain, but metimes relief. Thus the fingers and thumb may be straightened; but soon as they are released they again become flexed as before. Another ay of arresting the spasms is by means of cold. Trousseau mentions that ersons in whom the lower limbs are affected can often regain the free use them, for the time, by merely standing with naked feet on a stone floor; nd the same result can be brought about by immersing the hands and rearms in cold water.

During the paroxysms the movements of the affected parts are much npaired. If, as is often the case, the patient is a mother nursing her hild, she cannot hold it in her arms. There is partial anæsthesia, so that ie patient cannot judge of the size and hardness of objects. In walking feels as if his feet were treading on a thick carpet. Pain is sometimes together wanting, but it is usually present in more or less severity, and is mpared to that which accompanies ordinary "cramps" of the legs. nother common symptom is that the backs of the hands and feet become dematous, and sometimes red, with swollen veins.

In from five to fifteen minutes the spasm generally passes off, but somemes it lasts without stopping for an hour or two, or even longer still. As subsides, sensations of formication are again experienced.

After a variable interval another attack commences, and this goes on for veral days, or sometimes for two or three months. Even after a long eriod of health the disease may return. A child, whose case is recorded y Moxon, was first attacked by tetany when five months old, and had it n five distinct occasions, at intervals of from five to twelve months, efore it reached the age of three years and a half. One of Trousseau's atients, a young man of twenty-one, had the disease every winter for four ears, the contractions coming on every day during a period of two months.

Trousseau found that in a patient affected with tetany one can at any time bring on the spasms by compressing the principal nerve or artery in the upper part of the limb.

The electrical relations of the motor nerves and muscles in this disease have been investigated by Erb ('Ziemssen's Handbuch,' xxii, p. 345), previously by Kussmaul and Benedikt, and more recently by Chvostek, Frankl-Hochwart, and Weiss, with the result that the excitability both to faradic and to galvanic currents has been found greatly increased except only where the facial nerve is distributed (see Dr Buzzard's observations, 'Clin. Lectures,' pp. 414, 417). We found in two of the cases mentioned below that the closing contraction was apt to cause physiological tetanus instead of an ordinary contraction. Chvostek has also observed that in these cases a slight tap over the facial nerve on the cheek is followed by contraction of the muscles it supplies.

The following summary of cases of tetany admitted into Guy's Hospital between 1868 and 1888 was extracted out of our clinical records by Mr Lefevre, and those between 1888 and 1898 by Mr W. S. Richardson for the present edition:

1869. M., æt. 6. Swelling of right foot, hot and tender; history of cramps *before* admission only. Relieved.
1870. M., æt. 3, with condition of talipes equino-varus. *Intermittent tetany*. Relieved.
1872. M., æt. 18, with cramps in calves and hands. *Intermittent tetanilla*. The diaphragm was also involved. Improved.
1873. M., æt. 2. Hands cone-shape; feet extended. Slight opisthotonos. Cured.
1876. M., æt. 6. *Tetany* associated with rickets and diarrhœa. Cured.
1883. F., æt. 10½. Right hand only affected; thumb flexed and adducted; fingers semiflexed; hand drawn to ulnar side. Rapidly disappeared.
1887. M., æt. 5. Hands and feet affected. Fatal by dyspnœa.
1888. Brother of above, æt. 7. Hands and feet, with much pain. Mitral regurgitation. Slowly recovered.
1889. M., æt. 4. Upper and lower limbs, painful. History of fits and convulsions. Associated with bronchitis. Recovery.
1890. M., æt. 18. Upper extremities. Previous amputation for morbus coxæ. Chronic suppuration. Lardaceous disease, albuminuria, and diarrhœa. Death.
1890. F., æt. 31. Spasms in hands, ascribed to washing with cold water, three months after delivery. Associated with mental disturbance. Discharged without much improvement.
1890. F., æt. 4. Upper and lower limbs. Associated with rickets, stomatitis, and diarrhœa. Cured.
1891. F., æt. 3. Tetanilla. Left the ward relieved.
1894. F., æt. 17. Fingers and thumbs of both hands. Associated with hysteria and choreiform movements. Improved under treatment.
1896. F., æt. 5. Carpopedal spasms; worse in hands than in feet. Associated with rickets. Relieved.
1897. F., æt. 22. Hands and feet; after removal of thyroid. Relieved.
1898. M., æt. 14 months. Hands. Vomiting and diarrhœa. Cured.

The third of these cases, in a boy three years old, was described by Dr Wilks in a paper in the 'Guy's Hos. Rep.' for 1872, and in his lectures he narrates two others in youths of sixteen and eighteen. The former was a private patient; the latter was the third of the above series.

The writer had two boys, brothers, under his care within a few months with the typical symptoms of tetanilla (Cases 7 and 8). One aged five, in Mary Ward, developed severe symptoms. The spasms became general, he lost his power of speech, and his temperature rose. After further local and general treatment, he died, September, 1887, the illness having lasted about three weeks. There were no lesions discovered after death.

His elder brother, aged seven, was admitted into John Ward, November, 1888, with the same painful tonic spasms of his hands and feet. He slowly and gradually recovered, and at last went out well in February, 1889. In neither of these cases were there any clear signs of rickets.

In 1890 we had a ninth case in Mary Ward in an adult which resembled tetanilla in its localisation, and in being in all probability functional (Case 11). A woman of about thirty was suddenly attacked, as the result of strong and sudden emotion, by tonic contractions of her fingers and toes. This had lasted several months when she came under the writer's care, and two fingers of one hand had become contracted. The other hand was nearly well, and the toes could be flexed, though with some pain. The case might be called carpo-pedal spasms in an adult. It had the character of an "hysterical" disorder.

In tetany the spasms are not always limited to the extremities. Trousseau describes instances in which the face and trunk were also affected: the face became distorted, the eyes squinted, the sterno-mastoidei and the pectorales were rigid, and the recti abdominis stood up like tense cords. The jaws were firmly clenched, and the speech was impaired. Laryngeal pasm sometimes occurred, causing lividity and apparent danger of suffocaion. Febrile disturbance was also present. Yet even in such severe cases, he patient would often get up; and if an adult she would attend on other patients, although suffering from pains in the loins, and feeling bruised and xhausted.

Sooner or later recovery almost always takes place. Trousseau menions one instance in which death occurred from phthisis during a relapse of tetany, and another which seems to have ended fatally within a few hours of its commencement. This can scarcely have been really a case of tetanus, for the hands and feet were characteristically affected; and these are the very parts which escape the spasms of tetanus.

Varieties.—It appears that a very common affection is really a minor variety of tetany, namely, "*carpo-pedal contractions*," or "turning in" of the thumbs and great toes, which one so often observes in rachitic children. Dr Jackson regards these as rudimentary epileptiform convulsions; but surely they are more closely related to tetany.

Another condition closely allied to tetanilla is *Laryngismus stridulus*, a spasm of the muscles of the glottis common in young children who are the subjects of rickets (cf. p. 537). Notwithstanding occasional extension to other parts, tonic spasm of the fingers and toes is the distinctive feature of tetany, and this approximates it to another affection of early life, *Trismus neonatorum*. It occurs in infants a week after birth, and sometimes even in the first twelve hours. There is clenching of the hands, flexion of the feet upon the ankles, and bending of the toes; and when the fit subsides the child still lies with its head drawn back, its hands clenched, and its thumbs drawn into the palm.

The frequently fatal result and the presence of opisthotonos seem to separate trismus neonatorum from tetany. But tonic spasm of the back of he neck is common in children with "carpo-pedal contractions."

The relation of trismus to tetany is confirmed by its ætiology. The trismus of newly born children was at one time supposed to be a traumatic tetanus, excited by irritation which started from the umbilical cord. But that notion was refuted by the subsidence of the disease in the Dublin Lying-in Hospital, in consequence of the introduction of an effective system of ventilation. Previously one in every six of the infants born there had died when less than a fortnight old; and nineteen deaths out of twenty were due to trismus. Afterwards the mortality was only one in fifty-eight and a half, and but a ninth part of it was from the disease in question. In the West Indian islands it is still common, and also in St Kilda; probably it is due to a vitiated state of the air in the lying-in chamber. In London it must be very rare, for Dr West saw only one case.

Ætiology.—In a valuable paper in the 'Lancet' (May, 1887) Dr Cheadle relates a case of tetany in an adult, and remarks on the association of this disease with previous diarrhœa or lactation in women. He believes that rickets in infants is apt to produce various spasmodic affections, which sometimes take the form of infantile convulsions, sometimes of carpo-pedal contractions, sometimes of laryngismus stridulus, and sometimes, though less commonly, of tetany. Dr Eustace Smith and Dr Goodhart take the same view; and the latter writer quotes Mr Hutchinson's observation of the frequent coincidence of zonular cataract with infantile fits and rickets. Rickets has been present in most cases of tetany seen at Guy's Hospital.

The majority of cases of tetanilla seen in England occur in children. In 142 collected from various sources Dr Gowers found 42 between one and ten years of age (and of these 34 under five), and 36 between ten and twenty; making 78 patients under twenty, to 64 adults. Of the latter the majority were between twenty and forty (34 women to 13 men), 13 between forty and fifty (8 women to 5 men), and 4 (all men) above fifty. In children it is more common in boys than in girls (in 42 cases under ten, 31 males to 11 females). In adults it is more common in women than in men (22 males to 42 females). It occasionally occurs in families.

The geographical distribution of tetanilla is remarkable for its variation. It is most prevalent in the winter months.

Trousseau speaks of tetany as occurring most frequently in women between the ages of seventeen and thirty, particularly in those who are suckling their children. One is tempted to think that there may be something in the spare diet adopted during the puerperal period by French physicians which brings on a disposition to the disease. Trousseau thought that tetany is the direct result of exposure to cold, but he regarded diarrhœa as a frequent predisposing, and enteric fever and cholera as an occasional, cause of tetany. He had seen it in women over forty years of age, and even in adult males. Tetany has been observed as a sequence after removal of the thyroid,—in seven out of seventy cases according to Wölfler. It has also been observed in cases of dilatation of the stomach.

Tetany sometimes occurs as an epidemic neurosis in girls' schools and in convents, as in the school at Gentilly in 1876. Such cases bring it into relation with hysteria, chorea major, and the extraordinary attacks of "convulsionnaires." But it has also been seen among youths during severe winters in Germany.

Moxon remarked that the disease which has been called *spasmodic ergotism*, and which was prevalent in certain parts of Germany in the earlier part of the present century, was similar in its symptoms to tetany. Wright's account of it ('Edin. Med. Journ.,' 1839) almost proves that the two disorders are identical, and that the former was not due to a specific poison, but to general deterioration of the health from a defective supply of nutriment. He states that in the eighteenth century a similar disease occurred in Sweden. (See also on this point a paper by Bauer in the 'Berliner klin. Wochenschr.,' 1872.)

It is difficult to explain the various ætiological conditions of this curious disease, and more than one physician has proposed to exclude from the definition the severe cases of trismus, or the slight ones of carpo-pedal contraction, or tetanilla in infants, or tetanilla in suckling women. Probably, however, we may look on rickets in young children, on cold at all ages, and on chronic diarrhœa or gastric dilatation, child-birth and lactation, as predisposing causes of tetanilla, and seek its efficient cause in some toxic influence on the nervous centres in the cord.

This poison may be the result of acute specific fevers, of which tetanus is often the sequel; and its influence may be increased by the lowered power of resistance produced by cold, especially in young children. Some such toxic effect seems to be shown by the cases of tetanilla which follow removal of the thyroid, usually separately, but in some cases accompanied by symptoms of myxœdema. The same toxic hypothesis would bring under the same category the cases of ergotism just referred to, and cases apparently due to poisoning by alcohol or by lead.

The immediate *pathology* of tetany is quite unknown; and it has no ascertained morbid anatomy. In the fatal case of the writer's referred to above, the brain and spinal cord were both normal to the naked eye, and after hardening and staining nothing abnormal could be found. Dr Risien Russell has carefully described the various conditions of the brain and cord met with after death, but none of them are constant or characteristic. The disease seems to be a functional one, but the seat of the disturbed functions would appear to be in the neurons of the anterior cornua of the cord, and the reflex arc connected with them.

Tetanilla has no affinity whatever with rheumatism in any intelligible sense of the word; all that French and German writers mean by calling the contractures "rheumatic" is that they often follow exposure to cold. Dr Hughlings Jackson suggests the hypothesis that cerebral influence is diminished, and that (supposed) cerebellar influence over the motor nerves is increased.

The *diagnosis* of tetany is not difficult, spinal meningitis, especially epidemic cerebro-spinal fever, being the only affection with which it is likely to be confounded. There is no rise of temperature in tetany, and when it occurs epidemically it is obviously a neurosis, dependent on a predisposing epidemic fever, or upon the contagion of example.

The *prognosis* is favourable in genuine cases, but death may occur from general convulsions or other complications to which rachitic children are liable. In adults the only grave cases are those in which the stomach is dilated.

The unfortunate habit of naming symptoms after their describers has affixed the name of Trousseau to the fact that the paroxysms of tetany may be produced by compressing the nerves of the limb; that of Chvostek to the ready contraction of a muscle on percussion; and that of Erb to the remarkable electrical phenomena of a more ready anodal than cathodal response, and the frequent appearance of tetanus instead of a single clonic spasm. They are, however named, important diagnostic points.

The *treatment* which Moxon adopted in his case of tetany was the administration of bromide of potassium in five-grain doses; in four days the spasms ceased to return, and the child afterwards took cod-liver oil and steel wine. There is little question that such remedies are to be preferred to the bleeding and cupping advised by Trousseau. Indeed, that writer himself makes an exception for weakly and debilitated subjects, for whom he recommends quinine with small doses of opium or belladonna; but cod-liver oil and good food are the best medicines. He mentions a case which Aran cured by applying to the affected parts pieces of linen soaked in chloroform. Wilks also found cold applications the best. Internally chloral has been found very useful at Guy's Hospital, and in severe cases the inhalation of chloroform has given marked relief.

Dr Cheadle believes that Calabar bean will sometimes succeed in overcoming the spasms when other measures fail. Warmth for infants, and fresh air and exercise for adults, are probably the best treatment in many cases. For infants chloral hydrate (with or without bromides), followed up by good milk, cod-liver oil, meat juice, and other nutritious food, will in most cases cure by removing the cause as well as the symptom, in cases of tetany, or carpo-pedal contractions, or trismus.

When there is degeneration or absence of the thyroid body, the treatment is, as in myxœdema, to supply thyroid extract. When gastric dilatation is present, this determines the treatment.

THOMSEN'S DISEASE.*—The first full account of this rare and remarkable spasmodic affection was given in 1876 ('Arch. für Psychiatrie u. Nervenkr.') by Dr Thomsen, a Danish physician, who was himself, with several members of his family, the subject of the infirmity. In the same year Seeligmüller published a characteristic case. Others have been observed in Germany, France, Denmark, and Italy, and Dr Buzzard described two cases in England ('Lancet,' May 14th, 1887). Erb published a monograph on the disease in 1886. A full bibliography was given by Dr Paul Chapman in the sixth volume of 'Brain' (April, 1883), and by Dr Hale White in the same periodical for April, 1886, and in the 'Guy's Hospital Reports' for 1890.

Symptoms.—The peculiar features of the affection are that when the patient endeavours to perform voluntary movement after rest, the muscles of the limbs are thrown into tonic spasms, which prevent movement and keep the parts in a state of stiffness, like that of cramp, though without pain. As in ordinary cramp, the contractions, though violent, are ineffective, portions only of the muscle being raised into bulging elevations. After a time the difficulty is overcome, and the patient is able to rise from his chair, and walk, run, or perform any action he desires. But the difficulty returns again and again, so as seriously to interfere with his daily life. Seeligmüller's patient was a healthy young recruit, who found himself slow and awkward in performing his military exercises.

Not only the arms and legs, but the muscles of the trunk, are subject to this difficulty, and also those of the tongue and lips, so that there results a kind of stammering—a violent effort to speak, inability to do so, and at last an explosive utterance, followed by ordinary articulation for the rest of the sentence.

The muscles supplied by the portio dura and those of the eyeball and the larynx seem always to escape. Moreover the involuntary muscles of respiration and micturition and the several sphincters, are quite unaffected.

There is sometimes a feeling of numbness or "pins and needles" in the limbs, and occasionally pains, but these are in some cases absent, and are never severe or long continued. There is no anæsthesia.

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* Called by Thomsen "Tonic contractions in voluntary muscles, as the result of inherited psychical predisposition." Strümpell and Erb call it "Myotonia congenita."

The knee-jerk and plantar reflexes are usually present, and sometimes excessive; but in one of Dr Buzzard's cases the former was completely and the latter almost completely abolished. The galvanic and faradic irritability of the muscles is greater than normal, and they readily respond to mechanical stimuli, but contract and relax very slowly; this is called the myotonic reaction. The nerves respond normally; there is no reaction of degeneration. Fibrillary tremors have often been observed, and the contraction of a muscle after stimulation is slow.

There is no atrophy of the affected muscles. On the contrary, they undergo true hypertrophy, especially, it would seem, the gastrocnemii, external vasti, glutæi, and trapezii, but always in a symmetrical manner. Yet the muscular strength of the patients is not at all proportionate to this apparently athletic development.

Some authors have described the mental faculties of the patients as deficient, but there seems no reason to regard this as a character of the disease, and it was one of these patients who taught us how to recognise it. The general health is unimpaired, and there is usually neither pain nor disability for movement, but the patient feels awkward and unready; before doing anything he must get his muscles prepared, and hence he is incapable of prompt and rapid movement in response to the will.

We had a typical case of this curious disorder in Guy's Hospital, in a healthy and well-made young Welshman. He was apparently strong, and of more than average intelligence, but he could not readily make the start in walking or rising from a chair. A full account of his case is given by Dr White in the 'Guy's Hosp. Rep.,' vol. xlvi.

Pathology.—The primary change might be in the motor nerves, in the motor tracts of the cord, in the motor cerebral cortex, or in the muscles. Fragments of muscles have been removed by the "harpoon," and the fibres are found to be thicker than usual; but the striation is unaffected, and there is no fatty overgrowth, as in pseudo-hypertrophic paralysis. ('Guy's Hosp. Rep.,' vol. xlvi, pl. iii.) No autopsy was made on a case of Thomsen's disease for twenty years after its discovery; but in 1896 Déjérine and Sottas ('Revue de Med.,' Mars, 1895) made one on a man of thirty-two who had shown all the characteristic symptoms and who died of acute Bright's disease. The brain, cord, and nerves were found to be healthy. The seat of the lesion was in the muscular fibres. Their diameter was increased up to 80 or even 150; the shape was less polygonal; there was a marked hyperplasia of the nuclei, and some fibres were atrophied, while others contained vacuoles; in parts the longitudinal striation had disappeared, but the transverse striation was well marked. The connective tissue showed no changes, and the adipose tissue was not increased.

Dr A. McL. Hamilton, of New York, and some other writers, have argued against the symptoms of Thomsen's disease being more than accidental or secondary. Dr Thomsen himself, as appears by the title under which he described it, regarded the affection as rather mental or emotional than organic in origin.

Ætiology.—The reality of the disease as a recurrent combination of symptoms is, however, established by its family character. Dr Thomsen saw signs of it in his own children while still in the cradle; his own mother was slightly affected, and two of her brothers typically so; of her thirteen children, six, besides Dr Thomsen himself, were subjects of the infirmity; and of his own five sons, four inherited it from their father.

Benedikt, Leyden, Eulenburg, Pitres, Erb, all found the same hereditary and family distribution. It is more constant than in any other affection of the nervous system, except perhaps Friedreich's disease (p. 707); and like that rare and obscure condition, its duration is more extended laterally than vertically, *i. e.* it affects many brothers and sisters in a family, but does not so readily pass to the next generation, and rarely to a third.

Both sexes are liable to be affected, but males much more frequently. The disease is probably congenital, but the symptoms first become obvious when a boy goes to school and finds himself unable to take part in games,—in a case reported by Dr Banham, of Reading, this was not before the age of twelve.

Course and prognosis.—The symptoms do not appear to be aggravated after they are once fully developed, but there is no instance of their having disappeared, nor is any treatment even asserted to be efficient. The condition is probably as innate and ineradicable as the individual timbre of voice, or tricks of speech and gesture; and, like them, if ever "grown out of," the result is not likely to be obtained by direct therapeutical means, but by the gradual effect of circumstances and of time.

PARALYSIS AGITANS.*—Unlike the preceding spasmodic disorders, the curious malady called shaking palsy (in distinction from the 'dead palsy' of hemiplegia) has been known for centuries. But its true clinical features were only recognised by Parkinson in 1817.

The tremors.—The most striking symptom consists in oscillations usually affecting the limbs, sometimes the neck and the tongue also, the head or eyes very seldom. It often begins in one of the arms, sometimes in a leg. Charcot says that it may for a time be limited to one thumb. These tremors continue while the affected part is at complete rest—an important distinction from those of Insular sclerosis (p. 711). The movement is at first very slight, a mere tremor; but gradually the amplitude of the oscillations becomes greater and the whole limb is involved, so that the patient is unable to go on with his work, and has to seek medical advice. Sometimes, however, the agitation of the muscles is severe from the very first. In two cases under Dr Fagge's care the patient went to bed as usual and woke in the morning with the disease fully developed, the right arm being in one instance the part affected, in the other both the left arm and the left leg. In two other cases at Guy's Hospital one side of the neck was the earliest seat of the movements. After a variable interval—perhaps after some months—the oscillations appear somewhere else, most frequently in the leg of the same side as the affected arm, sometimes in the other arm. Sooner or later the remaining limbs suffer in their turn, but the tremors of the leg are easily overlooked, particularly while the patient is sitting in a chair. The head also may now begin to shake, even when no tremor is communicated from the trunk and limbs, and the tongue becomes very tremulous when it is protruded; but nystagmus, or oscillation of the eyeballs, seems never to occur, nor do the jaws take part in the movements except in the last stages of the disease.

During the early part of its course paralysis agitans is almost always paroxysmal. Each attack lasts some hours, and it is followed by a feeling of great fatigue, which gradually passes off in the interval of rest before the next fit begins. At a later period the trembling becomes continuous. The shaking of the limbs follows a regular rhythm, with, according to Gowers, a range in the fingers of only ⅛ to ¾ inch, and a rate of five to seven vibrations in the second: but it is liable to acceleration on mental disturbance or voluntary efforts.

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* *Synonyms.*—Shaking palsy—Paralysis cum tremore—"Parkinson's disease"—Scelotyrbe festinans—Chorea procursiva, senilis v. festinans.—*Anglice*, The trembles.—*Fr.* Tremblement paralytique progressif.—*Germ.* Schüttellähmung.

The tremors are independent of voluntary motions of the affected parts. Mr Parkinson began his monograph by defining it as follows:—"Involuntary tremulous motion, with lessened muscular power, *in parts not in action*, and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace; the senses and intellect being uninjured." He went on to distinguish the disease from the tremor of old age, as well as from that caused by the vapour of mercury.

During the early period of the disease the hand may become perfectly steady when it takes up a tool or a pen, and the patient is able to control the agitated limb by an effort of will. At this stage the affection is usually dormant so long as the patient lies quietly on his back in bed, and no movements occur during sleep except in the most advanced stage of the disease. Parkinson mentions that one of his patients had an intercurrent attack of right hemiplegia, in which the face was drawn to the left side, and this lasted a fortnight; during that time neither the arm nor the leg on the paralysed side was in the least affected with the tremulous agitation, but it returned as the limbs regained their power. In the later stage of shaking palsy the attempt to perform any voluntary movement brings on the oscillations with greatly increased violence. They are also much augmented by emotion or excitement. Even the presence of a looker-on often affects such patients to a remarkable degree; a man who can write well when alone may be unable to form a letter so long as he is watched by a clinical clerk. If we hold the agitated limb, so as to check its movements, they are aggravated; and if we forcibly restrain them, similar movements appear in the opposite arm or leg.

Paresis.—According to most writers there is in paralysis agitans, beside the tremors, a certain degree of impairment in the muscular power of the affected parts. Eulenburg, however, has found that even in severe cases of long standing the reaction to induced or galvanic currents is perfect, and both Trousseau and Charcot taught that the muscles retain their full force. Another point observed by Trousseau was that the contractility of the muscles was quickly exhausted; when told to open and shut his hand in quick succession the patient at first did so rapidly, but after fifteen seconds more slowly, and soon not at all. Charcot remarked that there is often a retardation of the influence of the will, shown by an unduly long interval between a thought and its expression in words.

M. Bourneville, the editor of 'Charcot's Lectures,' has since made observations on six patients with the dynamometer, which appear to show that their strength was really diminished to a considerable extent.

In most cases the cutaneous sensibility of the affected limbs is unimpaired. Charcot, however, says that a feeling of pins and needles in the hands and feet is sometimes complained of; and this was the case in a patient of Dr Fagge's, who also said that he could not feel the ground. Headache and vertigo are not uncommonly present: and at an advanced period of the disease there may be loss of memory.

Tonic spasms.—Perhaps the most constant symptom of paralysis agitans is *rigidity* in the muscles of the affected parts; and this causes an attitude which is characteristic of the disease. The head is bent so that the chin approaches the sternum, and the patient can with difficulty raise or turn it to the left or right when standing. The body is bowed forwards, the elbows are drawn slightly away from the chest, and partially flexed; the hands present a deformity somewhat like that which occurs in osteo-arthritis, the three inner fingers being inclined towards the ulnar side of the hand, while the thumb and forefinger are stretched out and brought close together as in holding a pen. In the lower limbs the rigidity is sometimes very marked; they are semi-flexed, the knees are brought together by a movement of adduction, the feet are curved inwards as in talipes equino-varus, and the toes are arched like the three fingers.

Articulation is unimpaired until the last stage, but the speech is slow and jerking, as though a considerable effort of the will were needed for the pronunciation of each word. Charcot compared it to a bad rider trying to talk when mounted on a high-trotting horse.

Festination.—The way in which the head and body are bent forwards in the more advanced stages of paralysis agitans accounts for a symptom which Parkinson noticed, namely, that the patient tends to fall upon his face when he attempts to walk, and that his steps are consequently hurried, so that he runs instead of keeping to his ordinary pace. This had, indeed, been previously described by Sauvages, but as a separate complaint, under the name of *Scelotyrbe festinans.*[*] The man seems, as Trousseau said, to be constantly trying to overtake his own centre of gravity. Parkinson mentions a case in which an attendant was obliged to walk backwards in front of the patient with one hand on each of his shoulders to prevent him from falling. When he first gets up from his seat, which he does very slowly, the patient makes a few hesitating and ineffectual steps before he seems to be able to start off, and in walking he treads only upon his toes, being unable to bring his heels to the ground. In some cases there is great danger of falling: and in an old gentleman whom the writer saw after many years of paralysis agitans, this occurred more than once, and was always followed by aggravation of the symptoms.

But the most remarkable symptom is that some persons affected with paralysis agitans walk backwards without intending it and when they mean to go forwards. Some years ago a striking instance of this occurred at Guy's Hospital. The patient, after a few ineffectual efforts to rise from his chair, would stand up, pause, make two or three abortive attempts at starting, and then succeed in walking a few steps towards the door, when suddenly he found himself hurried against his will backwards into the umbrella-stand in the corner of the room. It was like the sudden reversal of the engines of a steamboat. Charcot met with a case in which he could at any time induce such retrograde movements by unexpectedly giving a gentle pull at the patient's dress when she was standing up, and Dr Buzzard remarked exactly the same thing in an elderly woman, whose appearance and symptoms he graphically describes in his lectures. This symptom has been called "retropulsion," in contrast to the more frequent festination or "propulsion."

In the more marked cases of paralysis agitans the movements of the hands are not mere tremors, but are to some extent co-ordinated. The finger and thumb move as if the patient were rolling a cigarette paper, or crumbling a morsel of bread.

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[*] Σκελοτύρβη (disorder of the legs, staggering) occurs in Strabo, Pliny, and Galen. The term has been also applied to chorea.

Profuse sweating is an almost constant symptom when the oscillatory movements are at all severe. The patient may become so bathed in perspiration as to have to change his clothes many times a day. He often suffers greatly from a feeling of heat, especially about the epigastrium and in the back, so that he insists on being kept lightly covered; but occasionally local chilliness is complained of. With the thermometer the temperature is almost always found to be normal.

The features are immobile, and the face has a remarkable impassive expression, like a mask, as Parkinson described it. The voice is often shrill and piping. Dr Buzzard believes that this change of an old man's voice to a "childish treble" is an almost decisive symptom of shaking palsy. (See the quotation at the head of the chapter.) The progress of shaking palsy is exceedingly slow, with intervals of apparent improvement.

Pathology.—Paralysis agitans should be regarded as an affection of the cerebral cortex. Parkinson supposed its seat to be in the cervical cord and the bulb. But the cessation of the oscillations during sleep would alone disprove this view. Hitherto anatomical observations have thrown no light on the question. Dr Fagge made one autopsy, on a woman, aged forty, who died of phthisis after having suffered from paralysis agitans for eight years; for a year her speech had been impaired, and at last it was unintelligible. No serious change was discovered, and the more effectual modern methods of research have also failed to find any lesion. Slight thickening of the cortical layer was found by Cayley in the cord of a patient of Murchison's, aged seventy-one, who died of typhus after being twelve years the subject of paralysis agitans ('Path. Trans.,' 1871, p. 24). Charcot was able to examine the nervous centres in six cases: in three they were perfectly healthy; in three they presented slight microscopical changes, which were probably merely senile. In some cases there has been an obvious atrophy of the brain, but not more than is often found in persons of the same age who were free from symptoms during life. Eulenburg and subsequent writers place paralysis agitans among functional neuroses. Gowers regards it as a premature senile decay.

It is by no means a common complaint. During the ten years from 1866 to 1875 only fourteen cases were admitted into the wards of Guy's Hospital, from 1876 to 1885 eighteen, and from 1886 to 1889 eight.

Age.—Parkinson believed that paralysis agitans seldom occurs before fifty; but among the forty cases at Guy's Hospital there were twenty-one in whom it began at an earlier age, and in nine of them before forty. Of these nine, four were thirty-six, one thirty-two, three thirty-eight, and the youngest was but twenty-one years old. This last instance is not without precedent; for Charcot mentions a case of Duchenne's in which the patient was only twenty, and another which occurred in a girl of fifteen or sixteen who had been terrified by a bombshell during the siege of Paris. In thirteen of our forty cases the disease began between the fiftieth year and the fifty-ninth, and there were six in which the patient was still older when first attacked, the age varying from sixty-one to seventy-three. The majority of cases begin between forty-five and sixty-five.

Sex.—Charcot thought that paralysis agitans is as common in women as in men; but in all Parkinson's cases, and in twenty-nine out of our forty cases, the patients were of the male sex, and this is the general experience. Gowers finds the proportion two to one. The early cases are almost all in men.

Origin.—The disease seems frequently to arise without any definite exciting cause; but sometimes it follows close upon some violent shock of terror or other emotion. Charcot says that in many of his female patients it developed itself during the political commotions so frequent in France. The prolonged action of cold and wet has been thought occasionally to give rise to paralysis agitans, but without satisfactory grounds. It has sometimes followed a local injury, as in Charcot's case of a lady who severely bruised her left thigh in falling from a carriage, and in whom shortly afterwards that leg began to shake, and at a later period all the other limbs. The fact is that we are quite ignorant of the cause or even the conditions of shaking palsy. It is certainly more than premature old age, as some have supposed.

Diagnosis.—The recognition of paralysis agitans is seldom difficult. A disease which was formerly confounded with it is the insular or disseminated sclerosis described in a previous chapter; but the points of distinction between them are clear (cf. p. 713). All experienced observers depend most upon the characteristic facies of the disease, but admit that even this may occasionally leave room for hesitation.

In the tremors of old age the head shakes when the patient is sitting at rest, and the hands when he moves them. But Dr Fagge doubted whether a strict line of separation can always be drawn between paralysis agitans and the tremor which is so common in old people, and which (as Dr Maclachlan found among the inmates of Chelsea Hospital) has little or no tendency to shorten their lives. He mentions one case in a pensioner, aged 107, who had been affected with it ever since he was sixty. Possibly the same affection, which in young subjects is progressive, is in older persons stationary, or runs so slow a course that death overtakes them before it has time to develop itself fully.

One must not forget that local organic disease of the brain—a tumour, for example—may give rise to paroxysmal attacks of spasm in one arm, or in one arm and leg, which are not unlike those of the commencement of paralysis agitans; but the history of the case and the other symptoms will generally prevent one from making a mistake in this direction.

Hysteria may simulate this disease. Thus, in a girl of eighteen, the right arm began to shake three weeks after a fright. When she was admitted into the hospital it was in a state of continuous agitation; and if it was held, the other limbs began to move in a similar way. However, she had a screaming fit the very day after her complaint began, with globus and headache. She was treated with static electricity, and quickly [illegible]covered.

Prognosis.—Paralysis agitans is a disease which runs a very slow course, sometimes lasting thirty years. Even at an advanced period of the disease improvement sometimes takes place; but towards the end the patient, as rule, is bedridden and palsied. The movements, which are now incessant, at least while he is awake, may be so violent as to shake the bed in which he lies. He is unable to get up or to dress or feed himself without assistance, his speech becomes unintelligible, the saliva runs from his mouth, and he loses control of the sphincters. Bedsores may form in the last stages, or he may die of hypostatic congestion of the lungs, if not carried off at an earlier period by pneumonia or some other intercurrent disease. Charcot remarks that a few hours before death the movements cease entirely.

Treatment.—This is most unsatisfactory. Elliston supposed that he cured a case with chloride of barium. In three of our cases the last-named salt was given in doses of gr. ½ to gr. j.; and although in two it seemed useless, in the other marked improvement took place, the patient (who was under Dr Moxon's care) being able within six weeks to walk twice the length of the ward merely holding a nurse's hand, whereas he had been so helpless as to be unable to get in or out of bed, and for five years was dressed by others. One patient, after taking two grains of valerianate of zinc three times a day, went out of the hospital "cured," but the noise of a passing waggon, as he was walking home to Bermondsey, brought back the jerking movements, which had ceased entirely for several days. Dr Ramskill (Syd. Soc.'s translation of 'Trousseau,' vol. i, p. 499) had a well-marked case in which recovery occurred, after the failure of other treatment, during the administration of four minims of phosphorised oil of the Prussian pharmacopœia in a drachm of cod-liver oil three times a day. At Guy's Hospital phosphorus, nitro-glycerine, and physostigma, have all been given without benefit.

Eulenburg has tried galvanism and found it useless. He recommends the subcutaneous injection of liquor potassæ arsenitis diluted with two parts of water, in quantities corresponding to from 1½ to 2½ minims of Fowler's solution. But Charcot, who advised statical electricity, made trial of this treatment, and found it useless. In a patient of Dr Habershon's it was found that galvanisation down the spine for ten minutes was attended with marked benefit for a time. The electrodes were therefore fixed upon the neck for three hours without intermission, and after this treatment had been continued for some time the limbs became much steadier, but he was not cured. Wilks used to say that no medicines do any good, but that galvanism sometimes relieves.

Reynolds and Gowers agree, however, in believing that electricity has no influence on the course of the disease; and the latter says as much of phosphorus and silver (both remedies which are far from harmless). He thinks he has seen most benefit from cannabis indica, combined with arsenic and a very small dose of strychnia.

CHOREA

> **"De Chorea Sancti Viti: Convulsionis species quædam est, pueros puellasve a decimo ætatis anno ad pubertatem invadit, etc. . . . . Antequam poculum ad os possit adducere, mille gesticulationes, circulatorum instar (like a mountebank), exhibebit. Non enim rectâ lineâ ori admovet, sed deductâ a spasmo manu, huc illuc aliquandiu versat, donec tandem forte fortunâ labris proprius apponens liquorem, repente in os injicit et avide haurit."**
>
> **SYDENHAM, *Processus integri in morbis curandis*, 1692.**

History of the name—Symptoms—the cardiac murmur—varieties—chorea gravis—course and duration—fatal cases—recovery—slight cases—Hemichorea—Pathology—Distribution as to age and sex—Recurrence—Ætiology: fright: pregnancy: relation to rheumatism, to embolism—Prognosis—Treatment. Choreiform disorders—Hereditary chorea in adults—Myoclonus multiplex—Weir Mitchell's *habit-chorea—Dubini's disease—Congenital choreiform spasms (Birth-palsies).*

Synonyms.—Chorea Sancti Viti minor—Chorea minor—Chorea Anglorum.—*Chorea Sydenhamii.*—*Fr.* Danse de Saint Gui.—*Germ.* Vitustanz.

History.—The term St Vitus's dance—*chorea Sancti Viti*—was originally applied to the dancing mania which prevailed in certain parts of Germany in the fourteenth and fifteenth centuries, and was also known as St John's dance. It is said that the designation is first met with in the account of an epidemic at Strasburg in 1418, when those who were attacked were sent on pilgrimage to the chapel of St Vitus, at Zabern.* Similar cases will be hereafter mentioned under the head of hysteria, and approach the saltatory spasms mentioned in the last chapter (p. 858).

The disease now to be described was named chorea first by Sydenham, and for a long time afterwards Sydenham's chorea was distinguished as "chorea minor," or "chorea Anglorum," to distinguish it from the very different neurosis just described.

It is remarkable that while Romberg calls Sydenham's original description of chorea masterly, and von Ziemssen says that it leaves nothing to be desired for clearness and precision, the late Dr Sturges thought we must suppose either that the period and manner of its attack have changed since Sydenham's time, or else that this great physician failed in his description of what he saw. (See the quotation at the head of this chapter.)

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* **"Viel hundert fingen zu Strassburg an**
Zu tanzen und springen, Frau und Mann,
Am offnen Markt, Gassen, und Strassen;
Tag und Nacht ihrer viel nücht assen;
Bis ihnen das Wüthen wieder gelag.
S. Vits Tanz ward genannt die Plag."

Quoted by Hecker from John of Königshoven's Chronicle, first printed at Strasburg in 1698.

Symptoms.—The child affected with chorea (for children are, as a rule, the patients) cannot keep at rest while awake, nor perform voluntary movements with precision. If the patient is sitting or standing still, she (for chorea is more common in girls than in boys) soon begins to fidget. Perhaps she lays her hand palm-upwards on her lap, and then suddenly reverses it; or she shuffles her feet on the floor, or throws one foot over the other, or twists it first outwards and then inwards. Or she may shrug up one shoulder, or throw it forwards, or open and close her mouth or her eyelids without purpose. Indeed, the muscles of the face are continually working. If asked to show her tongue she often seems unable to put it out, but suddenly thrusts it forwards, and then as suddenly withdraws it, her jaws snapping together after it. If she wishes to carry a cup to her mouth she cannot help jerking her arms, and accomplishes her object only after several unsuccessful attempts, and that by gulping down its contents for fear of spilling them. When she tries to walk she moves by fits and starts, and jerks her body and limbs first to one side and then to another. Attempts at voluntary movement at once bring on the choreic symptoms; whereas in slight cases a child may lie quiet in bed or sit still, and nothing seems wrong until she is spoken to, or tries to move.

If told to count, the patient will ejaculate several numbers one after another, and then pause to take a deep breath; or she utters only one sound with each expiration, drawing in air hastily before she goes on to the next. Von Ziemssen says that he has seen with the laryngoscope unsteady and quivering movements of the muscles of the glottis, and that an imperfect degree of tension of the vocal cords is shown by the low pitch and monotonous character of the voice. Romberg relates a case in which there was annoying hiccough, and two others in which inspiration was attended with a whistling noise; in one of them the chest was observed to be suddenly drawn inwards by spasm of the intercostal muscles.

During sleep the choreic movements cease. Jaccoud also remarks that in slight cases they sometimes remain absent for a while after the patient awakes.

Rosenthal and Benedikt have tested the reaction of the muscles to faradic and galvanic currents, and have found their excitability greater than under normal conditions. There is no reaction of degeneration.

There is often a slight amount of loss of power in the limbs affected with chorea. It was first described by Todd as choreic hemiplegia, and has since been called *chorea mollis* by West, and paralytic chorea by Gowers. It is by no means constant, and is never more than paresis of movement. But it is of interest because it has been noted (as in two remarkable cases of Dr Clifford Allbutt) either before the choreic movements appear, or after they have subsided. Osler observed this weakness of muscles sixteen times in one arm, six times in both legs, and four times on one side; once it affected both arms, and once all four limbs.

Children affected with chorea are generally pale, often very pale. They may also be thin and delicate in appearance, but this is often due to previous rheumatism and cardiac disease; and chorea may be seen in stout and rosy children. We must remember that the favourite age of the disease is not early childhood, but the period between eight and fourteen years of age, when both boys and girls, even when in sound health, are usually slender and often pale if their appearance is compared with the red cheeks and plump limbs of earlier childhood.

The temperature is normal, but the pulse is commonly quickened, especially when the movements are severe.

The urine is sometimes scanty and high coloured, in marked contrast with the pale, abundant urine of hysteria.

The heart in chorea.—On auscultation a blowing *systolic murmur* is often heard at the apex. On the interpretation of this sign widely different opinions have been held. Many have maintained that the bruit is functional, or hæmic, or anæmic; while others have supposed that it is due to choreic spasms of the musculi papillares interfering with the closure of the mitral valve. But when the disease ends fatally that valve is found studded with vegetations like those of rheumatic endocarditis. Since the murmur also is the same in the two diseases it seems reasonable to attribute it to the endocarditis in both. In many cases no bruit is audible; but its absence is not a proof that the valve is unaffected, for Kirkes and Wilks have both recorded instances in which vegetations were found after death, although the heart-sounds during life had been normal. A murmur sometimes passes off as the patient recovers from the chorea; but a precisely similar disappearance of the murmur is often observed in cases of rheumatism. On the whole, therefore, it seems most probable that the cardiac murmur of chorea is really due to previous rheumatic endocarditis.

Among 150 cases of chorea in Guy's Hospital during the years 1870-71-72,* the state of the heart is not mentioned in eleven, and the presence of any murmur was expressly denied in eighty. A systolic bruit was heard in the remaining fifty-nine, and in forty-three of them it was distinctly loudest at the apex. In only nine is it described as basic; two of these were indistinct and two were combined with a diastolic murmur. Among 180 fresh cases in the same hospital during the five years 1874-78, collected for the writer by his house physician, Dr George Halstead, sixty-two were reported to have a murmur of some kind (about a third of the cases); of these it was an apex systolic bruit in fifty-two, a basic bruit in seven (of which two were diagnosed as aortic and two as pulmonary), a præsystolic apex bruit in two, and a pericardial rub in one. In the next ten years (1879-89) Dr E. W. Goodall, then Medical Registrar, reported a bruit in 143 cases out of 262. Thus altogether in 592 cases there was a cardiac murmur in 264.

Varieties.—In exceptional cases of chorea the symptoms are but little marked at its commencement, or even throughout its whole course; but we detect a loss of power in one or more of the limbs. Thus, the patient may complain that her arm feels heavy, or may drag her foot slightly in walking; and such cases are often brought to hospital as paralytic. In other cases the patient merely has a trick of making a grimace, or throwing her hand or arm into an odd position, or she may let a jug fall two or three times in a week, or she no longer plays so well as before on the piano.

Hemichorea.—Sometimes the spasms are confined to one side, so far as the limbs are concerned. Dr Hughlings Jackson has pointed out that, even in these cases, the muscles of the trunk and face are always affected bilaterally, a fact of much theoretical interest. Different statements have been made as to the relative frequency with which the right and left limbs are affected. According to Reynolds and Jackson, right hemichorea is the more common; according to Austin Flint, Trousseau, and Jaccoud, left; and Anstie also remarks that one may often at the first glance recognise a child affected with slight chorea from her sitting with her right hand grasping her left wrist to keep it still. Among thirty-three cases out of 150 in which the affection was unilateral, there were eighteen on the left to fifteen on the right side ('Guy's Hosp. Reports,' 1873, "Rheumatism and Allied Disorders"). Since then, among 164 additional cases, the writer found choreic movement decidedly predominant in the right arm or leg, or both, in seven cases; and in the left in thirteen. So that our experience supports that of Anstie and of observers in France and in America.

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* These cases were collected for the writer's article in the 'Guy's Hospital Reports' for 1873, by the late Surgeon-Major Manser, then a student in the wards, who fell a sacrifice to the Plague when it visited Bombay in 1897.

The pupils are generally dilated and sluggish, and in one case of hemichorea von Ziemssen observed that the pupil was much larger in the eye corresponding with the affected arm and leg.

Severe and dangerous cases are happily the exception. They have been separately described as *chorea gravis*. The patient becomes unable to stand; and her limbs are tossed about, so that boards well padded have to be fixed on each side of the bed to prevent her throwing herself on the floor. In spite of precautions bruises occur, and the skin is rubbed off from her elbows and knees. She ceases to sleep altogether, and the violent movements go on day and night without intermission. In such cases emaciation takes place with wonderful rapidity. Dr Tuckwell has related the case of a boy who was wasted to the utmost at the time of his death, but who six days before had borne the appearance of vigorous health, with remarkably well-developed muscles; and conversely von Ziemssen mentions one in which a girl, eleven years old, gained during convalescence 5½ lbs. in ten days, her weight rising from 55½ to 61 lbs. in that time. One cause of the extreme wasting which accompanies severe chorea is doubtless the difficulty with which food is administered; the patient often seizes a spoon, or the spout of a feeding-cup, as if she would bite it in two, and so injures her own teeth, or her nurse's fingers.

The mental state of patients with severe chorea is often one of mania; they shout, sing, or talk at random. A girl who was in hospital some years ago, suddenly got out of her own bed, and turned a somersault across that of another patient. There is no necessary relation between the intensity of the choreic movements and that of the mental disturbance. The mind remained perfectly clear in one of Wilks's patients, who had the worst attack of chorea which he ever saw terminate favourably. There occurred some years ago in Guy's Hospital a fatal case in which there was marked mania and complete loss of consciousness, while at the same time the movements were so like those seen in epilepsy, that the diagnosis remained doubtful until the autopsy was made, when recent vegetations were found upon the mitral valve. Dr Hills, of the Norfolk County Asylum, knew more than one choreic patient who was sent to that institution as insane.

Loss of power of speech is common in all but slight cases of chorea, and is constant in chorea gravis.

Course.—Chorea may be said to be a chronic disorder, but its duration is variable. Almost all the statistics which have been published concur in stating a period of from two and a half to three months as the average. Thus Wicke found it eighty-nine days in a series of 125 cases, and Sée sixty-nine days in 117 cases; while from much smaller data Gray, Tuckwell ('Lancet,' 1871), and Hillier in this country each made it about ten weeks.

Individual cases, however, range widely on each side of these limits; but far more widely *beyond* the average duration than *within* it. Hence the introduction of one or two very prolonged cases may greatly disturb the statistical result; patients sometimes apply for treatment in whom the disease has already lasted for many months or even for two or three years. A single instance of this kind would inevitably spoil, for the purposes of comparison, any series which did not embrace a very large number of cases.

Dr Fagge believed that chorea is more likely to last long when its symptoms are comparatively mild; he had often seen patients who had boards along the sides of their beds get well before those who were up and about the ward throughout their stay in the hospital.

Fatal event.—Patients very rarely die of chorea. At Guy's Hospital between the years 1848 and 1875, there were only twenty cases of the disease which terminated fatally. And of these twenty, five must be left out of count, since death was due to an accidental complication, dysentery, diphtheria, rheumatic pericarditis, or cardiac dropsy. Of the remaining fifteen, only four were below the age of fourteen; two were seven years old, one twelve, and one thirteen. Of the remaining eleven patients above fourteen, nine were between fifteen and eighteen, one forty, and one fifty years old. Few of the women seem to have been pregnant; but it is an ascertained fact that the mortality is very great in this condition, which frequently ends in abortion or premature delivery. It seldom happens, even in the most severe forms of chorea, that the patient dies within three or four weeks from the commencement of the disease. In 1853 a girl aged sixteen was attacked with maniacal chorea, and died in six days; and in the same year another case, in a boy of the same age, ended fatally in nine days. The immediate cause of death seems to be the intensity of the nervous symptoms themselves, but the movements often subside, and may even cease entirely during the last few hours, the patient lying comatose and passing urine and fæces involuntarily. The temperature of the body may rise during this final period: in one case it was 104·7° before the patient died, and Dr Frederick Taylor observed a case in our clinical ward where the thermometer registered a temperature of 108° immediately after life was extinct. It does not appear that endocarditis or pericarditis is directly concerned in bringing about the fatal issue. The presence of bronchitis has been noted in several of the reports of autopsies at Guy's; and perhaps in one instance it accelerated the patient's death. In 1873 Dr Habershon had a fatal case in a boy aged twelve, who had been three weeks in the hospital when his breathing became obstructed by swelling of the tongue, and he died after tracheotomy had been performed. Probably the glossitis was the result of injuries inflicted by the teeth, for in another instance, in which the tongue had been severely bitten in two places, there was discovered after death a foul ulcer which exposed the sublingual gland.

Four fatal cases occurred among the 150 tabulated by Mr Manser for the 'Guy's Reports' (Third Series, vol. xix, pp. 329—332). 1. F., thirteen, 5th March; mitral and aortic vegetations, ulcer from biting tongue. 2. F., seven; aortic vegetations, faucial diphtheria. 3. F., pregnant; mitral vegetations. 4. M., thirty-eight; aortic and slight mitral endocarditis.

Occasionally pericarditis has been found after death from chorea, but this is very rarely the case, and probably is due, like the rub occasionally heard during life, to intercurrent rheumatism.

Among the second series of 180 cases collected by Dr Halstead from the records of the same hospital, during the five years following those which furnished the above statistics, there were only four deaths. 1. In a girl of seven, who died after being six weeks in the ward, there were found the usual fibrinous vegetations on both mitral and aortic valves. 2. In a girl of fifteen, after a month's treatment, there was, beside the same almost constant lesion, lobar pneumonia and a bedsore. 3. In a girl of eight there were mitral and aortic nodules with broncho-pneumonia, adherent pericardium, and a "cardiac" lung. 4. In a lad of seventeen, who had suffered again and again from chorea since he was nine years old, no lesion but mitral vegetations was found.

If to these eight fatal cases we add the six additional cases numbered 5—10 in the paper above referred to, we have a total of fourteen *post-mortem* examinations. In every one of these there were fibrinous nodules found on the mitral or aortic valves. In only two of these was there a history of rheumatic fever, and in seven its occurrence was explicitly denied. Diphtheria was apparently the cause of death in three cases. In three cases there was acute lobar pneumonia, and in a fourth lobular inflammation of the lungs. Only one of the patients was pregnant.

Among 439 cases published by the Collective Investigation Committee only nine deaths occurred ('Brit. Med. Journal,' February 26th, 1887).

Recovery.—This is generally gradual. Sometimes, however, the breaking out of an exanthem, or of some febrile disease, is followed by the sudden subsidence of the movements. This is in accordance with an aphorism of Hippocrates: *spasmos febris accedens solvit.* Radcliffe met with seven cases in which it occurred; and some striking examples of a similar kind are recorded by Rilliet and Barthez. According to the observations of Sée, however, the chorea is not likely to be cut short unless it has reached its acme before the onset of the fever. In many instances the first effect of pyrexia is to aggravate the movements, and yet in those very cases they may cease when the temperature falls.

Occasionally the subsidence of the spasms in severe cases of chorea is not followed by any evident improvement in the patient's general condition. She may become perfectly quiet, and yet may remain for two or three weeks unable to stand, and with little power in her arms. This paresis may be limited to a single limb, or to the two limbs on one side, the "choreic hemiplegia" of Todd (p. 881). In association with such symptoms, or independently of them, the intelligence may remain defective; or a condition of melancholia may develop itself. Dr Barnes met with a case of chorea in a pregnant woman, who as she regained her strength became insane and had to be removed to Bethlem. Other patients, after the choreic movements have passed off, still remain silent for days together. Dr Hughlings Jackson associates this aphasia with right hemiparesis.

Alarming as these various symptoms are, they almost always pass off in their turn, and the patient ultimately regains perfect health. Sequelæ are either absent or extremely rare;* but there is a strong tendency in chorea to recur (p. 890).

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* Trousseau speaks of children who have never again shown the same intelligence as before; von Ziemssen says that slight defects in the co-ordination of the movements, a precipitancy in the performance of certain manual actions, or a tendency to facial grimaces, may persist for years; and Radcliffe believed that chorea is apt to be followed by other neuroses, particularly epilepsy, at a later period of life.

Pathology.—There are still differences of opinion as to whether the seat of the disease is in the spinal cord or in the brain. When it was discovered that frogs could perform co-ordinated movements after removal of the cerebrum, it was natural that chorea should be referred to disorder of the spinal centres. Thus Romberg, writing in 1851, placed chorea among the "spinal spasms"; and in 1873 Jaccoud defended a similar doctrine. Other pathologists have supposed that the disease is seated in the corpora striata.

In favour of the spinal theory of chorea, experimental results have been adduced. Dogs are liable to a similar complaint, and Chauveau divided the cord close to the skull during the progress of canine chorea: the movements went on, the dog breathing by the diaphragm or by artificial respiration; moreover, a second division of the cord, in the lower dorsal region, put an end to the choreiform movements in the legs and tail. Chauveau's experiments were repeated by Legros and Onimus with similar results.

It is, however, now ascertained that canine chorea is not identical with the disease of that name in man,* and, even if it were, there remains the question, whether in the human subject the higher nervous centres do not assume functions which in brutes are performed by lower ones. Some of the points in favour of the seat of chorea being above the cord were stated by Russell Reynolds as far back as 1855. As he remarked, the spasms produced by irritation of the cord are tonic rather than clonic; and it is inconsistent with a spinal origin that the choreic movements are in any degree controlled by the will, that they are increased by emotions or by voluntary efforts, that they cease during sleep, and that they should be diminished by diversion of the patient's attention. Broadbent ('Brit. Med. Journ.,' 1869) has insisted on the fact (which Romberg has previously observed) that tickling the palm of the hand or the sole of the foot of a child affected with chorea leads to no increased spasms; on the contrary, it is borne without difficulty, and the excitability to reflex actions sometimes seems to be less than in health. Another point on which he lays just weight is that the spasms are sometimes unilateral. Lastly, the fact that the mental faculties are frequently impaired in severe cases of chorea would incline one to localise the disease in the hemispheres.

Dr Dickinson reported a series of fatal cases of chorea ('Med.-Chir. Trans.,' 1876), in which he found congestion of the vessels, peri-arterial degenerations, and minute spots of sclerosis in the upper regions of the spinal cord, as well as in the brain. But even if the constant occurrence of such appearances were established, it would still be a question whether they are not merely effects of the disease.†

At present no constant changes have been found in the nervous centres: congestion and minute ecchymoses are too common to have any causal significance. Meynert, the late Dr F. C. Turner, and some other pathologists have described changes in the neurons of the cerebral cortex, but others with equally good methods have failed to find them, and the so-called "chorea-corpuscles" of Elischer have been found in the brain of those who never had chorea.

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* See Mr Victor Horsley's Lectures ('Lancet,' 1886, vol. i, p. 54).

† It is important to note that Dickinson's cases had all reached an advanced stage. Moreover, it is clear that the persistence of the morbid changes which he describes is compatible with the subsidence of the spasmodic movements and the restoration of health. For in one patient who had twice before had chorea (the last time having been a year previously), the duration of the fatal attack was only thirteen days; and yet changes of old date—periarterial degeneration and scattered spots of sclerosis—were found, besides recent congestion of the cord and basal ganglia.—C. H. F.

The corpora striata seem to be a more probable seat of chorea than the cord—one when the disease is unilateral, and both when all the four limbs are affected. Another possibility, however, is that the seat of the "discharging lesion" in chorea is the motor region of the cerebral cortex, and many of the objections to the other proposed localities do not apply to this—partly perhaps because its functions are still imperfectly known.

The next question is as to the nature of the anatomical change. Broadbent maintained that chorea is "a symptom, not a disease. It has been called an insanity of the muscles; it would be better designated a delirium of the sensori-motor ganglia, since it bears the same relation to those parts that the delirium which may occur in a variety of maladies bears to the cerebral hemispheres."

Now, it is possibly true that choreiform spasms may accompany different morbid states of the nervous centres. But it is no less true, and far more significant, that the disease described in the preceding pages as chorea is not met with as an accidental complication of other maladies, but occurs in a particular class of patients, and under conditions peculiar to itself.

Dr Sturges, in his interesting 'Lectures on Chorea' (1877), points out with much acumen how choreic movements find a parallel in the nervous twitchings of mental embarrassment or the restless and purposeless movements of a fidgety child. Yet, admirable as is the critical and negative part of his remarks, it remains true (1) that if a functional disease, the symptoms of chorea must yet depend on functional disturbance (*i. e.* disordered nutrition from irregular supply of blood, or from some poison, or from a molecular change of other origin) which affects one part of the nervous muscular apparatus and not another. Syncope results from anæmia of the bulb, not of the pons; tetanus from strychnia affects the cord, not the cerebrum; and epilepsy depends on some unknown molecular derangement in the cells of the cerebral cortex, not in those of the retina. Our business is to fix the "seat" of a disordered function as much as of an impaired structure. (2) It remains true that, while no line can be drawn between health and disease, or between the gravest premonitions of an impending malady and its slightest early symptoms when developed, we must nevertheless recognise certain limits beyond which slight or recoverable deviations become serious enough to threaten comfort or life, or where they lose the accidental character of individual variation and assume the constant characters of "recurrent concomitant symptoms" (*Symptomen-complex*). We then know that a disease is before us, according to the only reasonable definition of the word; a condition which has to be understood and treated by a skilled adviser. (3) The fact that an excitable and restless child may often be seen to make movements like those of chorea only proves that choreic spasms are physiological; that the "disease" is an exaggeration and perversion of the action of natural nervo-muscular mechanisms. So the diurnal variations of temperature in health are preserved in pyrexia; so the several primitive layers of the embryo maintain their peculiarities when they give rise to morbid growths; and so the reflex spasmodic neuroses described in the beginning of the present chapter are only perversions of normal excito-motor functions. (4) Like other diseases, chorea has its own natural history, its beginning, middle, and end, its proclivities, and its antipathies. In fact, according to the principles laid down in the first chapter of this book, there is scarcely a member of the whole nosology which better deserves to be called "a disease" than St Vitus's dance.

The relation of chorea to rheumatism, to be stated a little further on, is too important to be omitted from this discussion of its pathology. Whenever we gain better insight into the nature of rheumatic fever we shall probably find it throw light on that of chorea. Possibly the relation of the latter to the former disease may be somewhat like that of tabes and general paralysis to syphilis.

Ætiology: Sex.—Chorea is much more common in *females* than in males: the proportion varies between 1 to 2·5 and 1 to 3. Like hysteria it shows its predilection for the former sex in the case of children before the age of puberty; but, unlike hysteria, it is much more apt to occur in children than in adults.

Tabular statements in regard to this point were published by the writer in the nineteenth volume of the Third Series of the 'Guy's Hospital Reports.' He found that among a hundred and fifty patients 42 were males and 106 females. The corresponding figures for 1874—1878 were 43 male to 129 female patients. In the eleven years (1879-89) we had in our wards 74 male and 188 female patients with chorea. So that the proportion in a large London hospital would be about one fourth male to three fourths female cases (228 to 663).

The late Dr Hughes, among 100 cases, found 27 boys and 73 girls ('Guy's Hospital Reports,' Second Series, vol. iv, p. 372), and among 209 cases 42 male and 167 female patients (*ibid.*, Third Series, vol. i, p. 245).

Of 531 cases treated in the Hôpital des Enfans Malades at Paris during twenty-two years,* 138 occurred in boys and 393 in girls, *i. e.* rather more than one to three. (We shall presently see that the excess of female patients is less among those under puberty). Among 422 children under twelve treated for chorea at Great Ormond Street, Dr Hillier found 122 boys to 300 girls, again rather more than one to three. A similar proportion of boys was also found by Rufz at the Children's Hospital at Paris from 1824 to 1833 (overlapping the first three or four years of Sée's period at the same institution), viz. 51 to 183. But among 141 children at the Evelina Hospital, Dr Goodhart had 43 boys and 98 girls; and in the cases collected in Wincke's monograph on chorea, published at Leipzig in 1844, the proportion was 117 to 210, or more than half the patients were boys, whose ages ranged from four to eighteen. If we put together the statistics mentioned in this paragraph (statistics which, with the partial exception above noticed in the Paris cases, are independent of each other) we find that the total numbers are—of 1610 patients, 471 male to 1139 female, or a proportion of about 2 to 5.

Among 436 cases of the Collective Investigation Committee the proportion of the sexes was 115 to 322, or again rather more than one to three.

Among persons more than seventeen years old the preponderance of females is far greater than it is in children. Among Dr Hughes's hundred cases there were seventeen women above the age of sixteen to five men. In the writer's first series there were twenty-one women above fifteen to four men, and in the second series twenty-seven women to four men.

Age.—Chorea is characteristically a disease of childhood, seldom or never seen in infancy, and rare after twenty, except in the case of puerperal or pregnant women. Sydenham's lower limit of "the tenth year" is, however too high. From the sixth to the fifteenth year for boys, and to the seventeenth for girls, is the choreic period.

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* Sée, 'De la Chorée et des Affections Nerveuses,' Paris, 1851.

Of 216 patients with chorea in Guy's Hospital, the writer found 187 between six and seventeen years old. Of *first attacks*, among 322 patients recorded there, only two were two years old, and none under that age. The disorder first appeared in five cases between two and five years of age, in 102 between five and ten, in 134 between ten and fifteen, and in 55 between fifteen and twenty. Only two male patients were over twenty when first affected with chorea—one not yet twenty-one, the other thirty-eight; while there were six young women between twenty and twenty-six.

Of Dr Hughes's hundred patients in Guy's Hospital, only one was under eight years old (a boy of five), thirty-two were between eight and ten, forty-five between ten and fifteen, twenty between fifteen and twenty, one (a young man) was twenty-five, and two young women were twenty-two and twenty-eight. In his second series of 198 cases, nine were between four and eight years old, 148 were between eight and sixteen, and above sixteen there were only four men, aged 18, 20, 21, and 43.

In Paris, Sée found that out of 531 cases of chorea, 453 occurred between six and fifteen; in Philadelphia, Hammond found the proportion 67 out of 82; and in Germany, Rufz gives the corresponding numbers as 180 out of 189.

Late chorea.—Chorea is extremely rare over twenty-five, except in the case of pregnant women; but the writer once had an unexceptionable case in a man of thirty-eight, and several authentic instances have been recorded of the occurrence of chorea at an advanced age. Among the 439 cases of the Collective Investigation Committee there were only ten above twenty-five years old. Five of these were old women between sixty-three and eighty-six. Dr Graves mentions the case of an apothecary in Dublin who was attacked when seventy years old; Romberg saw chorea in an old woman of seventy-six, which had, however, begun when she was six years old; and Trousseau relates in detail an instance which came under the observation of Dr Henri Roger, in a lady, aged eighty-three, who recovered from the disease in five weeks. Charcot has described *senile chorea* as a distinct variety of the disease, but we may perhaps be allowed a doubt whether all the cases would stand criticism.

Congenital cases.—A few examples have been published of chorea in new-born infants. Thus Richter is quoted by von Ziemssen as having recorded two cases in each of which a female child was affected at birth, the mother having received a fright while advanced in pregnancy; the infant suffered while awake from clonic spasms, which were absent during sleep, and afterwards ceased almost entirely. A somewhat similar instance was related by Dr Long Fox as having occurred congenitally in an infant born six weeks before the proper time. Other cases are probably really cases of cerebral birth-palsy with athetosis (p. 897).

Still the fact remains, that in the great majority of cases chorea is a disease of childhood between the ages of six and fifteen years, or between the period of the second dentition and that of puberty. The preponderance of female cases is perhaps due to the sensitiveness and mobility of the nervous system in women. And of boys as well as girls those seem most liable to suffer who are delicate and excitable.

As to the question whether chorea is most apt to occur in families subject to other neuroses, there is difference of opinion. Trousseau and Anstie thought so, while Sée gave a contrary opinion. We must first agree what maladies we will include under the title. Epilepsy, hysteria, and insanity would be admitted by all; but some would take in infantile paralysis, others tuberculous meningitis, or hemiplegia, or idiocy, or tetanus neonatorum—diseases which pathologically are widely different. In the writer's opinion even when these organic diseases are left out of count, there is no reason to suppose that inheritance has any share in the ætiology of chorea, except inheritance of rheumatism.

Recurrence.—An important point in the ætiology of chorea is its liability to recur again and again in patients who have once suffered from it. A large proportion of the patients admitted into any hospital have had the disease once or oftener before. Of 262 choreic patients admitted to Guy's Hospital from 1879 to 1889 inclusive, 102 had suffered previously. The writer has recorded the case of a young man who suffered from chorea every autumn from the age of fourteen to that of twenty-two, and of a girl who was attacked each May, from her eighth year to her fourteenth. The duration of a relapse is generally less than that of the first illness, but there are exceptions to this rule.

Race and climate.—Weir Mitchell, Sinkler, and Osler agree on the extreme rarity of chorea among negroes in America, and also among the aboriginal Indians. It is emphatically a hospital disease, and is somewhat rare in private practice.

There is no reason to suppose that seasons have any influence on the prevalence of chorea. The observations made to this effect in Philadelphia were not found to apply in Boston, and any difference observed in this country has been neither constant nor large enough to exceed the limits of accidental coincidence.

Mental shocks.—The immediate exciting cause of chorea is often a fright. Thus Romberg relates the case of a girl, aged ten, who was one morning alarmed by a dog jumping at her and barking, and who was seized with chorea the same evening. Ziemssen speaks of a boy, aged ten, who was terrified by a shot falling unexpectedly close to him on a field, and in whom marked chorea followed within a few hours. Trousseau gave the case of a girl, aged sixteen, who had been caught hold of by a man as she was going downstairs one evening without a light, and who was so frightened that she from that moment became affected with St Vitus's dance. Bright described the case of a boy who had already recovered from an attack of chorea, and who was sleeping with his father when the latter was seized with a fit of apoplexy; the boy was so alarmed that his chorea returned. A child, admitted into Guy's Hospital, had been frightened by seeing her brother in flames, ran out of the house screaming, and took refuge in a neighbour's house. She was brought home and put to bed, when her mother at the time noticed a twitching movement of her face and limbs; next morning she was unable to stand, and chorea developed.

There is no doubt that much exaggeration has prevailed in regard to the association of chorea with mental emotions, and that parents arrive too hastily at conclusions with respect to the "causes" of this as of most other maladies. But numerous cases like those just quoted occur at every hospital, and cannot be reasonably ascribed to coincidence. All we can say is that in many cases of chorea there is no evidence of a fright or a nervous shock having preceded the attack. Dr Hughes ('Guy's Hosp. Rep.,' 1856) related a fatal case in which the symptoms were slight when the patient was admitted, but became suddenly aggravated in consequence of a fright.

It has been thought that chorea may be the result of *imitation*. So Dr Addison taught at Guy's Hospital, and would not allow other children to be placed in the same ward with cases of chorea. But it does not appear that any of the sisters or nurses remember an instance in which chorea has actually been so acquired.

Bricheteau relates how, one afternoon, a young girl was admitted to the Necker Hospital, suffering severely from the disease; in the evening a patient already in the ward, who had previously had chorea and was suffering from hysteria, began to exhibit choreic movements, and in twelve hours had to be tied down; next day two other cases occurred, and within the four following days five more, making eight in all: the disease might probably have spread further had not the patients been then isolated. But might not this have been hysteria simulating chorea?

Heredity.—Two children in the same family often have chorea, but we rarely find that the parents of a choreic child have themselves suffered from the malady when young. This is the same kind of "transverse" hereditary relation which we observed to obtain in certain other maladies,—Friedreich's ataxia (p. 705), Thomsen's disease, and some forms of muscular atrophy. Chorea is certainly less often inherited than rheumatism.

Pregnancy.—A condition which predisposes to chorea in young women is *pregnancy*. Dr Barnes collected a series of fifty-eight cases of this kind ('Obstet. Trans.,' vol. x). The period of gestation at which the spasmodic movements are most apt to begin is from the first to the third month, but sometimes it is much later; and two instances have been recorded in which they followed parturition. First pregnancies are much more often accompanied by chorea than subsequent ones. This of itself suggests that one element in the production of the disease may be the emotional excitement which arises in a woman who finds herself for the first time pregnant, and the suspicion is confirmed by Wilks's observation that many of those who are attacked are unmarried girls, to whom their condition is one of shame and distress. Thus a connecting link is established between the chorea of pregnancy and that following mental shock; and a still stronger one is the fact that in a considerable number of cases the patient has already had the disease on one or more occasions at an earlier period of life. Among the fifty-eight cases already referred to there were fourteen in which previous attacks had occurred.*

Rheumatism.—So far the causal relations of chorea differ but little from those of the neuroses in general. We now pass to a predisposing cause which applies to this, but to no other nervous disease,—rheumatic fever.

As far back as 1811 rheumatism was stated to be one of the causes of chorea, in the Syllabus of Lectures on Medicine delivered at Guy's Hospital, by Drs Babington and James Currie, "It often follows rheumatism, and these two diseases often come on alternately." Dr Bright (whose testimony carries back the tradition nine years earlier) was himself convinced of the relation.

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* See also Dr Lever's paper ('Guy's Hosp. Rep.,' Second Series, vols. v, p. 3, and vi, 233).

Beside Bright and his pupils, the younger Babington and Hughes, Dr Copland and Sir George Burrows were early advocates of the belief that there is a real connection between chorea and acute rheumatism, and that the latter predisposes to the former. Sée introduced the same fact into France, and it is generally acknowledged in Germany and America. The connection is all the more probable because it is quite unexplained, and supports no theory of either disease.[*]

The close association between chorea and rheumatism is shown by the fact that these two disorders are the chief and almost the only causes of non-septic valvular endocarditis. Out of over eighteen fatal cases of chorea which occurred in Guy's Hospital between 1848 and 1876, in only one was endocarditis absent; and that its frequency was not dependent on the severity of the chorea is evident from the fact that in five of these cases the patient's death was due to some complication. The same conclusion is confirmed by the frequency with which chorea and rheumatism occur in the same patient; a child who has had rheumatism falls ill with St Vitus's dance a few months afterwards, or we find in a case of chorea evidence of a slight febrile attack with transient pains in the joints which has been almost forgotten; or slight choreic movements appear in the course of a rheumatic attack, or rheumatic synovitis recurs in the course of chorea.

Statistics are as follows:—Among the patients of Guy's Hospital, (*a*) in the late Dr Hughes's first series ('Guy's Hosp. Reports,' 1846), special inquiries were made in fifty-eight cases, and there was a cardiac murmur in nine, and a history of rheumatism without bruit in eight more; (*b*) in his second series, compiled by Mr Burton Brown (*ibid.*, 1855[†]), special inquiries were made in 104 cases, and in eighty-nine of these there was either a bruit or a rheumatic history; (*c*) in the series of 150 compiled by Manser, and published in 1874 (*ibid.*, Third Series, vol. xix), forty-two had suffered from rheumatism, and in three of these rheumatic fever supervened while they were under treatment for chorea; there was a bruit in fifty-nine, none in eighty; (*d*) in the fourth series, of 163 cases compiled by Halstead, fifty-three had had rheumatic fever or distinct pains in the limbs, believed to be rheumatic, and in thirty-five of them the rheumatism preceded the chorea by less than six months; there was a bruit in sixty-one, none in one hundred and eleven.

Of Prof. Sée's 128 cases, sixty-one had probably had rheumatism.

Of Dr Sturges' 100 cases, only twenty; but in 104 consecutive cases of chorea (also at the Westminster Hospital) Dr Donkin found twenty-seven cases of previous rheumatism.

Of Dr Goodhart's 130 cases (collected from several sources) eighty-nine were on positive evidence believed to have had rheumatism.

Of Dr Angel Money's 214 cases (also collected) thirty-three had had rheumatic fever, twenty-three "rheumatism," and nine were doubtful.

In 172 cases of chorea at the London Hospital, Dr Stephen Mackenzie found a history of true rheumatism in forty-seven ('Trans. Int. Congr.,' 1881, vol. iv, p. 97).

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[*] The only plausible arguments against it were well put by Dr Sturges ('Chorea and Whooping-cough,' p. 16).

[†] The first volume of the Third Series was published in 1855, not 1856, as stated by von Ziemssen, Bd. xii B, S. 443.

The Collective Investigation gave 116 instances of precedent rheumatism (excluding mere "rheumatic" pains) in 439 cases of chorea.*

Embolic theory.—Various attempts have been made to explain the connection between chorea, rheumatism, and cardiac disease. One theory has had the support of some eminent physicians but has little other claim to acceptance. Starting from the frequent association of endocarditis with chorea, the late Dr Kirkes suggested that the cause of the spasmodic movements might be the conveyance of particles from the diseased valves into the arteries, and the consequent disturbance of the nervous centres. Dr Hughlings Jackson and Dr Broadbent have expressed a similar view in a more definite form, maintaining that the cause of chorea is embolism of minute arteries in one or both of the corpora striata.

A few observations have since been made which have been thought to corroborate this theory; one by Dr Tuckwell ('Med.-Chir. Rev.,' 1867) and another by Dr E. L. Fox ('Med. Times and Gaz.,' 1870). Dr Angel Money afterwards tried to produce chorea in monkeys and cats by injection of starch-granules or insoluble salts into the cerebral arteries, and the results will be found in the 'Medico-Chirurgical Transactions' for 1885; see also 'British Medical Journal,' July 17th, 1886.

But can we always be sure whether clots in minute cerebral arteries are of *ante-mortem* formation, and, if so, whether they entered the vessels from below, instead of being formed *in situ?* The minute, firmly adherent vegetations which are found on the valves in chorea, do not seem likely to be carried away by the blood-stream, and we do not observe chorea in adults with rheumatic endocarditis, or in patients of any age with septic endocarditis, although embolism is far more frequent. Moreover, no emboli are found in the spleen and kidneys after death from chorea.

Lastly, we have to account for the relation of chorea to mental shocks. Such cases are certainly not distinct from those in which it is associated with endocarditis. On the contrary, as already stated, vegetations are found on the cardiac valves in all fatal cases, almost without exception; for instance, a child died under Wilks's care who was attacked by chorea after being terrified by the gunpowder explosion at Erith, and *post mortem* the mitral valve was found inflamed.

No doubt any theory of chorea presents difficulties. In the writer's belief the connection with rheumatism (*i. e.* of course with rheumatism as defined in the chapter on that disease) and the connection with a definite period of life are the two most important pathological features of chorea. He believes that endocarditis is not the result of chorea, but of the rheumatism which usually precedes it, and that all three conditions, fever with synovitis, cardiac inflammation, and chorea, will prove to be the results of one and the same, probably toxic, condition—though certainly no microbe has at present (1900) been discovered, which has even a presumptive claim to provide the condition.

Prognosis.—The usual benign and occasionally dangerous course of chorea has been indicated above. The unfavourable symptoms which mark *chorea gravis* are inability of the child to feed itself, inability to speak, and inability to sleep, the first being the least grave and most frequent, while the last symptom, unless checked by treatment, is fatal. The more chronic cases are almost always mild and recover in time.

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* See also statistical papers by Dr Herringham and Dr Archibald Garrod in the 'Medico-Chir. Trans.,' vol. lxxii, pp. 117, 145.

Treatment.—This is a very difficult question; for chorea offers peculiar obstacles to the satisfactory investigation of the action of remedies. Some of the severer cases tend to a more than usually rapid recovery; and in other instances (as Wilks long ago proved) the child being admitted into the ward of a hospital and kept in bed is sufficient to bring the complaint quickly to a termination. Under either alternative, the medicine which may have been prescribed is apt to get undue credit. Lastly, in most cases chorea subsides of itself after eight to twelve weeks, if not prolonged by unfavourable surroundings.

It is almost impossible to obtain statistical proof of the value of drugs in the treatment of chorea.* But there is strong evidence, of another kind, in favour of at least one remedy. Very protracted cases, which had resisted other methods of treatment, have sometimes yielded quickly to *arsenic.* Some striking examples of this were recorded by Romberg.

One is that of a girl, aged eleven, who had for eight years suffered from intense chorea, affecting especially the right half of her body. All drugs had been found useless until she began to take Fowler's solution; in about two months there was a marked improvement, and at the end of two months more she had entirely recovered. Another patient, a girl aged ten, had had the disease two years; arsenic was prescribed, and within three weeks the symptoms presented a marked abatement; and ten or eleven weeks afterwards she was discharged cured. A third instance is that of a girl, eight years old, who for six months had been the victim of chorea to such an extent that she could not walk, nor stand, nor speak articulately. The remedies which had been tried had failed; Fowler's solution, in doses of four drops three times a day, established a cure in eight weeks.

Another argument in favour of the effect of arsenic upon chorea is derived from our experience among out-patients. There the children have none of the great advantages of quiet, with careful feeding and nursing which cure many cases of chorea when admitted to a hospital ward. No change in their condition is made, except the administration of a drug; and when under this treatment a complaint which, in some cases, has lasted for months improves in a week, we can scarcely help attributing the cure to the remedy.

In prescribing arsenic it is important to increase the dose every other day or, in severe cases, every day, and to push it until there is some evidence of its physiological action. So given, children are very tolerant of the drug.

For many years *sulphate of zinc* has been largely used at Guy's Hospital; one-grain doses of it used to be given at first, which were gradually increased until the patient took a scruple. It seems more advisable to begin with a larger dose—for instance, with five grains. This seldom causes nausea or sickness more than once or twice. Another drug which once had a great reputation is the *carbonate of iron.*

Salicyl compounds have been sometimes given, and, in some cases, with seeming success ('Brit. Med. Journ.,' 1887, vol. i, p. 436).

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* The plan of treatment usually adopted is to give one drug for three or four weeks; if that fails, to change it for another, which is continued for about an equal period of time; and, if there is still no result, to begin the administration of a third. The result necessarily is that even if all these medicines are really inert they gain very different degrees of credit. The uselessness of the one which is first given is sure to be apparent; but the second runs a chance of apparent success; while the last one is almost certain to acquire the reputation of having cured a case in which its competitors had altogether failed. Yet that very drug, if placed first on the list for a succeeding case, would perhaps show itself as impotent as the others. Probably much of the uncertainty as to the value of remedies in chorea is explicable in this way.—C. H. F.

Static electricity, used in the old-fashioned way, was formerly much used by Addison, Golding-Bird, Gull, Hughes, Babington, and Lever ('Guy's Hosp. Reports,' Series I, vols. ii, vi, and vii; Series II, vol. viii), but it has long been given up. Galvanic and faradic electricity have likewise been tried, praised, and abandoned.

There is reason to believe that mild chronic cases of chorea may to some extent be checked by gymnastic exercises, by military drill, or by the use of a skipping-rope. French physicians have laid stress upon this method of treatment, and it has had undoubted successes.

On the other hand, one sometimes has to deal with cases which are so severe that the administration of arsenic or zinc, requiring time for their operation, is inapplicable, because the patient is in imminent danger of dying within two or three days. There is the greatest difficulty in determining the value of remedies in cases of this kind. Probably death is sometimes inevitable. But even then the inhalation of *chloroform* gives great relief to the patient's sufferings; and if there be a chance of recovery it may do something towards economising his strength. It also saves him from the sores which form over the bony prominences if the movements are uncontrolled. In cases somewhat less severe *chloral hydrate* appears to be the best medicine. Several writers have related cases in which it seemed to be effectual; not the least striking is one of Gairdner's, of a girl who took a drachm of chloral by mistake, and was poisoned, but who on her recovery was found to be cured.

In severe cases it is most important for the patient to be plied with fluid nourishment, including brandy; and nutrient enemata or suppositories should be frequently administered.

Habit-chorea.—This term was applied by Dr Weir Mitchell to those involuntary tricks which are truly spasmodic neuroses. They begin in childhood, but often persist in adult life. Blinking with the eyelids, tapping with the foot, suddenly twisting the head or jerking the hand, hiccough, choking or snorting at intervals, sniffing in a lecture and stammering in a speech—all these are examples of what the French call *tic convulsif*. They were treated of above (p. 853).

Hereditary chorea in adults—Huntingdon's chorea.—Under these names a choreiform malady has been described, which is hereditary in certain families, and passing over the earlier years of life, first makes its appearance at about the age of thirty. The clonic spasms begin in the face, gradually spread to the arms and the legs, and continue so for several years. The gait acquires a peculiar hasty and hesitating character, and the speech becomes intoned. Not only is the malady long continued, but it has usually ended in mental decay, and at last in death. This variety of spasmodic neurosis is obviously different from true chorea in its course as well as in its pathological relations. Dr Huntingdon's cases were observed in Long Island, New York. Others have been described in this country, and Huber has recorded a remarkable case which occurred in Eichhorst's practice at Zürich.

*Friedreich's spasms.**—Under this name a spasmodic disease in an adult patient was described by Friedreich in 'Virchow's Archiv' (vol. lxxxvi), and similar cases were recorded by Hammond, Reynolds, and other physicians under the title of Spinal epilepsy. It is called *Myoclonus* by Unverricht, who published five cases of his own on the subject in 1891. Dr Ferrier brought two brothers before the Neurological Society, in 1897, who were the subjects of myoclonus and also of epilepsy, a combination which is denoted by the term Myoclonus epilepticus. Gowers considers that the malady is perhaps intermediate between chorea and facial spasm or torticollis, and more nearly allied to senile chorea than to any other malady. A good account of this form of muscular spasm is given by Dr Risien Russell in the seventh volume of Allbutt's 'System,' p. 888.

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* *Synonyms.*—Paramyoclonus multiplex—Myoclonus multiplex—probably identical with the Convulsive tremors described by Pritchard in 1822.

*Dubini's disease.**—This name has been given to an obscure malady consisting of choreiform spasms which has been observed in Northern Italy, and is supposed to depend upon malarial influence. The suddenness of the spasmodic shocks, the fact that many of the patients are of advanced age, and that some cases are fatal, widely separate this so-called electrical chorea from true chorea. There are, moreover, tonic paroxysms and epileptiform fits, and there is muscular atrophy in the later stages of the disease. It was first described by the late Dr Dubini, of Milan, in 1846.

Congenital chorea (*Birth-palsy*).—A remarkable condition, which was formerly described under the above name, is as distinct from true chorea as Mitchell's "habit-chorea," Huntingdon's "adult chorea," or Dubini's "electrical chorea." It was described among spastic paralyses in the last edition of this book, but might better find a place among the effects of cerebral hæmorrhage.

For these cases of hemiplegia (or sometimes mono- or paraplegia) combined with choreiform spasms and spastic symptoms, which occur in new-born children, are now ascertained to be the result of meningeal hæmorrhage, occurring in prolonged and particularly in instrumental labour. Certain parts of the cerebral cortex are thus compressed and permanently injured, with the result, as the child grows up, of symptoms both irritative and destructive. The former are the "choreiform spasms," the athetosis often present, and the spastic paralysis of one or more limbs. The latter are the mental imbecility, the paralysis, and the muscular wasting.

In April, 1890, was admitted into Philip Ward a boy, about eight years old, whose case is abbreviated from the writer's notes. He "makes faces" much as a child with chorea does; but on pulling down the bedclothes it is plain that the case is not one of ordinary chorea, for the movements of the limbs are slower, and the spasms are tonic as well as clonic. The arms are extended and the hands go through slow twisting movements, not choreiform, but exactly what is described as "athetosis" (cf. p. 735). The legs are thrust out and very stiff, so that the trunk can be lifted on raising them: the adductors and extensors are in tonic spasm. The feet do not show the characteristic attitude of tetany. The spasms are much increased when the visit to the ward brings many strange faces round the bed, but they do not disappear when the child is quite undisturbed, and even in sleep he lies always on one side, with his legs and arms thrust out in front of him to their full length. Handling the muscles increases the rhythmic as well as the spastic contractions, and if they are continued the child sometimes cries, but it does not appear that he suffers much pain even then. He can put out his tongue; there is no squinting or nystagmus; the functions of the bladder and rectum are normal. He is well-grown, rather slender in build, but not ill-nourished; and there is no deformity of the head, no curvature of the spine, and no clubfoot. A normal knee-jerk can be detected with some difficulty: there is no clonus. He can talk but almost unintelligibly; and though he understands what is said, he is obviously behind his years in intelligence. He is docile and good-tempered, but timid. On further inquiry we learn that this condition is congenital, that he "could not move at all" as a baby, and could not talk till he was much older than usual, and that at one period he was "subject to fits." Lastly, Dr Todd, of Brigg, Lincoln, who brought him into the world, informs us that the labour was unusually protracted, and that there was a well-marked caput succedaneum when he at last was born.

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* *Synonyms.*—Electrical chorea—Typhus convulsivo-cerebralis—Myelitis convulsiva. The same term, electrical chorea, has been applied by Bergeron to spasms of the neck probably hysterical.

The clinical features of this case agree with those of a reflex spasmodic neurosis, which has been described as "congenital choreiform spasms;" but the presence of a definite anatomical lesion is rendered almost certain by the absence of family predisposition, and by the history of the patient's birth. Probably the lesion was meningeal hæmorrhage occurring in the course of a protracted parturition.

Dr Sarah MacNutt, an American physician, observed such cases in 1885. Many prove fatal in infancy, but in others the hæmorrhage is followed by circumscribed atrophy of the cortex, either from pressure of the clot, or perhaps more likely from disturbance of the nutrition of the convolutions. To this, secondary descending lateral sclerosis probably succeeds. There seems to be abolition of inhibitory motor centres in the cortex, which allows co-ordinated reflex movements to take place like those of a decapitated frog or pigeon. See Osler's 'Cerebral Palsies of Children.'

The spasms are not always general in these cases; they may affect one side, or both legs, or one limb only. In some cases clonic spasms predominate, bringing them nearer to "hammer-spasms" and "histrionic spasms;" in others the resemblance to primary spastic paraplegia is very close; in others again, that to chorea. The long duration of the malady, the more or less deficiency of speech and intellect (not more, however, than in certain cases of chorea while they last), and the history of the origin at birth are the diagnostic characters to dwell on. These cases must be carefully distinguished from those of congenital idiocy with paralysed and deformed limbs, due to intra-uterine disease or defective development of the cortical centres.

The prognosis is unfavourable, but some of the patients gradually acquire increased power of grasping objects, of talking, and even of locomotion.

No treatment is promising. Physostigma, bromides, and chloral hydrate have been tried without much benefit. Gowers, who has observed more than thirty cases of this curious malady, says that electricity, in whatever form applied, is useless.

MEGRIM

MIGRAINE OR SICK HEADACHE

> "When your head did but ache,
> I knit my handkerchief about your brows."
> *King John.*

Nomenclature and definition—The visual disturbance, pain, nausea and other symptoms—Pathology and causes—Diagnosis—Prognosis—Treatment. Headache generally—Its ætiological varieties, peculiarities, and treatment.

Synonyms.—Hemicrania (ἡμικρανία), whence *Fr.* Migraine, and our vernacular term, "the Megrims." *Germ.* Hemicranie; Migrän.

Paroxysmal sick headache; Bilious headache (in part); Blind headache; Clavus hystericus. Hemicrania periodica and Hysteria cephalica refer rather to supra-orbital neuralgia than to Megrim.

In strictness the term "hemicrania" is inapplicable when both temples ache at the same time; but such cases are the more numerous, and cannot be separated from the others. Again, many cases of bilious headache present no other indication of hepatic disorder than nausea; and other kinds of headache are far more closely related to dyspepsia and constipation.

Definition.—A functional neurosis occurring in paroxysms which are marked by disorder of vision, severe headache, and nausea or vomiting.

Symptoms.—An attack of migraine in its most typical form begins with dimness of sight; after this pain in the head comes on, often accompanied by vomiting; and the same succession of events recurs again and again at intervals.

The *affection of sight* usually precedes the other symptoms. The patient first notices that he cannot see distinctly some part of what he is looking at. The portion of the visual field thus blotted out is at first very small. It may be at the exact centre of the field, but more often it is a little to one side of it, so that in reading from a printed page he has to glance away from the word he wishes to see. The dim spot is not black, but seems like a faint cloud, of the same colour as the rest of the surface upon which the eyes are directed. It quickly begins to enlarge, and gradually overspreads more or less of one lateral half of the field. This symptom is known as Hemiopsia (cf. *supra*, p. 732). Dr Hubert Airy calls it *teichopsia* (τείχος, a city wall; ὄψις, a vision), from the peculiar zigzagged outline, with angles like those of a fortification,* which often marks the edge of the blind half of the field ('Phil. Trans.,' 1870). This blind half seems to have an undulatory motion like boiling water; and the edges flicker or revolve. The form of the cloud is originally oval, but as it grows larger a gap forms towards the centre of the field, so that it becomes horseshoe-shaped. Gradually one part of the curve approaches the yellow spot, and here the angles are much smaller and closer together than where the cloudy crescent spreads away into the outer region of the field. Sometimes the cloud is uncoloured; sometimes it presents brilliant gleams of red, blue, and other tints. If the eyes are closed, or if the patient goes into a dark room with his eyes open, the whole cloud appears to be faintly luminous. As it increases in size, the central part gradually clears up and accurate vision is regained; on a printed page, for instance, a few letters can now be plainly recognised in the midst of the glimmering crescent. Before long the latter likewise disappears, and the patient can see as well as ever. The whole process occupies from ten to twenty minutes, or at most half an hour.

While the oval cloudy patch and its zigzagged border are visible, they are seen in their minutest details by both eyes alike. To this rule a single exception was recorded by Sir John Herschel, who once satisfied himself that his left eye was alone affected. But, as Dr Airy remarks, everyone is at first inclined to suppose that one eye only is concerned, namely, that of the side on which part of the visual field is blotted out. This fact that the impairment of vision is referred to both retinæ proves that the seat of the affection is above the optic chiasma. The limitation to one half of the field is just what we should expect from the anatomy of the cerebral centres, the separate halves of which communicate with corresponding halves of the two eyes. Sir John Herschel, indeed, stated that in his own person he once observed "the shadowy pattern of a fortification which passed completely across the field of vision from left to right." If so, we must suppose that on that occasion the disturbance extended over to the opposite side of the brain at an early period of the attack. We shall see that at a later stage this is not unusual.

In a few cases, as soon as natural vision is restored, the attack is at an end. But in the great majority a more or less severe headache comes on at this period.

A curious circumstance in regard to the visual affection is that some of the best and most careful descriptions of it have been written, not by physicians, but by astronomers and natural philosophers. Wollaston, Arago, Sir David Brewster, Sir John Herschel, Sir Charles Wheatstone, Dufour (of Lausanne), Du Bois Reymond, Sir George Airy, and his son, Dr Hubert Airy, were all liable to this visual paroxysm, and all carefully noted its phenomena; no similar malady has, within the present century, been the subject of two papers admitted into the 'Philosophical Transactions,' as well as of communications to the 'Philosophical Magazine' and other scientific journals at home and abroad. It may be a question whether persons who are not accustomed to employ the eyes for minute observation would notice the dimness of sight, or mention it to their physician. Indeed, when it begins at some distance from the centre of vision it is sure to be overlooked, unless the patient's attention is specially directed to it. This may, perhaps, be the reason why Du Bois Reymond did not mention it in describing megrim from his own experience. Whether the remarkable visual phenomena just described are invariably present is disputed: certainly in the complete form described above from the personal accounts of Airy and Liveing they are not; and in some cases there is nothing but a certain dimness of the excentric part of the field, which may easily pass unnoticed by patients not accustomed to minute and accurate observation. Among physicians who have themselves suffered from the disorder they describe, may be mentioned Haller of Göttingen and Bern, Dr Fothergill (the founder of the Medical Society), Prof. Lebert of Zürich, Sir Samuel Wilks, Dr Hubert Airy, the late Dr Anstie, and Dr Edward Liveing.*

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* The comparison is not to the battlemented wall of a mediæval fortress or church tower which presents quadrangular outlines, but to a fortress seen *in plan*, with the bastions and their re-entering angles. Dr Airy gives actual figures in his paper, which Dr Liveing has reproduced at the end of his treatise.

The *pain* of sick headache varies greatly in severity, both in different cases, and in the same case in different attacks. It begins at some one spot in the brow or temple and gradually spreads over these regions. Sometimes it remains confined to a single point, in the temple or over the parietal bone on one side. It was to these cases that the special name of *clavus* was formerly applied. A strict limitation, however, to one spot is exceptional, and it is not even the rule that the headache should keep to one half of the head. Dr Liveing finds that in the majority of cases it affects the whole forehead and both temples, although with more severity on one side than on the other. It often extends to the orbit, and is referred with special intensity to the back of the eye; and occasionally it passes behind the ear to the occipital region. Some writers describe it as of a stabbing, cutting, or boring character; others as throbbing, *i.e.* increasing with each beat of the heart. It is augmented by every movement, by exposure to light, and by noise. The patient instinctively lies down, and keeps the room as dark and quiet as possible. One patient of Sir S. Wilks, however, found that the recumbent posture aggravated the pain, and would sit up all night rather than lie down until the attack passed off: and Dr Liveing cites cases in which the pain is so intolerable that the sufferer cannot lie still, but is obliged to get up and move about.

The headache scarcely ever remains long at the same pitch of intensity. Generally it goes on gradually augmenting in severity until it reaches a culminating point, after which it begins to decline. Its increase is usually steady, but sometimes by fits and starts.

When the pain becomes intense the patient begins to feel *nausea;* and presently he retches and vomits. Anything that the stomach contains is rejected; if it is empty, the retching is ineffectual, or some mucus at first is brought up, and afterwards bile. After free vomiting the pain often passes off, and many patients say that they get well as soon as they are sick.

In some cases, however severe the headache may be, no sickness occurs. The pain gradually passes off of its own accord, but often lasts for the rest of the day, until at length the patient is worn out and drops off to sleep; when he wakes on the following morning he finds only a slight soreness of the forehead or temple left. Some persons lose the pain if they can sleep for a short time, even during the early part of an attack. Dr Liveing mentions a gardener who, if he could at the onset of an attack leave his work and lie down under the shade of a tree, would wake at the end of half an hour as well as ever. Lastly, cases have been recorded in which the attack ends by epistaxis, by a copious secretion of tears, by profuse perspiration, or by an abundant flow of pale urine.

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* A patient subject to migraine writes: In my case the zigzags of teichopsia are luminous—a vivid white, like the arc electric light (Nov., 1900).

Exceptional symptoms.—In certain cases, generally severe ones, common *sensation* is impaired in one hand, especially towards the ends of the fingers; and a feeling of tingling, thrilling, or formication may also be experienced. Sometimes the surface of one arm and of the corresponding leg seems to have "gone asleep," or numbness may extend to the mouth, the lips, tongue, or throat. Anstie noticed in his own person that, even in the intervals between the attacks of pain, the power of distinguishing impressions was permanently less in the skin round the inner canthus of the right eye than on the opposite side; during and soon after the paroxysms the impairment of sensation was greater, and affected a larger area.

Occasionally *muscular power* seems to be impaired; at least ptosis and strabismus have each been present in cases which have been regarded by good observers as megrim.

In some cases the faculty of *speech* is disordered. The patient has difficulty in finding the expression which he wishes to use, or in constructing a coherent sentence. He may substitute one name for another; and an instance is recorded by Dr Liveing in which not a single word could be uttered. This may occur without any confusion of thought, or the patient may be painfully conscious that his memory is at fault. Hallucinations are very rare; but there is often much mental depression, with a vague sense of anxiety and dread.

Such serious symptoms are uncommon, and generally begin before the headache, and after the affection of sight has continued for some time.

An oppressive *drowsiness* is sometimes noticed, so that the patient lies half unconscious, not heeding when he is spoken to; and another more common occasional symptom is *vertigo.*

The *pupil* appears to be most often contracted, but Du Bois Reymond observed that in himself it was always dilated. In one instance Möllendorff found with the ophthalmoscope that the background of the affected eye was of a bright scarlet colour, the optic papilla red and œdematous, the central artery and the veins enlarged and tortuous. In some instances the temporal artery becomes enlarged, and plainly visible on the affected side of the head. These facts indicate that vaso-motor nerves may be affected in a paroxysm of migraine.

After the subsidence of the paroxysm, moreover, certain curious trophic changes are occasionally observed. Anstie relates that when he himself had a severe attack of megrim the eyebrow would show a distinct patch of grey opposite the supra-orbital notch, but that subsequently the individual hairs regained their natural colour. He found that as many as eleven out of twenty-seven patients showed more or less greyness of the hair of the forehead and temple on the side on which they suffered most pain. In other cases the hairs become brittle or fall out.

Dr Fagge was once asked by a student to examine the hairs from his eyebrows with a microscope to see if any fungus was present. More than half of each eyebrow, at its outer part, had become denuded of hair; and this condition was more marked on one side than on the opposite. On inquiry he was found to be liable to migraine, and it was more severe on the side on which the eyebrow was more deficient. In a few weeks, under treatment for the neurosis, the hair began to grow again.

Another affection observed in persons subject to migraine is *xanthe-*

Mr. N. M. Gaul

lasma of the eyelids. It always begins near the internal canthus, generally in the upper lid. Mr Hutchinson found that most of the patients in whom he noticed xanthelasma of the eyelids had suffered from frequent sick headaches, some of them severely. Xanthelasma is, however, often seen in persons who have never had migraine in their lives. In the writer's experience it is far more closely related to chronic jaundice from whatever cause, than to sick headaches or headaches of any kind. (See table of cases in the 'Guy's Hosp. Reports' for 1877, 3rd series, vol. xxii, p. 129; in the 'Pathological Transactions' for 1882, vol. xxiii, p. 372, and third table, p. 383.)

The general *circulation* is interfered with in severe attacks of migraine. Möllendorff found the beats of the heart reduced to fifty-two or even forty-eight per minute. The pulse at the wrist becomes small and contracted, the hands and feet are cold, the face is pale and haggard, and dark circles appear round the eyes.

The side affected.—It seldom if ever happens that megrim keeps to the same side of the head in all its attacks in the same patient. As a rule, one side is more often affected than the other; but Tissot mentions the case of a lady who had it alternately on each side with great regularity. Wollaston and Sir George Airy were equally liable to the affection on either the right or the left side. Dr Latham describes the headache as beginning, and as more intense, on the side *opposite* to that on which the dimness of sight is noticed, but Dr Liveing concludes from the observations which he has collected that they often both occur on the same side. Both authors say that when the affection of sight and the numbness in the fingers are present together, it is on the same side of the body. Lastly, Dr Liveing has observed that when speech is affected in migraine there is very generally numbness in the fingers, and that this always affects the right hand, either alone or in association with the left. He has not met with a single instance when aphasia was present, in which sensation was impaired in the *left* hand only.

The following graphic account of a paroxysm of megrim is taken from the experience of an eminent living physician.

> The pain "is most usually fixed to one spot, or is more concentrated on one side than the other. It may commence as a dull pain over the forehead, then, as it increases, pass down to one eye and so to the temple, where it remains fixed. Exceptionally the pain is at the top or back of the head. The pain is sometimes so violent as to deserve the name of neuralgia, but generally it is somewhat duller and of a more sickening character. Its great peculiarity is the throbbing that occurs with each beat of the heart, aggravated by every movement of the body, and more especially of the head itself. The movements required for washing and dressing on rising can scarcely be endured. The sufferer walks slowly, since everything which tends to make his arteries beat a degree more violently adds to his misery. In his head he perpetually hears and feels 'throb, throb, throb,' and his only relief is to support the head against a pillow or rest it on the hand and avoid all possible excitement. His whole attention is distracted by the painful throbbing, and he becomes utterly incapacitated for business. Every movement, every word spoken, aggravates the pain; his only desire is to be let alone and be unspoken to. During this time he looks exceedingly ill, very pale, with dark margin round his eyes, and the pupils contracted; there is a general feeling of chilliness over the whole body excepting the head; the pulse at the wrist is feeble, whilst that in the head is strong. Anorexia is complete; the loathing of food so great that it is often impossible to swallow a single mouthful, and sometimes there is actual vomiting. Indeed the stomach often refuses food for twenty-four hours. The duration of a bad attack is generally several hours. If the person awake with it, the headache persists during the day, and it is only after another night's rest that he rises free. If it should come on during the day, it gradually increases in force, and then the night brings little comfort, for the throbbing, aching head entirely precludes sleep."

Pathology.—Megrim is included among the paroxysmal neuroses. As regards its anatomical seat, the disturbance which causes the affection of sight must occur somewhere above the optic chiasma. Wollaston thought that the origin of the hemiopia which he described would probably be found in the thalamus of one side, to which Liveing would add the corpora quadrigemina, and the ganglia of the sensory nerves, down to the nucleus of the vagus.

It seems, however, more probable that the visual centre in the angular gyrus is the seat of the disorder of vision, and the "nerve storm." The pain and vomiting are like those of meningitis, and may be referred to the cortex of the brain. The numbness and tingling, the disorder of speech, and impairment of memory, seem to begin in the sensory tract of the same side, and to spread upwards to the frontal convolutions. That the disturbance may also extend to the opposite side of the brain appears to follow from the numbness in the tongue and throat being sometimes bilateral, and the headache frequently so.

As to the exact nature of the change which gives rise to migraine, nothing certain is known, but it is most likely due to vaso-motor disturbance. Dr Latham (1872) supposes that in the early stage the affected side of the brain is anæmic from contraction of the blood-vessels of this hemisphere, and that in the stage of headache there is a secondary hyperæmia, consequent upon exhaustion of the vaso-motor apparatus. Du Bois Reymond had previously (1860) maintained that, at least in his own case, migraine was due to tetanus of the muscular coats of the arteries on the affected side. On the other hand, Möllendorf (1867) and Wilks (1869) believe that the complaint is caused by paralysis of the very same nerves, with dilatation of the vessels and consequent hyperæmia. Eulenburg thinks that both theories are right, and that certain cases are due to spasm, others to paresis of the vaso-motor nerves. Möllendorff lays stress on the fact (which had been pointed out a century ago by Parry, of Bath) that compression of the carotid on the affected side of the head often removes headache as if by magic, though only for a time. But diminishing the blood-supply to one side of the brain may very well suspend for a time the disturbance which is felt as pain, and yet that disturbance may not have been caused by an overflow of blood. Moreover, as Liveing points out, the statements of different observers with regard to the condition of the pupil are so diametrically opposed that no other inference seems possible but that it must really differ in different cases; while as for the dilatation of the temporal artery, the flushing of the face, the redness of the conjunctiva, the injection of the fundus of the eye, no one of them is constantly present; so that the only possible conclusion seems to be that all these vaso-motor phenomena are only accidental, not essential characters.

We may trace an analogy between megrim and an epileptic paroxysm on the one hand, and certain reflex movements on the other, such as sneezing, coughing, and gaping, to which may be added ejaculatio seminis—a short convulsion, as it has been termed, and in certain cases accompanied by a true epileptic attack.

At present we can perhaps form no clearer conception of an attack of igraine than that it is a "nerve-storm," or an "explosive discharge of rvous irritability."

Relation to other neuroses.—Liveing believes that a transformation metimes occurs between megrim and epilepsy; and he relates cases in which persons who had suffered from the former afterwards became affected with the latter disease; but it is to be noted that some of them had relations who were epileptic. He also refers to an instance in which migraine became replaced after a certain period by asthma; and to another in which a constantly recurring gastralgia disappeared, and was followed by a typical migraine, while this in its turn was succeeded by a kind of spasmodic croup. He also relates a case in which attacks of sick headache were followed after a time by angina pectoris; and another in which insanity developed itself.

Ætiology.—The *hereditary* character of megrim is well marked. Liveing found that in twenty-six cases out of fifty-three it was said to be a "family complaint;" and the twenty-six patients in question had among them forty near relations who were liable to it. In many cases it is transmitted without the slightest change of type, and sometimes it passes from a parent to those children only who in other respects resemble him. Sometimes, however, a daughter inherits megrim from an epileptic mother.

As to the relative liability of the sexes, *women* are certainly more prone to this complaint than men. Eulenburg believes that five women have hemicrania to one man. Gowers thinks the preponderance is much less.

The *age* at which it commences is often about the seventh or eighth year, but sometimes it is the period of puberty or of early adult life. It rarely occurs for the first time in a person over twenty-five or thirty. The more marked the hereditary tendency the greater the probability of its beginning in childhood. At about the age of thirty, persons often suffer from it much more severely than before; and, as Anstie remarks, at this period the attacks cease in many instances to be accompanied by vomiting, so that the complaint is no longer regarded as mere "sick headache," and the patient consequently seeks medical advice for what is now called neuralgia. When fifty years of age is reached, or somewhat earlier, the liability to migraine appears to cease, for old people seldom suffer from it.

In some persons the attacks of migraine recur with regular periodicity. The period is sometimes a fortnight, sometimes a month, sometimes much longer. There are, however, cases in which the complaint may return every day, or every other day, as regularly as the paroxysms of an intermittent fever; and this fact, together with the striking therapeutical influence of quinine, often makes it difficult to exclude the possibility of miasmatic infection. But these facts do not justify the conclusion that a given case of migraine is really "brow ague." It is probable that in districts where malaria prevails, migraine and other forms of neuralgia are often wrongly ascribed to that cause; and in some parts of Spain a miasmatic migraine is said to be endemic. Dr MacCulloch has stated that this kind of headache may occur as a substitute for ague during the whole of one relapse of the disease, and that he has seen a "double tertian" ague, in which the headache and the ague fit occurred regularly on alternate days.

It was at one time supposed that migraine, instead of being essentially a nervous malady, is the result of "bilious" disorder; and the vomiting was supposed to expel a "*materies morbi*," in the shape of vitiated bile. It is astonishing how firmly fixed is the belief in question. The truth is that it is a relic of one of the most ancient doctrines in the history of medicine, that of the four Cardinal Humours, one of which was "yellow," and another "black" bile.

Although, however, it is certain that migraine is never solely due to disorder of the chylopoietic viscera, there is no question that some error of diet is often the direct exciting cause of an attack in a person who is liable to it. No doubt the diffused headache and giddiness which are apt to be more or less present in persons who suffer from dyspepsia or from so-called congestion of the liver have been often confounded with true migraine; but what is conclusive of the influence of indigestion is the fact that some persons can bring on an attack of sick headache by eating particular articles of diet towards the end of the interval between one paroxysm and another. Fothergill nearly a century ago stated that he had found nothing more apt to cause sick headache than "melted butter, fat meats, spices, meat pies, hot buttered toast, and malt liquors when strong and hoppy." A medical man who had suffered all his life from the complaint told Dr Liveing that he could never take the smallest quantity of wine nor eat the smallest fragment of pastry without bringing on a headache. Many persons speak of butter and pork as particularly frequent exciting causes of migraine; and, making every allowance for the influence of preconceived opinions, it seems likely that such statements are not all imaginary. That an attack of indigestion is not the "cause" of migraine is obvious, for even those persons who can produce a fit at pleasure by eating forbidden food when they have been free from attacks for a considerable period, find they can eat what they please without risk for a few days after the last paroxysm. As with epilepsy and some forms of mania, and one may add some kinds of ill-temper, of drunkenness, and of sexual vice, so in migraine we must recognise the discharge from time to time of accumulated nervous "irritability" by a stimulus which is almost accidental.

In the case of women, the recurrence of the *catamenia* is often an exciting cause of attacks of migraine, which, perhaps, generally precede the flux, but sometimes accompany or follow it. Not infrequently each monthly period brings with it a series of more or less distinct paroxysms. Liveing relates the case of a woman who was very liable to the complaint when menstruating, but who throughout repeated pregnancies was always entirely free from it.

The immediate exciting cause of the paroxysm is often excessive fatigue. Thus a patient of Dr Fagge who was employed at a bank had an attack every week-day, but was free on Sundays. In some persons a straining effort, such as lifting a heavy weight, will bring on a fit of megrim, in others the exertion of running. Many women are liable to be attacked after a hard day's washing, or after a long walk. A long railway journey is apt to be followed by a paroxysm in some ladies, and in others merely driving in the streets of London has the same effect. Many persons always have a sick headache after a day's sight-seeing, or after passing an evening in a crowded concert-room or ball-room; and in some susceptible patients an attack may be brought on by glaring lights, loud noises, or strong odours. Dr Airy mentions the case of a person in whom teichopsia was occasionally caused by looking at a striped wall-paper or a striped dress; and Sir John Herschel found that he incurred an attack if he allowed his mind to dwell upon its symptoms.

In several of the conditions already mentioned as exciting causes of migraine, one element is *visual exhaustion;* and this is true not only of overstudy, but also of railway travelling and the like. Many years ago, Piorry propounded the theory that the complaint, or at least one variety of it, was the result of straining efforts to see very small objects, or of want of care in regulating the amount of light. This view is quite untenable if applied to all cases of sick headache; but it is perfectly true that when the eyes are structurally imperfect the forced effort to use them may be the immediate cause of attacks of migraine. The defects which lead to this result are those of the refracting media of the eyes; their direct effect is the production of spasm of the ciliary muscles, and with this is associated an irritation of nervous filaments, which may diffuse itself over a wide area within the distribution of the fifth nerve. Every practitioner now knows that hypermetropia is a frequent cause of attacks of dimness of sight, headache, and giddiness, which recur when the eyes are used for near work for any length of time, particularly under artificial light. A student suffering from similar symptoms discovered that they were the result of the employment of too powerful concave glasses, which he had chosen without proper advice, in order to correct slight myopia, and which he wore even when reading or writing. In this connection, too, astigmatism must not be overlooked.

Another cause of headache and giddiness is weakness of the internal recti muscles. In practice, therefore, one should make it a rule never to prescribe for any kind of frontal headache without thinking of the possibility that it is due to imperfection of the eyes.

Dr Fagge once saw a bank clerk who had previously been compelled to give up work for a period of two or three months on account of cerebral symptoms. These had been thought very serious; but on examination by Mr Higgens it was discovered that the internal recti muscles failed to make the eyes converge properly upon near objects; and when suitable glasses were supplied he soon lost all his complaints.

Lastly, affections of the *teeth* must not be overlooked as causes of migraine, at least if clavus be included as a form of it.

Mr Salter records the case of a young lady, who for eight years was subject to attacks of headache, confined to a space of about the size of a crown piece, rather to the left of the vertex. They sometimes recurred three or four times a week, beginning after breakfast and lasting all day; they were attended with great prostration. The affected spot became hot, and pressure with the hand gave relief. At length the patient fancied that the left upper canine tooth, which was known to be impacted in the palate, was in some way connected with her sufferings. It was removed, and she never afterwards was attacked by the headache.

Diagnosis.—In its typical form the recognition of migraine presents no difficulty. The remarkable visual phenomena, the locality and severity of the pain, the vomiting and the complete recovery, together with the patient's experience of similar attacks, make a well-marked case of migraine one of the most characteristic and unmistakable of diseases. A case might occur in which embolism produced aphasia, sickness and headache,* or in which a tumour or abscess of the brain might simulate the symptoms of migraine, or where syphilitic periostitis of the margin of the orbit might produce a pain like brow-ague; but careful observation would speedily discover the real nature of such cases.

The real difficulty lies in the fact that comparatively few cases exhibit the regular and exact combination of symptoms, as above described. Not only may one or other of the exceptional symptoms introduce an element of doubt, but one of the principal and most constant symptoms may be absent.

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* Several months before his death the late Dr J. J. Phillips had a severe attack of headache, attended with marked aphasia. When he had recovered, I one day happened to discuss with him the question whether it could have been of the nature of migraine; but his fatal attack of apoplexy, which doubtless was the result of embolism from mitral disease, began in precisely the same way, and with the same symptoms.—C. H. F.

or so slightly marked as to make one doubt whether the missing link may not have been supplied by our own preconceived notions, or by the patient's expectation, or by her acquiescence in the statements implied by our questions. We have seldom the opportunity of verifying the accuracy of our diagnosis, for migraine is not a fatal disease; and if it were, we should not know what lesion to look for to prove or disprove our conclusions. As in the similar cases of epilepsy, chorea, and angina pectoris, the verification of our diagnosis depends on return of the malady after an interval of health, and return with the same characteristic features; but this does not always take place, and when it does we are not always there to observe it. Megrim is not a disease of hospital wards, but of the out-patient room and of private practice, and is often only seen a single time, after the paroxysm is over. Like many other functional disorders, its elucidation does not depend so much on new apparatus or improved pathology as on the combined observation of those engaged in family practice.

The present writer has found that patients with migraine seldom describe the *teichopsia* with even approximate accuracy: they often vomit early instead of late in the attack, and they sometimes complain of the giddiness more than of any other symptoms; while the seat of the headache varies in different patients, and even in the same patient in different attacks. Nevertheless, the more intelligent and observant patient will distinguish her sick headache from "ordinary headaches," and also from true facial neuralgia.

In addition to the symptoms already detailed, the most important points for diagnosis of one-sided megrim from neuralgia are: the greater length of the attacks, the fact that food is disliked, and, if taken, only adds nausea to the pain, and that stimulants aggravate the distress. On the other hand, the worst cases of megrim are never so prolonged, so severe or inveterate as those of *tic douloureux* of the form described in a previous chapter (p. 556). It must be remembered that some reputable authors—Tissot, Hasse, Lebert, and Anstie—have actually defined migraine as neuralgia of the fifth pair of nerves.

The diagnosis from mere headache rests (apart from the optical symptoms and vomiting) upon the seat of the pain, which is sometimes unilateral, sometimes confined to the temples, but never occipital; secondly, on the attack beginning with waking or early ~~in the day,~~ on its lasting a whole day or sometimes two days, but not longer, and on its then rather suddenly disappearing. Moreover, the aggravation produced by attempts to read and the relief afforded by darkness are much more marked than in ordinary headaches, even when severe.

Prognosis.—The forecast in typical cases of migraine is not very favourable. It never proves fatal, nor does it lead to other and more dangerous disease; but we have only limited power of either cutting short an attack or preventing its return. On the other hand, as life goes on, its attacks usually become less severe and less frequent. In this respect, as in its incidence on different periods of life, we have a much closer resemblance of migraine to rheumatism than to epilepsy. Moreover treatment, though it cannot abolish, can as a rule much alleviate the sufferings of a paroxysm, and may sometimes retard its return. Hereditary cases and cases beginning before puberty are the most obstinate, and those in men more so than the more common ones in women.

Probably no physician can have suffered again and again from migraine without the thought being forced upon him that such attacks must indicate some serious cerebral defect; and Calmeil believed that both the intellectual faculties and the moral disposition of the patient are sometimes impaired by repeated occurrence of the disease. Liveing refers to the cases of Parry and Wollaston, both of whom, after having long been subject to this complaint, died of organic cerebral disease. Such results, however, are extremely rare and probably accidental. No decay in mental power resulted in the illustrious sufferers from this distressing malady mentioned above (p. 900), and the whole College of Physicians will bear witness to the integrity of the "intellectual faculties, and the moral disposition" of the accomplished physician who gave expression to this foreboding.

Treatment.—This includes the management of the patient during the intervals between the attacks, so as to postpone their occurrence as long as possible; and the treatment of the paroxysm itself.

Under the first head hygienic measures are of primary importance. One must insist upon the importance of daily exercise in the open air; one must shield the patient as far as possible from domestic anxiety, and diminish the amount of both reading and writing, especially reading small print and deciphering illegible letters by artificial light. The state of the digestion must be inquired into, and due weight allowed to any indications that the attacks are brought on by errors of diet. But in many cases it is a mistake to restrict the patient to what is termed plain food; rather he should be advised to take more hydrocarbons in the form of butter or cream. Stimulants should generally be avoided, particularly ardent spirits; but malt liquor, and particularly stout or porter, are often very useful. In some cases a change of climate, even from the seaside to London, is found to be beneficial; and in others a long voyage will break the habit of years.

The family physician should look out for the early manifestations of migraine during childhood and puberty, and insist upon the avoidance of over-study and of undue excitement in those whose parents have suffered from the disorder. Migraine was once looked upon as being at once incurable, and almost unworthy of notice by a physician. But the truth is that if systematically taken in hand it is not unamenable to rational treatment; and those who suffer from it know best how serious a matter it is, interrupting, as it frequently does, both the pleasures and the duties of life.

Among drugs Liveing says that he has sometimes found the regular administration of belladonna and hyoscyamus of great service.

In many cases the preparations of iron are serviceable, and this even though there is no anæmia. Arsenic was long ago recommended by Dr Bright and Sir Thomas Watson, and Dr Fagge used to prescribe it with marked success. Quinine is much less serviceable in megrim than in true neuralgia, if we exclude neuralgic headaches of malarious origin.

Todd is said to have found iodide of potassium more successful than anything else; and Liveing mentions a case in which the attacks were so frequent and severe as to render the patient's life a burden to him, and in which five grains of the iodide three times a day set him almost free from the burden. In other cases chloride of ammonium seems to answer better than anything else. Valerian and valerianate of zinc were found useful in his own case by Fordyce, and Liveing has sometimes found them of great value.

Perhaps, however, the most generally useful drug to prescribe in the intervals of megrim is potassium bromide, and scarcely less so is nitro-glycerine. Gowers recommends the former as most efficacious when, during the paroxysm, the face is flushed, and the latter when it is pallid. Either of them should be given in a small dose, the nitro-glycerine after food in alcoholic solution, and the amount pushed until some effect is seen in lengthening the interval between the attacks, or in diminishing their severity.

In the paroxysms of migraine the patient of his own accord will keep in a darkened room and maintain absolute quiet. If the feet be cold, they should be placed in hot water, quickly dried, and wrapped in blankets. Chloral hydrate may then be administered in a dose of from twenty to thirty grains. Anstie speaks of it as being of the greatest possible value in quickly bringing sleep to the patient, who when he wakes up may be free from pain. In some cases, and particularly when connected with neuralgia of dental origin, butyl-chloral hydrate (or as it used to be called "croton-chloral") is still more decidedly efficacious. Bromide of potassium also is very useful; a scruple, or half a drachm, taken when the sight begins to be affected, sometimes cuts short the attack. At this early stage of the attack a cup of strong tea, particularly green tea, or black coffee will in some cases have the same effect, or even sipping hot water. Guarana—prepared in Brazil from the seeds of *Paullinia sorbilis*—has been recommended on his own experience by Sir Samuel Wilks ('Brit. Med. Journ.,' April 20th, 1872): half a drachm of it may be taken mixed with water; or about twenty or thirty minims of the liquid extract. It contains the same alkaloid, caffeine, which is present in tea and coffee.

Phenazone (antipyrin) in doses of from five to ten grains, and phenacetin in half the amount, have been much used for sick headaches, and in some cases undoubtedly cure the pain. The patient should take it in hot brandy and water, and lie down for half an hour before taking a second or third dose; but if it fails, another remedy should be tried at once.

The writer has seen great benefit result from administration of fifteen drops of the tincture of cannabis indica, more in the paroxysms than in the intervals. Anstie recommended it in doses of a quarter to half a grain of the extract. It is occasionally so strikingly successful that one is the more disappointed with its frequent complete failure, and no one seems to know when it is likely to be efficient.

On the theory of contraction of the arterioles of the brain being the immediate cause of the symptoms, nitro-glycerine has been recommended in the paroxysm; but Gowers thinks that its usefulness is greatest in the intervals or at the very beginning of the attack, and that it does harm rather than good if taken when it has developed. This would agree with Dr Latham's theory of initial spasm followed by continuous dilatation of the arteries.

A purely empirical method is to give a dose of blue pill or calomel on the first symptom of the paroxysm, and there is no doubt that some patients find it efficacious.

Locally some measure of relief may be afforded by the pressure of a handkerchief tied tightly round the forehead, the remedy prescribed by Prince Arthur for Hubert. A more modern treatment is to apply to the eat of the pain a piece of cotton wool, on which a few drops of ether have been poured, and to cover it with a watch-glass; bisulphide of carbon has been used in a similar manner. Some patients find great relief from rubbing a stick of menthol into the skin over the seat of pain; Trousseau speaks highly of the application of extract of belladonna to the temple; and Anstie found a diluted ointment of veratria serviceable.

The last physician, himself a sufferer from migraine, advised that the constant galvanic current should be passed from one mastoid process to the other, employing only three or four cells at first, and never more than ten; it should be applied for but half a minute at a time, and not more than once or twice a day. Galvanising the cervical sympathetic cord is a procedure recommended chiefly on theoretical grounds. It is doubtful whether the theory is a true one, doubtful whether the effect intended is produced, and most doubtful of all whether any practical benefit is obtained by the treatment.

Headache.—This seems the most convenient place to notice what is rather a symptom than a disease, and yet one which is so common and occasions such interruption to comfort, that every practitioner is daily brought into contact with it.

The pathology of headache is quite unknown, and it does not seem likely that it ever will be ascertained, particularly if the symptom is really caused by very different conditions even when its characters appear to be the same. It may, however, be usefully regarded (1) as a symptom of organic disease; and (2) as a functional neurosis.

In the former capacity we have found that it is an almost constant symptom of cerebral tumours, cerebral abscess, and cerebral meningitis, including pachymeningitis. Headache is exceptional in cases of cerebral hæmorrhage or thrombus, but frequent in those of embolism. The worst headaches are caused by syphilitic disease of the membranes, gummatous meningitis; in these severe cases sleep is entirely prevented, and the pain is accompanied by vomiting and vertigo.

Neuralgia and megrim have been already described, and their symptoms are very different from those of ordinary headache.

The clinical varieties of headache may be stated as follows:

The headache of injury, concussion, or fracture of the skull, is usually not severe, and accompanies more urgent symptoms. The headache of sea-sickness is probably due to concussion of the brain.

Headache from anæmia is, as a rule, vertical or occipital. It is observed among the symptoms of chlorosis, leuchæmia, etc., in cardiac disease, and in phthisis, but is often absent where we might expect it. It is almost always relieved by lying down.

Headache is often due to ocular fatigue from efforts to focus the lens. This is the headache produced in healthy persons by seeing pictures; and in children who have to learn their lessons while hypermetropia, myopia or astigmatism are uncorrected.

Nervous headache is a vague term most properly applicable to the headache of fatigue—of long railway journeys, of a day's shopping in London, and of sleeplessness.

Toxic headaches are represented by the morning headache after a debauch, and the headache of constipation and dyspepsia. They are chiefly frontal, and usually of "massive" rather than acute character. Such headaches are often called bilious, but without evidence; in icterus headache is certainly rare, and probably the toxines which presumably cause this form of headache are produced in the colon rather than in the liver. In the same large ætiological group of toxic headaches must be included the severe pain, chiefly in the forehead and temples, which is one of the most distressing symptoms of uræmia.

The headache of pyrexia is severe, massive, and diffused: not sharp and limited in range like a neuralgia. It does not follow the degree of fever, and is usually absent during hyperpyrexia—in rheumatism, for instance, and in pneumonia. It is most severe in influenza, where it is referred to the forehead and "back of the eyes," in smallpox, in typhus, in scarlatina, and in the earlier stages of enteric fever. In the pyrexia of measles, phthisis, rheumatism, pyæmia, pleurisy, pneumonia, it is frequently present, but is less constant, and when present less severe as a rule than in the fevers before mentioned. In ague true headache is present in the hot stage, but relieved when the sweating appears; and it is probably of different origin from the "brow-ague" or severe unilateral neuralgia which is one of the sequels of malaria. Among toxic headaches may also be mentioned those due to poisoning by lead and by opium, those caused by quinine, by iron, by inhalation of ether, and by the nitrites.

The diagnosis of headache consists first in separating the functional cephalalgia from that due to organic disease of the brain, from neuralgia, and from megrim. Headache, in the sense here used, is never confined to one side, and is seldom confined to one region. It is not accompanied by vomiting, and however severe preserves the massive, voluminous character in distinction from the thin, acute, high-pitched, pain of neuralgia. Headache may be compared to the "growling" of a toothache, neuralgia to the contact of a dentist's instrument with an exposed tooth pulp. Headache, again, never comes on suddenly, and seldom departs otherwise than gradually. Lastly, mere headache, as Wilks long ago observed, does not keep people awake: a pain that does so is more than headache.

Secondly, we must try to distinguish the cause of the headache in each case. When not a symptom of fever, of uræmia, or some other of the diseases enumerated above, headache is most often due to fatigue and want of sleep, to ocular disorders, to anæmia, or to constipation and dyspepsia.

When with a throbbing headache the tongue is furred, the last is probably the cause, particularly if there is a sallow aspect and an unpleasant taste in the mouth; and if this is combined with a hard pulse, thirst and nausea, and loss of appetite, or with morning diarrhœa, we shall seldom be wrong in supposing that our patient is suffering from the effects of free-living. A blue pill and a black draught, followed by alkalies and Euonymin, will usually set him right, and the obvious preventive measures must be urged upon him.

When there are dark circles round the eyes, dilated pupils and pale cheeks, with a quiet, soft pulse, we shall usually find that the headache is due to working or watching through the night, prolonged hours of railway travelling or of study, or sometimes to insufficient food. The remedies here are bromide of potassium, sulphonal or chloralamide—hypnotics, not anodynes, —fluid nourishment, and sometimes wine.

When the headache is worse after reading, and absent in holidays, when the patient is a child, and neither anæmic nor "bilious," we may be almost sure that examination of the eyes will show some error of refraction; and if this is ascertained and corrected the distressing headaches disappear.

When there is marked anæmia, the iron, which in cases of constipation would aggravate the headache, becomes its best cure, and this even if it is necessary to combine it with aloes or with magnesium sulphate, as it almost always is in cases of chlorosis.

When, on the other hand, there is plethora, the throbbing headache requires a different treatment. This kind is seen most characteristically in women when the menopause begins. An occasional saline purge with senna, helped by iridin, euonymin, or calomel, is of great service against headache during the whole climacteric period.

The ordinary headache of constipation, more common in women, but frequent in both sexes, and from which no age is exempt, is best treated by exercise and diet, with as little of purgative drugs as possible. Like the last form, it is often throbbing, and is always aggravated by stooping or coughing.

The headache of hysteria in women (and of the so-called neurasthenia in men, which is usually a compound of self-indulgence and pusillanimity) should not be treated with drugs, but by moral discipline, by fresh air, abstinence, cold sponging, and the awakening of unselfish interests.

EPILEPSY

WITH OTHER PAROXYSMAL NEUROSES

> **Quin etiam, subito, vi morbi sæpe coactus,**
> **Ante oculos aliquis nostros, ut fulminis ictu,**
> **Concidit et spumas agit; ingemit et tremit artus,**
> **Desipit, extentat nervos, torquetur, anhelat**
> **Inconstanter, et in jactando membra fatigat.**
> **Lucretius.**

finition of Epilepsy—E. major—aura—fit—E. minor—Status epilepticus—Sequelæ—Pathology, seat, and physiology—Ætiology—Relation to other forms of Eclampsia—Diagnosis—Prognosis—Treatment of the fit and of the intervals.
roxysmal vertigo—Auditory vertigo and Menière's disease—Paroxysmal mania.

Synonyms.—'Επιληψία or 'Επίληψις (ἐπιλαμβάνω), *i.e.* a seizure (Hippo-ates, Aristotle)—Morbus comitialis (Celsus, Seneca, and Pliny), so called, cording to the grammarian Festus, because the Roman assemblies were oken up if anyone present was attacked by a fit—Morbus sacer (Cælius relianus).—*Anglice,* Falling Sickness.—*Fr.* Haut Mal.—*Germ.* Fallsucht.

Definition.—Convulsions with loss of consciousness, recurring in par-ysms; an idiopathic disorder of the brain, without local organic lesions, or her known cause.

Convulsions which closely resemble the fits of idiopathic epilepsy may company the onset of variola or other exanthems in children; they occur ortly before death by hæmorrhage; they are important complications of ight's disease and of the puerperal state; and we have seen that they are mptoms of tabes, tumours of the brain, and general paralysis of the in-ne. These epileptiform fits are conveniently called *eclampsia.*

In remarkable contrast to these secondary fits are primary paroxysmal acks, which return again and again for years together, without being ceable to any cause, except in some instances an inherited tendency. h cases seem to be essentially distinct from those which are symptomatic, asional, and irregular; they differ from them in symptoms also, and [illegible] therefore [illegible]

[illegible]

[illegible] as usual.

[illegible] corresponding [illegible] *aut mal*, [illegible] as epileptic [illegible]

Premonitory.—Before an attack of the *grand mal*, some [illegible] sensations which afford a warning of the seizure. To [illegible] the name of *epileptic aura* is given by a convenient extension of the term [illegible] the feeling as of a draught of air passing over the [illegible] reached the head, and the patient became insensible. Sometimes [illegible] or dragging sensation is experienced, which begins in the fingers [illegible] and [illegible] the toes of one foot, and rapidly moves upwards. [illegible] case in which it first affected the wrist, and then spread towards [illegible] of the fingers. Ratcliffe had a patient in whom it was [illegible] always referred exactly to the foramen caecum at the base of the [illegible] Other cases are recorded of an aura in the form of pain at the [illegible] with nausea. Nothnagel mentions an instance in which numbness [illegible] the right shoulder and side of the head, and [illegible] the right leg, and the right half of the [illegible] cases [illegible] corresponds with the [illegible] Sir Thomas Watson quotes a case in which [illegible] in the side, and a patient of Sir Samuel Wilks [illegible] spot on her face.

In some instances it would seem that an epileptic aura [illegible] of the vasomotor nerves of some part of the body. The [illegible] experiences a sensation of coldness or weight in a limb, and [illegible] found on examination to be pale and cold to the touch, and [illegible] Trousseau says that when an aura occurs [illegible] swollen, so that the rings on [illegible] become suddenly [illegible] tight.

In some cases an epileptic attack is preceded by a profuse secretion of [illegible] of sweat, as in several cases that came under N[illegible] observation.

What has been termed a "motor aura" may take the form of [illegible] spasms. The eyelids may twitch, or some muscles of the [illegible] or more complex movements may take place, the patient running round, or running to meet, so to speak, the impending attack.

Again, the epileptic aura may be referred to one of the special senses. Joseph Frank is said to have met with an instance in which it always took the form of a sweet taste in the mouth. Still stranger is Dr Gregory's case, quoted by Watson, of a man who always fancied he saw a little old woman in a red cloak; she seemed to come up to him and to strike him a blow on the head, whereupon he lost all recollection and fell down. Sometimes the only warning is a vague sensation of fear. Reynolds was told by a gentleman that what always passed through his mind was, "This is what I had foreseen. I knew it would come on here; I ought to have avoided it by remaining away"— although in reality he had not suspected that a fit was impending.

An epileptic aura may last for a few seconds or for several minutes. Nothnagel mentions that when it has lasted more than ten minutes, he has been able to ascertain that there was distinct loss of sensation in the part affected. Occasionally it passes away without being followed by a fit. In one of his patients a vaso-motor aura in one leg sometimes occurred six times daily, whereas she had only about one epileptic attack in a week.

*Symptoms of the fit.**—An attack of the *haut mal* begins by the patient suddenly—almost instantaneously—falling unconscious, with strained and rigid muscles. As he is seized, he often utters a sharp, shrill cry; but he may be perfectly silent, the respiratory movements being arrested by spasm; or he may emit a smothered groaning sound. In most cases the face becomes ashy pale, and although the heart goes on beating, the radial pulse is sometimes imperceptible.

The *tonic spasm* which occurs at this period of an epileptic fit is peculiar; it usually affects one side of the body more than the other; the head is turned round towards one shoulder, and the eyeballs are strained in the same direction. The pupils constantly become dilated; but Reynolds in one instance observed a momentary contraction before dilatation began. They are generally, if not always, insensible to light; and touching the conjunctiva commonly excites no reflex movements, though Romberg once saw it followed by closure of the lids.

Soon the face flushes, and acquires a dull red or dusky hue. This appears to be partly due to the fact that the large veins of the neck undergo compression, and that the flow of blood through them is interrupted by the spasmodic contraction of the sterno-mastoideus and neighbouring muscles; but spasm of the glottis may also occur.

The stage of tonic spasm may last only two or three seconds, or it may be prolonged to thirty or forty seconds. Trousseau says that in some rare cases it lasts as long as two or three minutes.

Next follows a stage of *clonic spasms*. These also are generally more marked on one side than the other. The fingers of one hand are alternately flexed and extended; the like movements simultaneously occur in the other joints of the arm, and in the corresponding lower limb, while the eyes are twitched towards the affected side. The opposite limbs may escape entirely, or be affected with less powerful convulsions. The pupils sometimes oscillate between contraction and dilatation. The jaws are suddenly and forcibly closed, and the tongue is often caught between the teeth and bitten on one side. The blood is mixed with the saliva that is poured into the mouth; and a red foam is sputtered through the clenched teeth. The face remains of a livid purple hue. There is often profuse sweating; urine may escape from the bladder, and emission of semen or defæcation may occur.

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* "Inter notissimos morbos est etiam is qui *comitialis* vel *major* nominatur. Homo ubitò concidit: ex ore spumæ moventur: deinde interposito tempore ad se redit, et per se se consurgit. . . . . Modo cum distentione nervorum [*i. e.* convulsions] prolabitur aliquis, odo sine illâ." (Celsus, 'De Med.,' lib. iii, cap. xxiii.)

This second stage—that of clonic spasm—does not usually last above two or three minutes. Trousseau says that it scarcely ever continues more than four minutes: as he remarks, one is sure to think it longer unless one reckons the time by a watch. The convulsive movements become less frequent and severe, and are no longer wholly meaningless. The patient often draws a deep sigh; he tries to change his position, and looks about him with a bewildered stare.

When the fit passes off, the patient may at once regain his consciousness; or he may be more or less confused for a time; or, more frequently, he becomes drowsy and passes into a deep sleep for an hour or more, a sleep that may have the character of stupor or even of coma. During this time, if he can be roused at all, he is generally peevish. Slight clonic spasms not infrequently recur.

When an epileptic fit occurs during sleep, one can often make out its real nature by the fact that the tongue next morning is found to be bitten, or that the urine has been passed involuntarily. As Trousseau taught, if a patient who had never before had difficulty in retaining the contents of his bladder at night, should now and again find that he has wetted his bed, this mere fact should arouse the fear that he may be an epileptic. Dislocation of the shoulder, discovered on waking in the morning, may signify an epileptic fit during sleep. A similar inference may sometimes be drawn from the presence of minute scattered red petechial spots, like fleabites, on the forehead, throat, and chest. Wilks recorded a case in which these petechial hæmorrhages, following a first epileptic attack, were taken for an exanthem.

Another immediate and transitory sequel of an epileptic fit is albuminuria. In some patients this is a rule, in others it is seldom or never observed. It appears to be the result of congestion of the kidneys when respiration is long suspended, like the albuminuria of cardiac disease, of cyanosis and of suffocation. It must be carefully distinguished from that due to chronic Bright's disease, the cause and not the result of the attack.

Exceptional forms.—An attack of major epilepsy does not always conform strictly to the above description. It is sometimes attended with but slight convulsive movements, or possibly with none at all. Such cases were formerly described as examples of a form of apoplexy, or as "apoplectiform cerebral congestion" (*supra*, p. 754).

If Jacksonian convulsions (*supra*, p. 775) occur in genuine epilepsy at all, they are exceedingly rare.[*] With these very rare exceptions, whenever attacks of clonic spasm recur paroxysmally without loss of consciousness, there is local organic disease of the brain, *i. e.* the disease is symptomatic eclampsia, not idiopathic epilepsy.

Epilepsia minor.—A paroxysm of the *petit mal*, or minor epilepsy, may be a mere suspension of consciousness, sudden in its onset, and scarcely more than momentary in its duration. For two or three seconds the patient is lost, but he quickly recovers himself, and goes on with what he is doing. Wilks mentions the case of a shopman who often had a seizure of this kind while serving a customer, and who believed that no one noticed it. In some instances such attacks are attended with muscular rigidity or so-called catalepsy. The late Dr T. K. Chambers relates how a well-known lecturer would sometimes be seized while addressing his class, and would stop in the middle of a sentence, remaining perfectly still, with mouth open and arms extended; after a moment he would go on just where he left off without knowing that anything had happened. He afterwards developed epilepsy of the major form.

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[*] Reynolds, who gives this form of the disease the name of "abortive epilepsy," furnishes a list of references to various writers as having described it. But after looking at most of them I am not sure that they took care to exclude cases of cerebral tumour.—C. H.

In other cases, if the patient should happen to be speaking when he is attacked, he loses the thread of his discourse, and afterwards cannot remember what he was saying. He may suddenly feel a sensation of giddiness and stagger, or grasp at support. Wilks mentions the case of a little girl sitting in her chair stitching, when she would suddenly fall, but before the ward-nurse could pick her up she would be in her seat and again at work. To such cases the name of "epileptic vertigo" would apply if there were not another *paroxysmal vertigo*, which does not bear so close a relation to epilepsy (v. *infra*, p. 931).

If we have the opportunity of watching an attack of minor epilepsy we may notice that the patient's pupils dilate, and that he no longer seems to be looking at anything. The face often becomes pale, and afterwards slightly flushed. Reynolds says that the pulse may falter and become irregular, and Moxon observed the same thing; but it is quite the exception.

Some years ago, while I was one day listening with the stethoscope to the heart of a man whom I had never seen before, its beats suddenly ceased. I looked up and saw that his face had turned deadly pale. He said that he was going to faint, and reclined back on the chair from which he had risen. I could feel no pulse at his wrist, and for an instant I thought that he might be going to die, but I had hardly time to ring my bell when the colour returned to his face, and I found that his heart was beating again. Presently there was a little twitching of the muscles in one or both hands. In a minute or two he was able to tell me that he often had "fainting fits," and that some years back he had been subject to epileptic seizures. It appeared clear that the attack which I had witnessed was one of minor epilepsy.—C. H. F.

In a majority of cases the so-called fainting fits which are apt to occur in children are really of an epileptic nature. It is, however, certain that the circulation in the face and limbs often goes on without interruption during an attack of the *petit mal*, as Reynolds testifies from repeated observations.

Not infrequently an attack, which is in all other respects one of "minor epilepsy," is accompanied by some slight convulsive movement. There is a transient strabismus; the mouth is drawn to one side; the whole head is urned towards one shoulder; or the body generally becomes for an instant gid. Cases of this kind form links between the two chief varieties of the isease, and show how closely they are related to one another. A further roof is the fact that in perhaps the majority of cases in which attacks of e *petit mal* occur again and again, they are after a time replaced by those the *haut mal*. Or the patient may suffer alternately from the one and om the other.

Recurrence.—The frequency with which the attacks of epilepsy return ries greatly in different cases. A patient may have one fit without ever ving a second; or after an interval of two or three years he may be ain attacked. In some cases the paroxysms return once, or twice, or e times a year; in other cases more or less regularly once a month. women they are, on the whole, more apt to occur at the catamenial iods than at other times. But Reynolds says that monthly recurrence is lly more common in the male than in the female sex; and it is very e indeed for a woman to be liable to epileptic fits only during menstruation. More frequently the interval at which the attacks recur is less than a month, and in severe cases there may be one paroxysm or more every day.

Sometimes several fits occur on the same day or within a week, and then the patient is free for a month or longer.

When they return with great frequency during a lengthened period, they almost always belong to the minor form of the disease; in other words frequent and severe attacks soon prove fatal.

In one terrible form of the disease the fits follow one another in rapid succession, so that before the patient has recovered from one another comes upon him. This has been called by French physicians *état de mal épileptique*, and in England *status epilepticus*. In the worst cases the convulsions follow with extraordinary rapidity; Dr F. Brock once counted upwards of a thousand fits before consciousness returned. Crichton Browne in describing such cases says that the limbs are scarcely at rest after one fit before they are contorted by another, and even in the intervals there are frequent muscular twitchings. The patient lies perfectly unconscious; his heart beats rapidly; his respiration may either be quick and shallow or slow and laboured. His features are swollen and livid, and his lips purple. His body is bathed in profuse perspiration, and the temperature is raised to 105°, or even higher; Dr Merson found it in one case 107·8°. This high temperature is very grave, yet Charcot knew of a case in which 105·8° was reached without death ensuing.

Complications and sequelæ.—Certain occasional results of an epileptic fit have yet to be mentioned. Todd described a loss of muscular power in one arm, or in both the arm and leg on one side, under the name of "epileptic hemiplegia;" but probably such cases are Jacksonian eclampsia, not true epilepsy.

One must not forget that diminished mobility of the arm after a fit may be due to dislocation of the shoulder, produced sometimes by muscular spasm, sometimes by the patient having struck that part in falling.[*] At one time a woman who was liable to epileptic fits used frequently to come to the hospital to have her shoulder set. Still more serious injuries sometimes occur during a paroxysm; the skull may be fractured by the patient dropping down on the pavement, or he may be severely burnt by falling against the bars of the grate, or he may be run over in the street. Persons who are liable to epilepsy cannot be too closely looked after.

Mental disturbance accompanying or following epileptic fits is one of the most important occasional symptoms. In a person subject to *haut mal* the attack may be replaced by one of paroxysmal mania. Sometimes, again, although the patient falls down in an epileptic fit, he quickly gets up again and attacks those about him with the utmost violence, and if there were no witnesses of the beginning of the attack, the patient may be liable to a criminal prosecution. In other cases the epileptic stupor is succeeded by maniacal delirium, during which suicide may be committed, or murder.

More frequently a fit is followed by loss of memory, incoherence of ideas, and perversion of intellect, which may last several days. It is not surprising, therefore, that when the paroxysms recur frequently and at short intervals the mental powers become permanently impaired. Patients who have been long subject to epilepsy acquire a peculiar dull, heavy aspect; and iis, with their widely dilated pupils, sometimes enables one to recognise iem at once. They are apt to be irritable, morose, and gloomy, particurly just before the epileptic attacks; a fit seems to give temporary relief the brain, and the patient afterwards feels lighter and more cheerful ian for a long time before.

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[*] I myself once nearly overlooked this accident. A patient came with her arm hanging helpless. I was pointing out to the students that a transient paralysis often follows an epileptic attack, when I happened to notice that touching the limb gave pain. I grasped the deltoid muscle, and found that the head of the humerus was out of place.—C. H. F.

Impairment of the intellect is by no means confined to patients who ave suffered long from epilepsy. In children a series of fits, continued for few successive hours, often produces a permanent state of imbecility, or of iania. A considerable proportion of those who are admitted into asylums r idiots are children who were born with full powers of intelligence, and arned to talk as soon as others; but, having been attacked by epilepsy hen perhaps four or five years old, they have since lost their senses, and ecome dirty in their habits, violent in temper, and unable to recognise their arents. Such cases are frequently brought to our out-patient rooms.

In adults permanent impairment of intelligence occurs only when epipsy has been of long standing. Indeed, it is believed that some persons etain their full vigour of mind after having been liable to fits for years. he instances of Julius Cæsar, Mahomet, Petrarch, Peter the Great, and apoleon are often cited to prove that the repeated occurrence of epileptic ttacks does not always injure the intellectual powers; but in each of these ases the fits were only occasional, and the evidence of their true nature is ar from complete.* Every lunatic asylum affords abundant proofs that ementia overtakes those who have been long subject to epilepsy. The everity of the individual attacks has less influence in bringing about this esult than the frequency of their occurrence; indeed, Esquirol long ago tated that dementia more often occurs in persons who are liable to *le etit mal* than in those who suffer from *le haut mal.*

Pathology.—As soon as an attempt was made to distinguish the funcions of different parts of the nervous centres, the hypothesis was started hat in a fit there was a torpor of the brain, associated with excitement of he spinal cord. With the advance of physiology the theory arose that the isease might have its seat in some particular part of the brain, the "epieptic centre." Van der Kolk thought that the bulb was the seat of pilepsy; and subsequent writers, including Reynolds and Nothnagel, exressed a similar opinion, but included in the area of disturbance the pons r the cervical part of the cord.

We must, however, either confine the supposed "epileptic centre" within ery narrow limits, or else include in it the entire cerebro-spinal system. Ve cannot imagine that spasmodic movements of the eyes or of the head ear to such a centre any closer relation than do those of the upper or the ower limbs or of the bladder. But if we give up the idea of fixing the eat of the disease in a definite spot in the bulb and pons, we must include n the affected area the cerebral hemispheres also; for consciousness is susended in a fit, and delirium often follows.

Sir William Gull used to speak of epilepsy as a "function;" and by his he meant that the orderly development of the symptoms which make up the seizure must depend upon nervous apparatus, like those involved in the more complicated physiological actions; or, as he said, we all have the mechanism for an epileptic fit in our brains, although in most of us it is never put in action, at least in adult life.

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* In the case of Napoleon see critical remarks by Dr John Ogle in the 'Lancet' for January 30th, 1897. Cæsar was killed soon after epilepsy attacked him.

Hughlings Jackson showed that whereas a "destroying lesion," affecting a particular convolution, is capable of causing local paralysis, a "discharging lesion" of the same part gives rise to convulsions of the same distribution, which may implicate the face, arm, and leg in a definite order. He supposed that in particular convolutions movements are "represented," which involve the action of many different muscles.

In 1873 Dr Ferrier, following Fritsch and Hitzig, performed a series of experiments on the lower animals, with the express object of throwing light on Jackson's theories of epilepsy (v. *supra*, p. 779, *et seq.*), and the result of these and of subsequent researches, both experimental and clinical, has been to confirm the position first taken by Jackson.

No doubt the spasmodic movements produced by a local cortical lesion are very different in character from the slow and orderly actions to which galvanic stimulation of the brain gives rise, as when a cat raised the shoulder and advanced the fore-paw, as if to strike; or a rabbit munched with its lips and jaws. But clonic spasms and even more general convulsive fits may be also produced in dogs, cats, and monkeys by cortical irritation.

Moreover, Brown-Séquard discovered that in guinea-pigs section of the spinal cord, or even of one sciatic nerve, was followed after some weeks by well-marked epileptiform fits, which returned again and again, and could at any time be excited by gently pulling the whisker or the hair of the neck.

Dr Ferrier believes that in the cortex of the brain there are individual centres for each separate muscular action involved in the epileptic convulsion, that they are related to each other in a constant and definite order, and that the attack is due to the discharge of these centres in sequence. From whatever part of the hemisphere the march begins, the order most common is that the centres discharge from before backwards, beginning with those of the head and eyes, going on to those of the arms, and ending with those of the leg. He found that epileptic convulsions can be produced as readily by irritation of sensory areas as of the motor centres.

Dr Hughlings Jackson's present views as developed in the first of the Jacksonian lectures (1899) may perhaps be interpreted as follows. He recognises three planes of anatomical structure and of physiological development, each of which forms a reflex mechanism capable of producing convulsive movements in a more or less orderly sequence. The lowest plane of centres and reflex mechanisms consists of the cord with its prolongation the bulb, and the pons Varolii, and it reaches as high as the centre for the third nerve in the aqueduct. When "nerve-force" accumulates in the neurons of this extensive region, there ensue such local and limited spasms as we see in cases of Laryngismus stridulus, Tetanilla, Athetosis, and some other of the spasmodic affections already referred to (pp. 866—872). The middle plane includes the motor areas of the cortex, with the centres of sight, hearing, and smell. When accumulated tension in these regions leads to a "discharge," the result is an orderly sequence of movements in the way above mentioned, as noticed by Ferrier in experiments on monkeys, but without loss of consciousness; that is to say, we have the clinical features of Jacksonian eclampsia as described in a previous chapter (p. 775). The third and uppermost plane includes the extensive tracts of the hemispheres which do not respond to electric or other stimuli, and cannot be marked as either motor or sensory regions, *i. e.* the cortex of the frontal lobes in front of the ascending (and part of the third) frontal gyri, and a portion of the cortex of the occipital lobes. When a discharge occurs in these regions, the result is not only a convulsive fit, but also loss of consciousness; that is to say, we have what is clinically recognised as true primary idiopathic epilepsy.

Accepting this as a convenient hypothesis, the questions remain, what is the nature of the change in the centres which determines a convulsion? and secondly, what is the cause of the origin and periodical recurrence of these convulsions in true epilepsy? To neither question can a satisfactory reply at present be given.

Some years ago, the view widely prevailed that all but the initial phenomena of an epileptic fit result from vaso-motor disturbance. It was supposed that the cerebral arteries undergo spasmodic contraction, and that the consequent anæmia of the brain causes the patient to fall down insensible.

This hypothesis, however, only shifted the initial lesion of an epileptic fit from one set of neurons to another, from the cerebral cortex to the vasomotor centres in the bulb and elsewhere. The essential and primary change is in the cortex, and the vascular changes are secondary or concomitant, not primary.

It is true that the face turns pale at the commencement of a seizure; and there is ophthalmoscopic evidence that the retina, which derives its blood directly from the internal carotid artery, also becomes anæmic; for the optic disc has been actually seen pale or even white, and its arteries diminished in size. It is to be noted, however, that on the single occasion on which the ophthalmoscope has been used before the clonic convulsions ceased, the disc was at first pink, so as to be undistinguishable from the surrounding choroid; and that only afterwards it became slowly white.

It had long been known that both in animals and in man great loss of blood is followed by convulsions, and in 1870, Kussmaul and Tenner showed that the like result could be brought about by ligature or compression of the four great arteries supplying the encephalon. But their experiments only proved that a deficient supply of blood to the brain may be one cause of epileptic fits; and they themselves pointed out that ligature of the trachea, rendering the arterial blood rapidly venous, had the same effect. In both cases they referred the convulsions to sudden interruption of the nutrition of the brain.

The recurrence of epileptic fits at more or less definite intervals may be conveniently referred to a gradual accumulation of energy in the nervous centres, which is discharged during the attacks. As van der Kolk long ago remarked, this agrees with the fact that after a severe fit an epileptic patient usually remains free longer than usual; but a slight attack is soon afterwards followed by another, perhaps on the ensuing day. Moreover, we have seen that each fit is often preceded by a gradually increasing irritability and restlessness, which disappear or are notably diminished after it has taken place. Often patients become gay, loquacious, and excited for some hours before an attack; while others complain of failure of memory, of torpor, and of physical and mental prostration. Nothnagel mentions the case of a lady, generally a light sleeper, who always knew that she was about to have a paroxysm when she happened to sleep more heavily and longer than usual; nevertheless she would wake up feeling quite well, and would not be attacked until later on in the day.

We are thus brought to regard epilepsy as a paroxysmal neurosis, recur- g at more or less regular intervals, as the result of what may be called a —charge of certain cortical neurons; and to regard the alterations in blood-supply to the brain, which undoubtedly occur during the epileptic attack, as concomitant rather than essential to the development of its symptoms. This view is not inconsistent with the fact that the inhalation of nitrite of amyl is sometimes of service in the treatment of the disease; for when there is an aura, the attack can sometimes be overcome by a decided impression on the part to which the aura is referred; and the inhalation may fairly be supposed to act in a similar way, and to cut short the paroxysm by arresting one of its steps.

We must, then, at present be content to say that epilepsy is "dependent upon an unstable condition of the nerve-tissue in some portion of the nervous system, permitting occasional discharges;" or we may call epilepsy a "nerve-storm;" and it appears more satisfactory to refer the sequence of events in a fit to the gradual extension of some morbid condition from one part of the nervous centres to another, than to ascribe it to modifications in the blood-supply.

Of the epileptic aura, it seems to be clear that it does not really start from the periphery, travel along sensory nerves, and set up the paroxysm when it reaches the encephalon. The aura is itself part of the attack, and due to a change in some region of the brain connected with certain cutaneous nerves; the disturbance is accordingly referred to the peripheral distribution of these nerves.

There is at present no bacillary theory of epilepsy, but Jules Voisin has advocated the view that it depends on poisoning of the blood with products manufactured in the body. This view derives obvious support from the fact of plumbic and uræmic eclampsia. Contradictory statements have been made as to the toxicity of the urine before and after a fit.

Ætiology.—The true causes of epilepsy are at present unknown, as is implied by the qualifying terms idiopathic and functional. So far as we know it, it has no morbid anatomy, nor is it, in most cases, toxic. The following facts as to its antecedents are, however, of practical importance, though it is at present impossible to see their pathological bearing.

In a certain number of cases we can trace a *hereditary predisposition.* Reynolds made inquiries which led him to the conclusion that in 12 per cent. of his epileptic patients the same disease had occurred in other members of their families. In a much larger number of cases other nervous affections could be traced in relatives of the patient. Sometimes it was migraine; sometimes insanity, hysteria, hypochondriasis, or mere "nervousness;" thus in a family one child was epileptic, another insane or idiotic, and a third hysterical. Several independent observers state the proportion in which there is a history of some hereditary nervous disease at about a third of the total cases of epilepsy.

All the earlier statistics bearing on hereditary predisposition are vitiated by the absence of distinction between the true idiopathic disease and secondary eclampsia, due to cerebral tumours, meningitis, or injury, to the puerperal state, to Bright's disease, to plumbism, and other toxic conditions. Even now it is hard to say whether it is better to include alcoholic cases or not.

Again, the selection of other "nervous" diseases to be reckoned as allies of epilepsy, and due like it to a "neurotic diathesis," is arbitrary and often demonstrably misleading. Paralysis, for instance, was formerly put into the list: but there is, of course, no pathological connection between arterial disease, syphilis, tuberculous meningitis or caries, Bright's disease, and sarcoma. Nor, if we keep to "functional" disorders, does there seem to be any real relation between epilepsy and chorea, hysteria, neuralgia, or headache. The diseases which are, as a matter of fact, accompanied by epileptiform attacks, are not these, but some cases of tabes, more cases of insular sclerosis and hydrocephalus, many cases of dementia paralytica, and a certain but small proportion of cases of idiocy, mania, melancholia and insanity generally.

Lastly, there are the ever present, but often forgotten, fallacies of all medical "histories," particularly in the case of such dreaded diseases as phthisis, syphilis, insanity, and epilepsy. The parents of an epileptic child are loth to recognise that it is the manifestation of a family predisposition; and among the majority of epileptics a denial of the disease having existed in other members of the family means little more than ignorance of any facts, positive or negative, on the point. There is, moreover, a constant tendency to "explain" the occurrence of this or any other disease by attributing it to some accidental and extraordinary cause, rather than to acquiesce in it as the result of natural and inevitable sequence of events. A sudden fright, a slight blow on the head, mental excitement, fright, anxiety, or grief, or even such commonplaces as overwork, or exposure to cold, are constantly assigned as causes of the advent of epilepsy months or years afterwards; and in such cases the really potent hereditary disposition, or chronic intemperance, or other vices are willingly forgotten.*

Drunken habits in parents are supposed to predispose to epilepsy in their offspring; but the inclination to intemperance may itself be inherited. There is no reason to suppose that tubercle or rickets are concerned in the causation of epilepsy. Nor does the intermarriage of blood relations tend to cause this disease in the offspring, unless both parents should come of epileptic parents.

As to *sex*, epilepsy appears to affect males and females in about equal proportions, notwithstanding the remark of Celsus, "*sæpius viros quam fœminas occupat*," and the opposite result of some modern statistics. Reynolds makes some sagacious remarks ('System,' vol. ii, p. 295) on the fallacies of statistics in this respect.

The *age* at which epilepsy begins is, in the great majority of cases, between the tenth and the twentieth years. Reynolds found that in 106 out of 172 cases the first fit occurred during this period, and in most it was within the still narrower limits of puberty, from thirteen to seventeen. The more marked the inherited predisposition, the earlier is the average age at which the disease develops itself, and it appears in girls earlier than in boys. Apparently the development and commencing activity of the sexual organs are in some way concerned in the causation of the disease. It has been said that in women the menopause is often marked by the appearance of epilepsy, but Nothnagel denies this. According to Reynolds, the period between twenty-five and thirty-five years of age is one at which there is a comparative immunity from first attacks; but they often occur in persons about forty. Exceptional instances are recorded of epilepsy beginning at an advanced age; one, for example, by Trousseau, at about seventy.

Habits of intemperance, sexual excesses, and particularly the practice of asturbation are generally believed to bring about this dreaded disease. th a first fit and succeeding ones have in some epileptics occurred only hen they were actually intoxicated, and in others only during coitus.

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* Brown-Séquard found that the guinea-pigs in which he artificially set up epilepsy nsmitted it to their offspring as an idiopathic disease.

With regard to masturbation, evidence is inconclusive, although there is no doubt that this degrading vice has an evil effect on both body and mind.[*] That epilepsy may be produced by acute alcoholic poisoning there is no doubt, nor that it is sometimes the result of long-continued intemperance (apart from uræmic convulsions), but it is not certain that the pathology is the same in the two cases.

The *toxic conditions* which lead to epilepsy are, beside acute alcoholic poisoning, syphilis in the early stage before gummata have formed, and plumbism. The last cause is happily not common, and has probably been much less so since its effects have been recognised and to a great degree prevented.

In the case of a woman under the writer's care in 1890, chronic poisoning by lead produced a succession of epileptic attacks which ended in death, and the brain and cord were found to be apparently healthy.

With regard to definite *exciting causes*, there is still greater difficulty in judging of them. Jackson mentions a case in which a convulsive fit at the onset of scarlet fever proved to be the forerunner of habitual epilepsy, but this surely was a mere coincidence. It has been stated that the simulation of the disease by impostors has ended in their becoming really subject to it; and, if this were true, it would be a most important fact.

Mr Tomes relates the case of a farm labourer suffering from epilepsy, whose lower molar teeth were found to be much decayed. Although he had been treated for the fits during six weeks and complained of no pain, they were removed, and were found to be enlarged from exostosis. During the eighteen months that followed he had not a single fit, although for many weeks before the operation he had had two or three daily.

Another case, recorded by Dr Ramskill, is that of a boy who suffered for eighteen months from epileptic fits, and who used to rub his left cheek on account of an indefinite uneasiness, not amounting to pain, which preceded the fits. On examination a decayed molar tooth was found; this was removed, and from that time the boy did not have another fit, although he remained under observation for four months. It must be added, however, that during that period belladonna was administered.

A most extraordinary case is one related by Trousseau, of a young clerk, who for several years had been subject to monthly attacks of epilepsy; remedies had been tried in vain at the Hôtel Dieu, when Dr Foville suggested the extraction of some carious teeth. The suggestion was acted on, and from that day the fits disappeared.

Trousseau also records the case of a man, aged forty, who on several occasions, at very short intervals, was seized with violent epileptic attacks. Dr Monnier found that he had been passing fragments of *tænia*, and gave him large doses of castor oil; a whole tapeworm came away, and from that time the convulsive fits ceased.

An injury to the head may be the starting-point of epilepsy. Nothnagel gives the case of a boy who when eight years old fell from a height of twelve feet upon his head. He was stunned for a quarter of an hour, and ten minutes after recovering consciousness he had a characteristic epileptiform fit. There was a slight scalp wound, which healed in a few days. After six weeks he had a second attack, and from that time they recurred at periods which became shorter until he had them at intervals of from four to twelve days. He was twenty-one years old at the time when Nothnagel wrote, and his intellect and memory were already somewhat impaired. A slight scar remained, but this was not painful nor adherent. Nothnagel seems to have thought that no surgical interference would do any good; and perhaps he was right. But in the 'Lancet' for 1873 two cases were recorded, in each of which a piece of the skull was removed by the trephine on account of epileptic fits following an injury to the head; both of them occurred at Guy's Hospital, the one under Mr Cooper Forster, the other under Mr Bryant, and in both the operation was successful.

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[*] I do not believe in the existence of any unfelt irritation starting from the genital organs of modest young women, and giving rise to epilepsy or any similar disease. And I regard as an abomination the operation of clitoridectomy, which was some years ago practised upon a theory of that kind.—C. H. F.

The former patient had received a blow on the head four months before his admission; it left a slight swelling, from which a little pus exuded when it was incised. His first fit occurred the day before he came into the hospital. But a week later he was having four or five fits every hour, and his temperature was 103°. The skull was then trephined, and the piece of bone which was removed was very dense, three eighths of an inch thick in one place, and rough on the outer surface. A fortnight afterwards he could walk the length of the ward, and he never had another fit—at least until the time when the report of the case ceases, which was two months from the date of the operation.

In Mr Bryant's case the accident had occurred five years before, and attacks of the *petit mal* had occurred at intervals of about a week during the whole period. The cicatrix was still tender, and occasionally painful. Internal treatment having been tried without any good result, the trephine was applied, and a piece of thickened bone removed. The fits at once became less frequent, and seemed to occur only when he was depressed from want of food in his miserable home, or when he was exhausted by diarrhœa. The same medicines which he had before taken with no benefit were resumed, and after a time he became able to earn his living. When the case was reported sixteen months later, he seemed to be permanently cured.

Relation to other convulsive fits.—The late Dr Tyler Smith observed that puerperal convulsions are by no means frequent in women who are habitually subject to epileptic fits. But there may in practice be great difficulty in applying the distinction between true epilepsy and symptomatic eclampsia. So with infantile convulsions. On the one hand, it is said that persons subject to epilepsy in adult life are often found on inquiry to have had fits in early childhood. But, as Jackson remarks, there are at present no facts to show what is the proportion of those who, having had infantile convulsions, afterwards escape epilepsy; it can hardly be doubted that they form the immense majority. The fits of infants were formerly attributed to irritation of the nervous centres from teething or disorder of the alimentary canal; but the more closely such supposed causes are inquired into the less clear does their relation to the convulsive attacks appear to be. The tendency of modern observation is to associate infantile convulsions and laryngismus stridulus with rickets. Whatever part in their causation belongs to external sources of irritation, it is certain that a very important part is due to inherited or acquired conditions of the cortical neurons, disposing them to convulsive discharge. It may well be that in infancy the brain yields to influences which in after life it successfully resists. The higher cerebral centres have not yet acquired inhibitory or restraining influence over the lower excito-motor mechanisms. We cannot affirm that the occurrence of fits in childhood involves danger of epilepsy in adult life: yet it seems clear that no absolute line of distinction between them can be drawn.

Anatomy.—The only morbid changes which are to be found in the brain in epileptic patients must be regarded as effects and not causes of the fits. Perhaps the most important are dilatations of the capillary blood-vessels in the bulb, first described by van der Kolk. He maintained that there was a definite relation between their seat and the symptoms, the nucleus of the hypoglossal nerve and the olive (*corpus olivare*) showing dilated vessels ien the tongue had habitually been bitten; whereas in cases in which the ngue escaped he found them chiefly in the nucleus of the vagus. Similar iditions have been described in cases of hydrophobia, of tetanus, and of orea.

The skull is often exceedingly thick and dense in those who have long suffered from epilepsy, the membranes are opaque, and the cortex is often wasted; but these changes seem to bear a closer relation to the impairment of intellect which is so generally present in cases of long standing than to the primary paroxysmal attacks.

Diagnosis.—When a person is in a fit, which, if really epileptic, would belong to the major form, we have often first to ask whether it is possible that he is *malingering*. In the first place an impostor is likely to choose for his fits a frequented spot, where he is sure to be seen. When he throws himself upon the ground he takes care not to hurt himself; he may even put out his hands to break his fall. He probably overacts his part, crying out many times instead of only once, throwing his limbs violently about, and making the attack last much longer than a genuine paroxysm of epilepsy. There is no pallor of his face at the onset; his skin, instead of being cold, is warm and covered with sweat; his pupils are not dilated, nor insensible to light. He does not keep his eyes wide open, nor are the eyeballs distorted; generally he only separates the lids a little from time to time, so as to watch the bystanders. If an attempt is made to raise the upper eyelid he resists it, and instead of his conjunctiva being insensible, the slightest contact excites forcible contraction of the orbicularis muscle. He will allow the thumbs to be drawn away from the palms, and afterwards close them again. In a simulated fit the tongue is not bitten, and there is no foam about the mouth—unless, indeed, from a piece of soap in the cheek. If a little snuff be blown into a malingerer's nostrils he cannot help sneezing; in a real fit no such effect is produced. Another test is the absence of sensibility to pain: a test used by policemen is to press the thumb-nail beneath that of the person supposed to be in a fit.

An educated medical man would naturally succeed in imitating a fit better than one who has no professional knowledge. Trousseau relates that Esquirol, who did not believe that an attack could be simulated so as to deceive, was once discussing this point with him and with Calmeil in the asylum at Charenton, when the latter fell on the floor in convulsions. Esquirol looked at him for a moment, and then said, "Poor fellow, he is an epileptic!" upon which Calmeil got up and asked him whether he still retained his opinion.

Assuming the attacks to be genuine, one has next to consider whether they are really epileptic or only epileptiform. This question is often settled by the clinical history; for the case may be of many years' standing, and the fits may have gradually developed from the minor into the major form. But one must bear in mind that attacks may recur at intervals for a long time, and present all the characters of idiopathic epilepsy, and yet be symptomatic. Trousseau's case has been already mentioned, of the clerk who had been liable to epileptiform seizures for several years, at monthly intervals, and in whom they ceased when some carious teeth were removed. The same writer relates the case of a lady, aged seventy-one, who for thirty-one years had been subject to attacks recurring with daily increasing frequency, so that she at length had as many as twenty-one in the twenty-four hours. Her forehead and nose presented characteristic signs of former syphilitic disease; and the administration of mercury and iodide of potassium checked the fits.

We must distinguish eclampsia due to Bright's disease, lead-poisoning, chronic alcoholism, and organic lesions of brain by the presence or absence of the other symptoms which severally characterise them, and by the condition of the patient between the attacks (*supra*, p. 775). The relation of epilepsy to infantile convulsions and to puerperal eclampsia is a question rather of pathology than of practical diagnosis.

Hysterical convulsions may simulate those of epilepsy, but in this country it is seldom that we find any difficulty in making the diagnosis even without seeing the patient in an attack. In France Charcot described hysterical convulsions with catalepsy and other complications under the unfortunate name, Hystero-epilepsy. In these fits there is no loss of consciousness, no mydriasis, no biting of the tongue, and no subsequent stupor. The convulsions are far more violent and last much longer than those of true epilepsy. They require a totally different treatment, and one as far as possible removed from experiments by pressing the ovaries, performance before a crowd of spectators, metallo-therapy, hypnotism and such like, which, instead of curing, prolong and aggravate the patient's misery.

Fainting fits is the term often applied to the milder attacks of epilepsy in children. The diagnosis depends on the greater suddenness and shorter duration of the latter, the subsequent drowsiness, the slightest spasm of the eyes or hands, and involuntary micturition. A child who is liable to fainting fits and nocturnal enuresis, should be carefully watched for signs of epilepsy.

Prognosis.—If we separate the "true" idiopathic disease from symptomatic eclampsia due to injury, tumours or other "gross" lesions of the brain, from the epileptiform convulsions of uræmia and of the puerperal state, from hystero-epilepsy and from infantile convulsions—we shall find that epilepsy is no less grave in its prognosis than alarming in its symptoms; and although by treatment we can greatly reduce the number and severity of the attacks, it is only in exceptional cases that we can entirely get rid of them.

If epilepsy begins in childhood there is good hope that it will disappear about puberty. Eclampsia occurring for the first time after fifty is usually symptomatic.

The frequency of the fits is of worse prognosis than their severity. True *petit mal* commonly ends in *grand mal.* Hereditary epilepsy is, as a rule, the most difficult to cure.

When no cause or occasion of the attacks can be discovered, and no improvement takes place under treatment, the natural progress of the disease is towards mental imbecility.

Treatment.—We have to consider the management of the attacks themselves, and that of the intervening periods, with a view to prevent their recurrence.

(1) As concerns the paroxysm, the first point is to ascertain whether the patient has any warning of its approach. If there is a distinct aura, starting first from the hand or foot, he can often be taught, by compressing the limb above, to arrest the further development of the fit. Some years ago there was in the Evelina Hospital a girl who constantly wore round her wrist a piece of cord; this was pulled tight as soon as she felt the sensation which indicated an attack, and not a single one developed itself during several weeks; after a time bromide of potassium was administered, and the aura then ceased to recur.—(C. H. F.) Many instances of a similar kind have been placed on record by different observers. Bazire mentions the case of a woman whose fits were always preceded by spasmodic closure of the left hand; by forcibly extending the fingers, and keeping them open.

an impending attack could be warded off. A patient of Reynolds' had jerking of the left leg, which was drawn up behind him when his attack began; it was arrested by extension of the muscles.

When an epileptic fit is ushered in by no symptoms but pallor of the face and tonic spasms, it would appear that the prompt inhalation of nitrite of amyl may sometimes arrest it. Crichton Browne has related some instances of this. One is that of a man who started up suddenly in bed, with his eyes fixed and his head turned to one side; these symptoms were known to indicate the approach of an attack; the nitrite was administered, and the patient at once fell back on his pillow in a half-fainting state, but without the slightest agitation of the muscles. In another case a fit had actually begun in the ordinary way with rigid stretching of the hands by the side, and turning up of the eyeballs, when the nitrite was held to the mouth and nostrils, and in twenty seconds the paroxysm ceased.

After an epileptic attack has fully developed itself, there is some evidence that it may sometimes be cut short by compression of the carotid artery in the neck. This procedure was first suggested by Dr Parry, of Bath, towards the end of the last century. He relates a case in which it proved successful. A man who had been liable to epilepsy for two years was one day beginning to have a fit; his eyes were assuming a vacant stare, and convulsions were beginning about his throat, when Parry made strong pressure over the right carotid artery; upon this the convulsions ceased, and the attack proceeded no further. He instructed the patient how to compress the vessel, and the latter afterwards assured him that when he had sufficient warning he was often able to prevent the epileptic paroxysm. At Guy's Hospital this practice was adopted by the late Mr Stocker, and sometimes with striking results; but it was most useful in cases with a strong hysterical element. The plan which he used to adopt was to press both thumbs into the neck, one on each side, towards the spine; in doing so he doubtless compressed other structures beside the carotid arteries, and the pain caused may well be supposed to have been concerned in the rapid restoration of the patient to consciousness when the case was hysterical.

When we are called to a patient in an epileptic attack we must see that his clothes are loosened, especially about his neck, and we must take precautions to prevent his injuring himself. We may try to keep the tongue from being bitten by putting a piece of india-rubber between the teeth; but there is the risk of its falling back into the throat and causing suffocation. A thick glove or the sleeve of a coat is safer. Epileptics who are liable to attacks in the night should be careful to remove false teeth from the mouth before going to bed, lest they should become impacted in the pharynx during a paroxysm.

In the *status epilepticus* it would appear that the best remedy is the inhalation of the nitrite of amyl. Crichton Browne has recorded ten cases in which he employed it, and eight of them terminated in recovery. The effects of the remedy were of the most striking character. He adds that he has found no other plan of treatment of nearly the same value in the status epilepticus as inhalation of the nitrite of amyl.

For instance, a man, aged thirty, between May 6th and 10th had from twelve to sixteen epileptic fits a day. On the 11th he was in a most critical condition; he lay on his back, breathing stertorously, with livid purple features, and streaming with perspiration; the pulse was 140, the temperature 103°. It seemed useless to make trial of the nitrite, but as a forlorn hope he was made to inhale five drops every hour. His breathing at once became less laboured, he had only three more fits that day, and on the 12th there were gleams of consciousness while the pulse and temperature fell. On the 14th he could answer questions, and by the 17th the fits ceased, and he passed into his usual state of health.

The withdrawal of a few ounces of blood has sometimes suddenly restored to consciousness patients who were in a state of profound coma. Some years ago a striking instance of this kind occurred at Guy's Hospital in the practice of Sir S. Wilks. Perhaps the undoubted benefit of venesection in apoplectic epilepsy may explain the reputation of bleeding in cases of cerebral hæmorrhage. The present writer has seen good results from the practice ('Medico-Chirurgical Trans.,' 1891, p. 151).

(2) For the prevention of epileptic fits in those who are liable to them—in other words, for the cure of the disease epilepsy—the bromide of potassium surpasses all other drugs in efficiency. Sir Charles Locock, in 1857, was the first to recommend it, and he prescribed it particularly for women in whom the attacks coincided with the menstrual periods. But subsequent observations have shown that there is no such limitation of its curative power.

It is given in doses of from ten to thirty grains three times daily, and it must be continued for months, or even for two or three years. Its effect is sometimes to free the patient from the liability to epileptic attacks. In other cases it suspends them for a time, or diminishes their frequency and severity, but when it is suspended they soon become as bad as before. In yet other cases it does good for a time, but seems to lose its power, although the patient may go on taking it. Lastly, in a few instances, chiefly, according to Gowers, of the minor form, it appears to be altogether useless. No explanation has yet been found for the varying effects of bromide of potassium in different cases.

Most persons can take from ten to twenty grains of bromide of potassium three times daily without suffering any ill effects, and that for many consecutive months or even years. These are the patients in whom epilepsy is not only checked but cured. In some cases, however, full doses, when long continued, and occasionally after less than a month, produce the condition called *Bromism.* It was well described by the late Dr Bazire in his translation of 'Trousseau's Lectures' (vol. i, p. 100). The symptoms are headache, apathy, impairment of the special senses and of common sensation, loss of sexual appetite and vigour, enfeeblement of muscular power in the limbs, tremor of the hands, and impaired action of the heart. A young American lady once came under the writer's care, who, after taking bromides for several years to cure epilepsy, had fallen into a dull, stupid, listless condition, sitting for hours without speaking or moving, and incapable of joining in society. A very definite symptom of bromism is anæsthesia of the velum palati, uvula, and pharynx, which may be tickled without producing deglutition. The conjunctiva is also less sensitive than usual.

Another remarkable effect is the production of a cutaneous eruption. This more or less closely resembles acne; it consists of pustules surroundng hair-follicles, and arranged in patches or groups; they dry up into arge scabs, beneath which the skin becomes red and thickened. A case f this kind is depicted on the forty-third plate of the Sydenham Society's Atlas.' In that instance the scalp and the extensor surfaces of the limbs ere especially affected by the eruption, but the face and the legs are more ften its principal seat. This unpleasant effect of bromides may be preented or mitigated by adding arsenic to the medicine.

One is not infrequently consulted by a patient who has just had a first epileptic attack. The bromide should then be prescribed without delay. If one were to lay the matter in all its bearings before the patient himself, he would certainly wish to take the medicine regularly for a long time, though, in the event of no further attack occurring, it must always remain doubtful whether or not there was necessity for treatment.

If the bromide of potassium fails to check the recurrence of attacks of epilepsy, although given in large doses and with perseverance, we must then have recourse to some other remedy; and perhaps the best is belladonna. This was strongly recommended by Bretonneau and by Trousseau. At first a quarter of a grain of the extract should be given twice or three times a day; and the dose should be gradually increased. At one time a patient of Dr Fagge took two grains three times daily for a great length of time, with marked benefit. Trousseau insists that belladonna should be continued for a long period if its value is to be tested; a year, he says, is sometimes scarcely sufficient for the discovery of its influence; and if in the second year there should be some improvement it may be worth while for the patient to go on taking it for three or even four years. He speaks of it as completely curing the disease in some very rare cases; but Reynolds never knew it do more than diminish the frequency of the seizures.

The salts of zinc are useful in some cases of epilepsy. Wilks has observed more than one case in which the patient was always better when under their influence. Reynolds speaks favourably of the oxide, but has seen no good result from the sulphate. Gowers also prefers the oxide.

The nitrate and the oxide of silver have been recommended, but are probably useless; and one must not forget that, taken for a length of time, this drug will stain the skin of a bluish-black colour. It is believed that these preparations may be administered with safety if the course is not allowed to be continued for more than six weeks. Thirty years ago the writer often saw an American about London who had been castrated and afterwards completely blackened by nitrate of silver in the hope of curing his epilepsy. His skin looked as if it had been polished with blacklead, but the result was negative so far as the disease was concerned.

Another and more recent substitute for Bromides is Borax in full doses. The writer has more than once found it apparently efficacious, and it has no ill effects.

In some cases the introduction of a seton into the nape of the neck has led to the suspension of epileptic fits. A patient in Mary Ward, subject to epileptic fits, had a seton put in her neck; and the fits ceased entirely. After more than a year the seton was removed and they reappeared; it was again inserted, and again the convulsions ceased.

The application of ice to the spine, once strongly recommended, was fully tried by Reynolds, and was found to be useless.

Trephining is a dubious remedy in cases of idiopathic epilepsy, though useful in Jacksonian eclampsia from a cortical lesion or from injury. Numerous cases like the two cited above (p. 925) are on record of the good effects of the operation; but the prospect is far better when the disease is traumatic, and the locality for the operation obvious.

The food of patients suffering under epilepsy should be light and unstimulating, and their meals regular and leisurely. In many cases reducing the quantity of meat has been followed by a marked decline in the number and severity of the fits. The writer knew a lady who, by the late

Dr Radcliffe's advice, lived on vegetarian diet for some years; and the effect in checking the disease was most satisfactory; the attacks became fewer and slighter, the general health improved, and when last heard of the patient had been well for several years. Experiments on the influence of animal food were made at the West Riding Asylum by Dr Merson, who kept a certain number of patients for a month on a diet including much meat, and then for the same period on one in which there was none at all; there was no marked difference in the number of fits, but several of the patients were much more dull and languid when taking animal food than when restricted to farinaceous diet.

Both the mind and the body of epileptic patients should be kept in exercise, short of fatigue. The limbs should never be allowed to be cold at night. It is said that nocturnal seizures have been prevented by a prop being put under the upper half of the mattress, so as to keep the head and shoulders well raised.

In cases of confirmed epilepsy, the patients do much better when removed from home, or hospital, or asylum, to farms or colonies. These have long been established in Holland, and have now been opened in this country. One for men is at Chalfont, in Surrey, and another for women at Godalming. A large colony has been opened at Chelford, in Cheshire, and the London County Council intends to establish one on a farm in Surrey.

PAROXYSMAL VERTIGO.[*]—Vertigo, or giddiness, is observed in many diseases. It is a frequent symptom of cerebral tumours, particularly those of the cerebellum. It is common in sea-sickness, in megrim, and in heart disease. It is a physiological result of a blow on the head, of the motion of a vessel on the sea, or of a swing, and also of cerebral anæmia, whether from loss of blood, or chlorosis, or starvation. It occurs, as we have seen, in both the severe and the slighter form of epilepsy. Lastly, it is often associated with defects of vision and paroxysmal deafness, and may in this clinical connection be considered here.

In some cases of visual vertigo the patient feels as though he were made to turn round and round, or were against his will impelled forwards, or backwards, or to one side; in others he fancies that objects are revolving round him. He remains conscious, and remembers all that occurs. If he has had previous attacks, he may be well aware that his sensations are illusive; yet by the strongest effort of his will he is incapable of overcoming their effect. In a first attack he may be completely deceived. A patient, who happened to be travelling by railway, beheld one side of the carriage suddenly rise four or five feet, and throw him into the opposite corner. He naturally supposed that there was a serious accident; but in reality he had not moved from his seat. A patient of the late Dr Ramskill while walking in the street, suddenly felt the pavement uneven, with alternate depressions and elevations over which he seemed to be obliged to lift his feet; at the same time the shop windows appeared to be moving forwards, and the passers-by to be racing past. But he also felt giddy in himself; and the two forms of vertigo above described are not distinct affections. In most cases both are experienced.

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[*] Vertigo (*vertere*, to turn) answers to the English words giddiness or swimming of the head.—*Fr.* Vertige.—*Germ.* Schwindel. The term "paroxysmal vertigo" is better than "epileptic vertigo," for the latter has been used as a synonym for the less severe form of epilepsy—the *petit mal*.

The gait is unsteady or reeling; the patient feels afraid of running against other people or surrounding objects; he catches hold of any support; or he may lose his balance and fall to the ground. Sometimes closing the eyes removes the sensation of vertigo for the time. Nausea often accompanies the attacks, or even vomiting, as in sea-sickness, megrim, and concussion of the brain.

Visual vertigo is the result of a sudden change of accommodation, or of looking at rapidly turning objects, and probably depends on disturbance of the sense of equilibrium, so far as, in a vertical plane, it is derived from the ocular muscles, and in the third dimension of space from the ciliary muscle.

Auditory vertigo.—Paroxysmal vertigo is often connected with deafness on one or both sides, and sensations of buzzing or singing in the ears. This connection of deafness and giddiness has been long known, and was recognised in his own case by Dean Swift; so that neither the fact nor the name of auditory vertigo was new, when, in 1861, Ménière recorded in the 'Gazette Médicale' some remarkable instances of this kind, and offered an anatomical explanation of the symptoms. Such cases have since received the name of *labyrinthine vertigo* or Ménière's disease; but the prevalent interpretation of their pathology is far from proved.

In the first place, it is certain that affections of the middle, and even of the external ear may give rise to attacks of giddiness, faintness, and sickness. For example, in the 'Archives of Ophthalmology and Otology' for 1871, both Knapp, of New York, and Brunner, of Zürich, mention cases of aural catarrh in which such symptoms appeared; and Toynbee many years ago asserted that cerumen accumulated in the external meatus might by its pressure on the membrana tympani produce similar effects.

In the great majority of cases of auditory vertigo, however, the more accessible parts of the organ of hearing are free from disease. If the deafness is of one ear, a tuning-fork is not heard on that side, even when placed upon the teeth or upon the top of the head. It is hence inferred that the seat of mischief must be the internal ear.

Here some interesting physiological observations appear to find their application. Many years ago Flourens discovered that in pigeons and rabbits section of the semicircular canals causes strange disturbances of equilibrium. More recently Mach, of Vienna, and Crum Brown, of Edinburgh, showed good evidence that the function of these structures is to furnish the impressions which form the principal basis of our knowledge as to the relation between our movements and those of surrounding objects. They also showed what are the several disturbances of equilibrium which irritation or destruction of each canal may be expected to produce. Charcot observed a case of paroxysmal vertigo in which the lesion was chiefly in the left ear, and in which the direction of reeling was principally forwards, but sometimes backwards, while occasionally there was a sense of rotation on a vertical axis, always from left to right. This last would, presumably, be due to irritation of the left horizontal ampulla, while movements forwards and backwards would answer to irritation of the posterior and superior canals respectively. Destruction of the same parts would produce precisely the converse effects; and thus there is no difficulty in accounting for the fact that some patients have shown a tendency to reel towards the side on which they were deaf. In either case the actual movements are supposed to be the reflex results of the impressions re-veyed from the several canals, impressions which under normal conditions balance one another, but do so no longer when one of the ampullæ is diseased or injured.

Even when morbid changes in the meatus or tympanum are obviously present, it may be supposed that the direct cause of vertigo is disorder of the labyrinth; for pressure upon the fenestra ovalis may easily cause increased tension in the semicircular canals. Thus all cases of "auditory vertigo" might come to be included under labyrinthine vertigo.

It is due to Ménière to say that he did not pretend to draw attention to the fact that vertigo is apt to occur in those who suffer from deafness or from some disease of the internal ear; for this was well known before. What was really new in his paper was that he endeavoured to show that sudden apoplectiform symptoms (including transient loss of consciousness) might occur in a person previously healthy, might be followed by deafness, and that the cause of such attacks might be an affection of the internal ear. He relates several cases of patients who fell down insensible, and who, when they recovered, were found to be deaf; and a similar instance has been since recorded by Knapp.

The only one of Ménière's cases in which a *post-mortem* examination was made is the tenth and last of his series. A young woman, while menstruating, undertook a night journey outside a coach. She suddenly became completely deaf, and was admitted into Chomel's wards. The principal symptoms were constant vertigo and vomiting. She died on the fifth day. At the autopsy no disease could be discovered in the nervous centres; but the semicircular canals in each ear contained a reddish plastic substance.

"Surely," remarks Dr Fagge, "this observation is very inconclusive. Cases in which an autopsy fails to reveal a satisfactory explanation for cerebral symptoms present during life are, after all, not very rare; and it seems rash to assume that the state of the labyrinths was the real cause of the fatal illness in Chomel's patient. Moreover, as Brunner points out, even if full value were allowed to the case in question, one could hardly take it as demonstrating the nature of those other cases in which cerebral symptoms come on suddenly and rapidly pass off. In these it has been supposed by Knapp that hæmorrhage takes place into the semicircular canals. But since the deafness is often simultaneous in the two ears, the blood must be effused on both sides at or about the same time, and this makes the explanation very improbable. It is true that hæmorrhage into both retinæ occurs in cases of Bright's disease; but surely not so as to cause sudden and total blindness."*

Wilks inclines to put the seat of auditory vertigo not in the semicircular canals, nor in the branch of the portio mollis, which supplies them, but in the brain itself. This view applies well to cases of Ménière's disease in the stricter sense of that term; the sudden loss of hearing may be attributed to an affection of the auditory centres in the temporo-sphenoidal lobes, and the giddiness to the probably adjacent centre for equilibrium. Whatever peculiarities in the direction of the vertigo have been supposed to depend upon affections of particular ampullæ, can be referred to corresponding changes in the centre, for here each canal must be represented.†

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* The writer once saw retinal hæmorrhage in the course of chronic Bright's disease cause sudden and total blindness; but in that patient the other retina had been long affected without his knowledge.

† The analogy of the other paroxysmal neuroses strongly supports Wilks's view. Impairment of sight is a frequent symptom of migraine, and it is certainly due to

A strong argument in favour of Wilks's view is afforded by the fact that bromide of potassium may remove both the giddiness and the deafness at the same time; and the late Mr Hinton recorded in 1873 under the name of Ménière's disease a case in which paroxysmal vertigo and sickness had been associated with only transient deafness, and in which all these three symptoms were brought back together by the patient being ordered quinine.

Knapp has observed that in certain cases the impairment of hearing is particularly marked for certain musical tones, those of the middle octaves being distinctly perceived, while those of the lower, and still more those of the higher octaves are heard very imperfectly. He regards this as a proof that the seat of the affection is in the labyrinth; but the force of the argument is not obvious. More important is an observation of Charcot's, that some patients experience sensations of vertigo and buzzing in the ears only so long as the deafness is partial, and lose these symptoms as soon as it becomes complete. This, however, is not always the case, for a patient of the writer's who was permanently deaf with one ear, became the subject of vertigo as the other gradually became deaf also; until, with paralysis of both cochlear and utricular branches of the portio mollis, the deafness was complete, and the tinnitus and vertigo constant. Even if we admit that in some of these cases the labyrinth in the internal ear is really the part primarily affected, the same thing need not be true of the "apoplectiform" cases. Epilepsy and migraine may be excited by a variety of causes, and why not paroxysmal vertigo?

Hinton published nine cases of vertigo, mostly paroxysmal, and associated with deafness, tinnitus, and nausea or vomiting, in the 'Guy's Hospital Reports' for 1873 (vol. xviii, p. 193). In some of these the hearing was affected on both sides, and in two (Cases 3 and 9) the perception of certain musical sounds was definitely impaired.

Dyspeptic vertigo.—We may describe this "gastric" or "bilious" vertigo as distinct from other varieties of giddiness; but it does not appear to differ, except in origin, from that which depends on deafness. Wilks remarked that the vertigo caused by bilious derangement occurs chiefly when the patient stoops or lays his head upon the pillow, and ceases when he stands upright; but this distinction is not constant.

A classical instance of vertigo, nausea, deafness and pains in the head, coming on as a paroxysmal disease early in life and persisting until old age and senile imbecility came on, is that of Dean Swift. Dr Wickham Legg and Dr Bucknill independently argued in favour of its being a typical case of Ménière's disease; but we have, of course, no knowledge of the condition of the labyrinth. Swift's account of the origin of his attacks in a fit of indigestion while he was living with Sir William Temple would point to *vertigo a stomacho læso.*[*]

In some cases the ingestion of food which disagrees with the patient leads so quickly to swimming in the head that the connection cannot be

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affection of the brain and not of the eyes. Indeed, "cloudiness before the eyes" and "obscuration of the visual field" are mentioned as having been present with the vertigo in some of Ménière's and Knapp's cases; and it may be that in these instances the attacks presented a combination of the two neuroses, the nerve-storm spreading beyond its usual limits and encroaching upon the area concerned in migraine. So also in the "apoplectiform" cases described by Ménière, we may account for the loss of consciousness by supposing that the disturbance spread over the hemispheres, as in epilepsy.—C. H. F.

[*] "I was seized with so cruel a fit of that giddiness" ("and weakness and sickness in my stomach") "which at times has pursued me from my youth, that I was forced to lie down on a bed for two hours" (Dec. 17, 1734).

overlooked. Murchison mentions a medical friend of his, a sufferer from gout, and who, whenever he drank a cup of tea or a glass of champagne, was seized with sudden giddiness; his head felt empty, neighbouring objects seemed to whirl about him, and he had to lay hold of something to prevent falling. After a few seconds or minutes the attack passed off, but in many patients the vertigo often lasts longer.

Dr Ramskill recorded the case of a merchant who was one day quietly walking in the city from one office to another, when he was seized with giddiness, so that he reeled, and had to lay hold of a post which was near at hand. In a few hours, after a purge, he became better, but he felt weak and shaken, and complained of a heavy diffused headache. About three hours before the attack he had eaten hastily, and with imperfect mastication, a breakfast of which sausages and Devonshire cream formed a part; and to this the vertigo was ascribed, no doubt with justice. Yet, during the following month, the same patient had five similar attacks, not one of which could be traced to any such cause, he having in the meantime become very particular as to his diet. There were no positive signs of gastric disorder: the proof was the success of treatment directed to the digestive organs.

A second medical friend of Dr Murchison's, who had never had gout, was seized with dimness of sight every night while writing. He took iron and quinine without any benefit. He was advised to give up practice for a time, and try the effect of a change of air; but while he was making up his mind to so serious a step, he took a few grains of blue pill, and his symptoms at once disappeared.

Other forms.—Trousseau quotes from Boerhaave the case of a man who, during two years, was seized with symptoms of vertigo whenever he attempted to stand up. In vain had the ablest practitioners attempted to cure him. He had a first attack of gout, and from that time the giddiness ceased.

Vertigo may replace other paroxysmal neuroses. Giddiness is occasionally present during the paroxysms of migraine; and Dr Liveing refers to two cases, in each of which an attack of intense vertigo, of short duration, several times replaced the ordinary sick headache.

Idiopathic or "essential" vertigo appears sometimes to be transmitted by inheritance. One of Dr Ramskill's patients suffering from vertigo had a father, aged seventy-one, who had been subject to it for thirty-five years; and who also was subject to asthma. Another patient suffered for three years from hereditary vertigo, for which no other cause could be discovered, and which resisted all treatment. It is true that in that case the giddiness after a time became almost continuous; and persistent vertigo is most often due to obstructive disease of the cerebral arteries. Nevertheless a case is recorded by Charcot ('Progrès Méd.,' ii) of a woman, aged fifty-one, who had for six years suffered from continuous vertigo. It did not intermit even at night, and was so severe that she could neither walk nor stand, for the slightest movement of her head made her clutch at surrounding objects for support. The complaint had lasted twenty-six years; but for a long time it only occurred in paroxysms. She had disease of the tympanum on each side.

Treatment.—Bromide of potassium is of more service than any other medicine in treating vertigo, and its value is sometimes as striking as in epilepsy itself. Many aural surgeons use chloride of ammonium for those cases of vertigo associated with deafness. In April, 1890, the writer had a marked case of paroxysmal vertigo in Philip Ward, with severe headache, tinnitus, and temporary deafness with vomiting, but with no evidence of organic disease of the brain or the ear. The patient, a man about fifty, had been subject to the attacks for several months, and recovered rapidly under moderate doses of bromides.

Inquiry must be made for symptoms of dyspepsia or of gout. A good plan is to give a few grains of bicarbonate of soda or bismuth, with aromatic spirits of ammonia in chloroform water. Blue pill, with or without colchicum, sometimes works marvels. Wine is valuable in senile vertigo. One of Charcot's patients was cured in two months by fifteen grains of quinine daily; but in a very characteristic case of auditory vertigo under the writer's care the patient had, by Professor Charcot's advice, taken quinine in full and repeated doses without the least benefit.

Paroxysmal Mania.*—There is a paroxysmal neurosis in which the attacks take the form of transitory mania. A good example was recorded by Dr Maclaren in the 'Medical Times and Gazette' for 1876.

The patient was a slightly built lady, aged forty-three, with winning manners and a soft quiet voice. She was characterised by exalted religious feelings and morbid sensitiveness. She would be reading her Bible and talking gently to her attendant, when suddenly, without a moment's warning, she would throw the book out of the window, and make a rush to run her head into the fire; or she would, perhaps, turn on the attendant and try to strangle her. She would then struggle on, keeping all the time perfectly silent, or uttering only an occasional word of Scripture, until she was exhausted, or until by a kind of awakening she became restored to her former condition. Sometimes in the attacks she would expose her person. During the intervals she had little or no recollection of what she had done.

One peculiarity of the paroxysms in this case was that in each of them the patient made efforts to get at one particular picture, which at other times excited no emotion. Thus it seemed that she followed out in successive attacks trains of thought of which she had no knowledge during the intervals; and her condition might so far be termed one of "dual consciousness."

Trousseau gives the following instances:

A gentleman was attending a literary meeting at the Hôtel de Ville, when he ran out and walked for some minutes on the quays, avoiding with success both the carriages and the passers by. When he recovered he found that he had neither greatcoat nor hat; he returned to the room and resumed, with a perfectly lucid mind, the historical discussion in which he had before taken an active part.

The same patient, a magistrate, was presiding at a provincial tribunal, when he suddenly got up, muttered a few unintelligible words, and went into another room. He was followed by the usher, who saw him make water in one corner, after which he returned to his seat.

Another patient of Trousseau's, an architect, used often to have an attack while walking across a narrow plank at a height from the ground. He never met with any accident, although he would run rapidly over the scaffolding, shrieking out his own name in a loud abrupt voice. A moment afterwards he would resume his occupation and give orders to his workmen, without any recollection of what had occurred to him.

Most patients subject to paroxysmal mania have before suffered from the *petit mal*, if not the *haut mal*. Hughlings Jackson, indeed, believes that a transitory epileptic paroxysm really occurs each time before the mental symptoms develop themselves. In other words, he thinks that the affection is identical with that form of mania which sometimes follows an epileptic fit (p. 918). This view supposes that the necessary condition for the occurrence of the mental disorder is the removal of the control of the highest centres, which are exhausted by the discharge during the fit.

Even when paroxysmal mania occurs in those who are subject to frequent epileptic fits, one may be unable to ascertain the fact at the time. It often happens that a patient in this condition is brought to a hospital by the police, and it may be impossible for one to say whether he is an epileptic, or drunk, or suffering from meningeal hæmorrhage.

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* *Synonyms.* — Epileptiform mania — Paroxysmal insanity — Furor epilepticus.

Jackson records an interesting case of a woman who was brought to the London Hospital in a maniacal state, with a deep gash in the left arm, by which the elbow-joint was opened. She accused different people of having inflicted this wound upon her, but it was ascertained beyond doubt that she had done it herself. She had been cutting bread for her children's tea when she suddenly sent them all out of the room. A short time afterwards she was found lying in a pool of blood on the floor. On the following day she was rational, but furious mania returned several times during the next week. On inquiry it was ascertained that she had been subject to epilepsy, in both the minor and graver form.

A patient suffering from this form of disease is liable to find himself in the hands of the police for some offence committed during the paroxysm: and although to a skilled medical observer it is evident that he is irresponsible, there will be difficulty in making this clear to others. Jackson has done much towards the elucidation of such cases by studying other instances in which the acts performed are not criminal but absurd; they may be marked by precisely the same adaptation of means to ends, and yet they leave no trace on the memory, and were performed without the actor's intention or even knowledge.

Thus one of his patients had been talking about supper, and it had been agreed that he and his wife should have some cold fowl, and her sister some cocoa. Soon afterwards he felt the symptoms of an attack and sat down on a chair against the wall of the kitchen, where he happened to be. He remembered nothing further, but his sister-in-law came in and found him standing by the table mixing cocoa in a dirty gallipot, which was half filled with bread and milk for the cat, and stirring the mixture with a mustard-spoon, which he could not have obtained except by going to the cupboard for the purpose. If the object fetched had been a knife, and if he had inflicted some injury with it, this purposive action would have seemed a strong point against him.

Jackson himself admits that the form taken by a man's mental automatism during the paroxysm depends very much on his previous character. A savage and suspicious man would, when his higher faculties were temporarily in abeyance, be more likely to kill some one than to mix cocoa for his sister-in-law. Indeed, the actions performed during a state of unconsciousness are sometimes exactly those which would have been performed if the patient had been in full possession of his faculties. The man last referred to had on another occasion ordered dinner at an eating-house, when his mental condition underwent a change, and he remembered nothing more until he found himself at his desk in the office, feeling rather confused. He had to go to the place and ask whether he had had his dinner, and he then found that he had eaten it and paid for it, and that neither landlady nor waiter had noticed any peculiarity about him.

In other cases the patient goes through ordinary actions in an absurd way. Thus another of Dr Jackson's patients, while in an omnibus, was one day observed to blow his nose upon a piece of paper, and when he got out he gave the conductor £2 10s. instead of the usual coppers. A third patient one day found the extinguisher of a candle in his waistcoat pocket. For some years he invariably looked at his watch after each fit, and he must, in an attack, have mistaken the extinguisher for his watch.

THERMIC AND TOXIC NEUROSES

SUNSTROKE, AND THE EFFECTS OF DRINK, OPIUM, ETC.

> "And when the child was grown, it fell on a day that he went out to his father to the reapers. And he said unto his father, 'My head, my head!' And he said to a lad, 'Carry him to his mother.' And when he had taken him, and brought him to his mother, he sat on her knees till noon, and then died."—2 *Kings* iv.

> "O that men should put an enemy into their mouths to steal away their brains!"
> *Othello.*

> "I paused seasonably: but with a difficulty that is past all description. Nothing short of mortal anguish in a physical sense it seemed, to wean myself from opium; yet, on the other hand, death through overwhelming nervous terrors, death by brain fever or by lunacy, seemed too certainly to besiege the alternative course."—DE QUINCEY.

HEAT-STROKE.—*Origin and pathology—Symptoms of the cardiac and cerebro-spinal forms—Prognosis—Sequelæ—Pathology—Diagnosis—Treatment.*

ALCOHOLIC NEUROSES.—*Delirium tremens—its symptoms and course—prognosis and treatment—Chronic alcoholic poisoning—its symptoms and course—antecedents—diagnosis and treatment.*

NARCOTIC NEUROSES.—*Opium-eating—The morphia habit—Symptoms and results—Treatment—Neuroses due to other narcotic drugs.*

Neuroses from lead poisoning—from Arsenic—from Mercury—from Zinc.

BEFORE concluding the long series of functional disorders of the nervous system, we must find room for some which are the direct result of external disturbing causes, namely, heat and poisons.

HEAT-STROKE.*—That exposure to intense heat is sometimes followed by alarming or fatal cerebral symptoms is well known. The affection is not uncommon in the hotter parts of India and China, and is also frequent during the hot season in New York and in Australia; but heat-stroke is very rare even during the hottest summers in England, and is far from common in the South of Europe or in South Africa.

The common term "Sunstroke" is inaccurate, for the direct rays of the sun are not required to produce it; it often comes on at night if the temperature is very high, particularly when a number of persons are crowded together, as among soldiers in barracks, or sailors or emigrants on board ship. Sir Joseph Fayrer has seen it at Aden among the stokers in the engine-rooms of the Red Sea steamers. *Heat-stroke* is therefore a better name.

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* *Synonyms.*—Solis ictus—Insolatio—Siriasis—Sunstroke—Heat-apoplexy—Thermic fever.—*Fr.* Coup de soleil—La calenture (in part).—*Germ.* Hitzschlag, Sonnenstich.

As might be expected, this disease often attacks several men simultaneously or in rapid succession. Maclean witnessed many soldiers of the 98th Regiment struck down, of whom about fifteen died on the spot, in taking possession of a steep hill in China in 1842.

But the men who suffer from heat-stroke appear to be never more than a minority of those who are exposed at the same time to the sun's rays. This depends upon the fact that it is not the degree of heat of the air but that of the tissues which determines the symptoms. So long as heat is given off freely from the skin by radiation, conduction, and evaporation, and from the lungs by evaporation, the body-temperature does not rise above normal; and Dr Fordyce proved in his well-known paper before the Royal Society that men can exist without danger in a room so hot as to cook a mutton chop. Maclean remarks that the closely shaven heads of the Chinese bear the hottest sun without ill effects, although it is true that they generally use their fans to keep up a free current of air about their faces. Sportsmen in India expose themselves to very high temperatures with comparative impunity so long as they take care to wear light clothes, to protect the head and neck, and to abstain from stimulants. Thus it is of the highest importance that soldiers and others who have to bear fatigue in hot climates should be suitably dressed, and should have no accoutrements which can interfere with the play of the lungs.

More than one observer has noticed that fat and heavy men are most apt to suffer from sunstroke. Apart from crowding and defective ventilation, exhaustion from prolonged exertion is a predisposing cause. An attack seems more likely to occur when the air is loaded with moisture, since this must interfere with evaporation from the skin; but there was extreme dryness at the time of an outbreak which Surgeon-General Longmore saw at Barrackpore. Europeans are supposed to be more likely to suffer from the disease when they have only been a short time in India, but this is only because they are unaware of the necessary precautions or despise the advice of others. There is good evidence that natives are by no means free from heat-strokes, and the same is true of the negroes in America.

Symptoms.—The symptoms of heat-stroke vary in different cases.

There is a "cardiac" form, in which the man falls down suddenly insensible, and after a few hurried gasping respirations, death by syncope takes place. Dr Morehead also describes milder cases, in which a sense of prostration and faintness is experienced, with vertigo, dimness of vision, dilated pupils, and drowsiness. The patient can be roused by speaking to him, pinching him, or sprinkling his face with cold water. There is constriction of the chest, with sighing respiration, a sense of weight or sinking at the epigastrium, nausea, and sometimes vomiting. The face and lips are pale; the skin generally cold and clammy; the pulse feeble, and generally slow. Under judicious management such cases often recover; but sometimes the pulse sinks, the breathing becomes more sighing and irregular, and death follows, with or without convulsions. "Cardiac" cases seem to be met with only among those who are attacked in consequence of direct exposure to the sun's rays. When recovery takes place, it is complete and leaves no sequelæ.

There is a "cerebro-spinal" form, of which coma is the principal feature. This often comes on gradually. It may be preceded by nausea and loathing of food, giddiness, congestion of the eyes, extreme debility, and (as more than one observer has noticed) an unusual frequency of micturition. "I cannot hold my water" is said to have been in a large number of cases the first thing complained of. Maclean speaks of the attack beginning with a delirious shout of laughter, or an attempt to escape from an imaginary danger; headache, he says, is seldom present. When heat-stroke attacks a man sleeping in barracks, the first thing his comrades notice is his stertorous breathing; he is then already insensible, with contracted pupils, deeply congested conjunctivæ, and a quick sharp pulse. As far back as 1860 Dr Morehead noticed that the skin was pungently hot to the close of these cases, and for some time after death. It is now known that the condition is really one of hyperpyrexia; and in consequence the name of "thermic fever" has been proposed for this condition. Three instances of this form of the disease occurred in 1876 at Bristol, and were fully recorded in the 'Lancet' by Dr Shingleton Smith and Dr E. L. Fox. They seem to have presented the symptoms of "thermic fever," as it is described by those who are familiar with sunstroke in India.

One of them was in a man, aged forty-five, who had been turning a winch on board a steamship, exposed to the full glare of the sun, from 8 a.m. The temperature in the shade was from 92·3° to 96°. At one o'clock he felt unwell, and began to talk incoherently and to throw his arms and legs about. He had been drinking water freely. He was admitted into the hospital half an hour later, and he was then comatose, with pin-point pupils; the temperature was then 107°; the pulse 160, weak and intermitting; the breathing laboured; the face not flushed; the skin sweating. At twelve minutes past two the thermometer registered 109°. Under energetic treatment the temperature gradually fell to 100·4°; the pupils became normal and afterwards dilated; but the pulse failed more and more, until it could not be counted, and at 7.40 he died.

On the same day, another man, aged thirty-five, was taken into the same hospital in a state of only partial consciousness, with general muscular tremor, soon followed by convulsions and opisthotonos. His temperature was then 110·2°; his pulse was too rapid and too feeble to be counted. He died twenty minutes later, the thermometer at that time registering 111°.

Two days afterwards a third case was admitted, at 2.30 p.m., in the person of a man, aged fifty-five, who had been driving a hearse, when he fell backwards, and the reins dropped from his hands. He was comatose; there was tonic spasm of the muscles of the legs; the pupils were contracted, but sensible to light. The temperature at 2.40 was 106·4°; at 2.50 it was 107°. The pulse was 141; the breathing was stertorous and at the rate of 27 in the minute. Under treatment the temperature quickly fell; at 3.25 it was 102°, at 3.40 it was 100°. In this instance recovery took place.

Dr Morehead speaks of a "mixed form" of heat-stroke, in which the symptoms are a variable combination of those of the other two forms, while admitting that these are only varieties of the same disease.

Sir Joseph Fayrer distinguishes syncopal, asphyxial, and hyperpyrexial varieties of heat-stroke as observable in India.

It is well known that *sequelæ* of a very serious kind are not infrequent. Those mentioned by Maclean are persistent headache, a chorea-like affection of the muscles, generally those of the forearms and hands; epilepsy, particularly in those who have inherited a tendency to that disease or have had fits in youth; and mental weakness.

Fatal event.—When heat-stroke ends in the patient's death, this generally occurs within nine hours, but sometimes not until towards the end of the second day. The average mortality of the disease is estimated at from 45 to 50 per cent. Fatal relapses, after recovery from incomplete coma, are not infrequent; so that cases which seem to be doing well require to be carefully watched with the thermometer until the skin becomes moist and cool.

Anatomy.—The usual *post-mortem* appearances are not distinctive. We find reports of congestion of the viscera, and the blood is said always to remain fluid. In twenty-five cases of death, out of forty-eight of heat-stroke, among the troops at Assouan in 1886, Surgeon Douglas Hunter remarked the following conditions after death:—intense lividity of the surface and ecchymosis of the conjunctivæ; venous engorgement; muscles of dark colour. In only one case was meningitis observed; and then there was an interval between the exposure and the onset of symptoms.

In 1856 there was in Guy's Hospital a sailor who four years previously, when crossing the equator, had been attacked by sunstroke in company with another man who died. He himself came to in a few hours, but he could not speak for a month afterwards, and for a time he lost the use of his right arm and leg. From all these symptoms, however, he had perfectly recovered; and he was now suffering from renal dropsy, which at length proved fatal. At the autopsy Dr Wilks found the arachnoid opaque and marked with white spots, the ependyma granular, and an excess of fluid in the brain. It is to be noted, however, that the vessels at the base were much diseased, so that the morbid appearances may have been unconnected with heat-stroke.

Pathology.—Dr Norman Chevers and some other Indian physicians have attempted to ally sunstroke to malignant malarial fever, while others have seen some relation between it and cholera; but the opinion of Sir Joseph Fayrer, and of most of those familiar with the disease, is that it is a physiological result of overheating the body. More recently an article has appeared by Dr Sambon ('Brit. Med. Journ.,' March 19th, 1898, p. 744) advocating the recognition of sunstroke or, to use an old name, *siriasis* (star-stroke) as a specific infective fever. There is no attempt, however, in this paper to demonstrate contagion, still less to offer experimental evidence of it, or of the presence of any recognisable contagium.

Diagnosis.—Heat-stroke is no doubt easy to recognise in most cases; but this often is because the patient is known to have been exposed to a high temperature, rather than because the symptoms are in themselves distinctive. Maclean places reliance on certain characters of the pulse and respiration, and on the state of the skin and of the pupils as serving to exclude apoplexy from consideration. But, as Dr Fagge remarked, if hyperpyrexia is not present from the commencement, a person attacked on a hot day in India with cerebral hæmorrhage, or embolism, would be exceedingly likely to have his case set down as one of "sunstroke," even by good observers, especially if there were no paralysis.

Mild cases.—Apart, however, from the classical coup de soleil or thermic fever of India and the tropics generally, there is a very much milder malady which is probably due to exposure to the sun, and is most often seen in children in England. A boy, after playing cricket in the sun, or a child after paddling in the sea on a hot day, will come home complaining of feeling sick and faint; he may perhaps vomit, looks very pale, and has a bad headache. Cool drinks and rest in a dark room generally set him right in a few hours.

It often happens that patients tell one that their complaints all result from a "sunstroke" which they had in England during the previous summer; but such statements must be received with caution. Even a history of sunstroke in the tropics is not always trustworthy. The writer has found it conceal sometimes a first epileptic attack, and sometimes cerebral syphilis.

Treatment.—The most important remedy is the bold and immediate employment of cold. Ice should be applied to the head; a stream of iced water may be passed through coils of elastic or leaden tubing (as used by Leiter for the purpose) in contact with the back and chest; with or without ice, the patient should be got into the air, and cold water should be poured over his head and chest. If the patient can swallow he should be allowed to drink water freely. In the one successful case at Bristol (which, however, was less severe than the others) Dr Fox, besides applying cold, injected a grain of quinine under the skin in five different places; and at the end of half an hour the patient took ten grains of quinine by the mouth. Indian practitioners are agreed that venesection is injurious; but Dr Hunter still recommends the old practice of a large enema and a dose of calomel.

If syncope is threatened, brandy or liquor ammoniæ (♏viij to ℥iss of water) or strychnia must be injected subcutaneously. Maclean says that the application of a blister to the nape of the neck, or to the shaven head, may be of service. Artificial respiration should be resorted to in the asphyxial form of sunstroke. If convulsions set in, the inhalation of chloroform is recommended.

For *sequelæ* of heat-stroke, occurring in India, removal to a temperate climate appears to be the best treatment. Maclean states that at Netley there are always some cases of this kind, and that they are often very obstinate. He has seen long-continued counter-irritation to the nape of the neck, and a course of iodide of potassium, permanently relieve some patients troubled with severe fixed pain in the head; but in other cases the same measures have altogether failed. He gives a favourable prognosis for those who come home with epilepsy after sunstroke.

ALCOHOLIC NEUROSES.—Intemperance in drink may affect the nervous system in two ways—acutely, as a form of delirium following a heavy bout of drinking, and in a more insidious but scarcely less dangerous manner as the result of long-continued dram-drinking, possibly without there ever having been what could be called drunkenness.

Acute alcoholic poisoning.[*]—When the nervous symptoms of alcoholism take an acute form and endanger life the condition has for many years been known under the name of "delirium tremens." The earliest account of it appears to have been published in 1813 by Dr Thomas Sutton, of Greenwich; and the first writer to describe its course, undisturbed by the administration of medicine, was an American physician, Dr John Ware, of Boston, in 1831; his work, based on the observation of nearly a hundred cases, was of great original worth. Sutton, however, mentions that Dr William Saunders, a former lecturer on medicine at Guy's Hospital, had forty years before (*i. e.* in 1773) described it as distinct from phrenitis[†]

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[*] *Synonyms.*—Delirium tremens—Delirium potatorum.—*Fr.* Intoxication alcoolique.—*Germ.* Säuferwahnsinn. An absurd criticism on the term delirium tremens is that it is the patient not the disease which shakes, and delirium cum tremore has been proposed as more correct. The same would apply to Paralysis agitans, and the English terms a shaking palsy or a burning fever.

[†] This term (literally inflammation of the diaphragm, metaphorically, of the mind) or its equivalents *encephalitis* and *meningitis*, was formerly used as the pathological explanation of delirium or mania with febrile symptoms. But it is now known that the clinical

Symptoms.—The patient has for two or three nights been more disturbed by unpleasant dreams than usual, and has by day become more restless and tremulous. Now he becomes unable to sleep, but as he lies awake at night his dreams still haunt him. Next morning he may again be rational, but towards evening the delirium returns, and the second night is worse than the first. During the second day there is again a slight amelioration as compared with the night, but the mental disorder is now fully established and persistent.

The delirium is in many respects peculiar. The patient is not violent, nor is he depressed in spirits. He is loquacious, and restlessly anxious to follow his accustomed vocation, but he sets about his business in a blundering manner, and his mind quickly wanders away to something else. If spoken to, he is sufficiently intelligent to answer, and for a minute or two he may converse rationally, but before long he starts off on some fresh topic. His friends try to keep him in bed, while he is always wanting to get up and dress himself. Yet, if firmly opposed, he forgets his intention, at least for the time. He suffers from hallucinations of vision; he fancies that rats and mice, or snakes, or cockroaches, are running or crawling over his bed; or he may address persons who are absent as if they were in the room. He will look suspiciously behind the curtains or under his pillow, or will stretch himself out of bed to see if some one is not concealed beneath it. His hands are in constant motion, and he picks at the bedclothes, or grasps at imaginary objects. If asked to put out his tongue, it is very tremulous, and is quickly withdrawn; it is commonly moist, and more or less thickly coated. The pulse is quick, soft, and feeble. The skin is moist and often in a state of profuse perspiration. There is an entire absence of appetite and even of desire for drink. As the late Dr Bence Jones discovered, the amount of phosphates in the urine is greatly diminished.

Event.—The duration of these symptoms is not absolutely constant, but it is more regular than is often supposed. The disease almost always goes on for two and a half days without showing any tendency to subside, and then between the sixtieth and the seventy-second hours it comes to an end. Towards this period the patient is apt to show signs of exhaustion; his pulse becomes more rapid and feeble; his face, at first flushed, is now pale and haggard, and his pupils are widely dilated.

It is, however, precisely at this time that a favourable change is to be hoped for. Towards the end of the third night the patient commonly falls asleep. At first he is still uneasy and restless, his breathing is irregular, and after an hour or two he may wake up for a little while; but he soon goes soundly to sleep again; his breathing is now slow and deep; a profuse sweat breaks out over his body. After six or eight hours he awakes and is relieved. In the next twenty-four or forty-eight hours he sleeps almost continuously, and after this his restoration to health appears complete.

In some cases, particularly in those who have previously been in good health, and in whom the attack has been the direct result of a debauch, the attack terminates earlier—perhaps at the end of twenty-four hours. In other cases it lasts beyond the specified time. Ware speaks of having once known it to extend to nearly six entire days. A duration of more than

type of disease in question is associated with enteric or other specific fevers, with the hyperpyrexia of rheumatism or erysipelas, or with the toxic effects of alcohol; and in none of these disorders is there inflammation of the brain or its membranes. The symptoms of true meningitis are, as we have seen, very different, and there is no evidence that primary acute inflammation of the encephalon ever occurs.

three days is most apt to occur in those who have for a long time been habitually intemperate.

But the end of the disease is by no means always favourable. Sometimes a sudden attack of convulsions occurs, by which the patient is carried off; sometimes he becomes comatose, and sometimes he falls back in his chair and dies unexpectedly by syncope.

The disease has a marked tendency to attack the same patient over and over again, unless he makes an entire change in his habits.

Ætiology.—So far as is known, delirium tremens never occurs except as the result of alcoholic intemperance; but it is often exceedingly difficult to discover how intoxicating liquors are obtained. Women, in particular, will suborn their servants, or procure tincture of lavender or sweet spirits of nitre from the chemist, or drink eau de Cologne.

At one time it was generally believed that instead of the immediate exciting cause of delirium tremens being the alcoholic stimulus itself, the disease usually attacked persons who, from whatever cause, had been suddenly deprived of the drink to which they had been accustomed. There can be no question that before the attack the patient has sometimes left off drinking for a few days; and, again, that it often shows itself in those who have been kept for a long time without stimulants, in consequence of their having broken a limb or received some other injury. But many patients are attacked by delirium tremens as the immediate result of a bout of hard drinking; and the experience of those who have the management of prisons where abstinence is enforced, has demonstrated that this does not in itself bring on an attack, even in the most intemperate.

The facts which have been supposed to establish the contrary are otherwise explicable. A dislike of stimulants is sometimes an early symptom of the disease, so that the reason why the patient leaves off drinking may be that he is already beginning to suffer from its effects. Again, a sudden shock may act as a direct exciting cause of delirium tremens in those who are predisposed to it by intemperance. It is this which renders the disease so common in the accident wards of hospitals; indeed, it often comes on within the first few hours after the patient receives an injury, when there can be no time for the withdrawal of stimulants to take effect. It is also frequently observed in patients of intemperate habits as a complication of acute pneumonia, erysipelas, or some other febrile disease.

Diagnosis.—As stated above, delirium tremens has been formally recognised only during the present century. Sutton had become acquainted with it when practising in East Kent, where spirits brought in by smugglers could be had in abundance and at a cheap rate. Certain of the practitioners in that district, he says, had learnt to treat such cases with opium; and this practice was attended with very marked success in comparison with that of others (including at first Sutton himself), who regarded the disease as phrenitis, or inflammation of the brain, to be combated by venesection, blisters, and purging. Sir Thomas Watson, in his classical 'Lectures,' approaches the subject from this point of view, and tells how he was once summoned to a man who was supposed to be mad, or to have brain-fever, but was really suffering from delirium tremens. At the present day the opposite mistake is more likely to be committed, and a case to be set down as one of this disease when it is one of acute mania or acute melancholia, diseases which (it must not be forgotten) may also follow alcoholic intemperance. Sir Samuel Wilks saw general paralysis of the insane mistaken for delirium tremens on two different occasions and by the same physician. He has also known rheumatic fever, when attended with cerebral symptoms, resemble delirium tremens in its superficial characters.

In the surgical wards of a hospital there is often difficulty in distinguishing alcoholic delirium from the effects of injury to the head. Again, fever or acute pneumonia may be overlooked, and a case wrongly set down as one of delirium tremens, particularly if it is known that the patient has been intemperate. Moreover, we must remember that pneumonia may be almost latent in drunkards.

Prognosis.—Anstie speaks of old age as unfavourable, and one has been always accustomed to think that for young subjects the disease ought to be unattended with danger in a first attack, and even in one or two subsequent ones. Aitken, however, quotes some statistics, based on observations made by Dr Macpherson in the General Hospital at Calcutta, and it is remarkable that there the highest average mortality was in persons between twenty-five and thirty-five years old, being from 23 to 24 per cent. of those attacked, whereas the mean mortality at all ages was about 15 per cent. Among fifty-nine cases of fatal delirium tremens which occurred at Guy's Hospital, there were three in persons between twenty and twenty-nine years of age, eight in those between thirty and thirty-nine, ten in those between forty and forty-nine, and eight in those between fifty and sixty. What ratios, however, existed between these numbers and those of all patients attacked by the disease during the corresponding periods is uncertain.

The state of the kidneys affects the prognosis in delirium tremens very materially. The urine must be repeatedly examined for albumen and for casts; and the quantity passed each day must be noted. If the secretion should begin to fail, Anstie recommends hot fomentations or dry cupping to the loins, hot foot-baths, and the administration of half-ounce doses of infusion of digitalis every three hours. Digitalis was recommended as a remedy for delirium tremens itself by the late Mr Jones, of Jersey, who used to give enormous doses of the tincture, and clearly showed that digitalis is one of the poisonous agents for which there is marked tolerance in this disease.

Next in importance for the prognosis of delirium tremens is the state of the heart. Anstie remarked that, beside feeling the pulse, one should carefully watch the first sound of the heart with the stethoscope; and he laid still more stress on the use of the sphygmograph. An "irregularly undulating" character of the pulse-wave—such as occurs in the typhoid condition—was found by him to be of the most unfavourable augury. In his article in Reynolds' 'System of Medicine,' a tracing is given which was taken from a man, aged forty, who, after being delirious for nearly a week, fell into a sound sleep of six or seven hours' duration, and when he woke appeared to be so much better that a confident opinion was expressed that he would recover. The sphygmogram, however, led Anstie to a grave prognosis, and about twenty-four hours later the patient died.

Treatment.—The observers who first distinguished delirium tremens from acute phrenitis obtained what seemed to them wonderfully successful results by treating it with opium. Watson, for example, recommended that three grains of solid opium should be administered as soon as the bowels have been cleared out by a moderate purgative; and that if at the end of two or three hours the patient should show no inclination to sleep, one grain should be given every hour afterwards until the result is attained.

But this advice was based on the supposition that the disease, instead of subsiding spontaneously, would run on, and prove fatal unless the patient were made to sleep. Watson remarks that delirium tremens is not likely to be "healed with a *placebo*, or by waiting upon nature;" and it is clear that the physicians who laid stress on the value of opium measured their success by comparison with cases of supposed meningitis or phrenitis treated by venesection, leeches, and blisters. But most modern writers endorse Ware's original statements as to the natural course of the disease.

It is still, however, a question whether this course may not be shortened by hypnotic medicines. Anstie thought that this might be effected by chloral hydrate, of which he gave thirty grains for the first dose, and repeated it in an hour if the patient did not sleep. He found that the patient almost always got two or three hours of sound repose, sometimes much more; and he believed that in delirium tremens there is a tolerance for chloral, so that as much as ninety grains or even two drachms may be safely given in divided doses within twenty-four hours, at least for a day or two. But Wilks is disposed to share Ware's opinion that the duration of the disease cannot be shortened by giving medicines to send the patient to sleep. He thinks that a certain time is required for the subsidence of the disorder, and that a severe attack must last at least three days.

If this view is correct, it would seem to follow that at the first onset of delirium tremens neither opium nor morphia should be given at all, or at least that their administration should not be repeated so as to cause contraction of the pupils. For, when hypnotic remedies are actively pushed, one is apt to find oneself after two or three days in a difficult position. The patient, instead of sleeping, may be as excited as ever; his pupils are perhaps reduced to the size of pins' points, and he has had as much opium as would kill two or three healthy persons. Under such circumstances—which are precisely those in which a consultation is most likely to be sought—there is no question that the proper course is to wait, for the further administration of hypnotics is dangerous. Wilks has many times seen persons suffering from delirium tremens sent to their last sleep by opium, and the same result has followed the subcutaneous injection of morphia. A patient of Dr Fagge's in these circumstances became collapsed rather than comatose; indeed, as already mentioned, the disease sometimes terminates by sudden collapse, even when no hypnotics have been given.

Another question, which has sometimes been raised when a patient has died after the administration of a large dose of opium, is whether one can infer that this could not have been the cause of death from the fact that a certain interval of time had elapsed. In such a case Sir Thomas Watson decided that the medicine could not have been concerned in bringing about the fatal result, because nine hours passed after the last dose (one of three grains) was given before the patient became comatose. This conclusion might be true of a healthy man, but not of a patient suffering from delirium tremens, for, as the late Sir Robert Christison long ago pointed out, the effects of opium are much retarded by intoxication with alcohol.

But whatever doubts may fairly be entertained as to the part played by opium in bringing about a fatal result in cases of this kind, there can be none as to the importance of treating delirium tremens so that no such question can be raised. We have seen that most patients do well when no opium at all is given, and therefore the fact that many recover after having taken heroic doses is no justification for administering them.

If a hypnotic is needed, sulphonal, trional, or chloralamide is safer and not less effectual than opium or chloral.

When faintness, irregular pulse, and a livid countenance indicate approaching failure of the heart's action, stimulation is needful.

Bristowe believed that opium may still be used even in full doses in delirium tremens with good results, if given early and watched; but before giving either opiates or stimulants it is important to see that the bowels are freely open.

The patient should be well supported by nourishment from the first. He may have milk if he will take it; but if not, he must be compelled to swallow strong beef-tea, or soup. He may even have small pieces of underdone chop or steak, if he can be induced to eat them.

The administration of alcohol in moderate doses was formerly recommended in cases of delirium tremens as a routine practice; it was thought to aid in inducing sleep. But all those who have studied the natural course of the disease are now agreed that there is no proof of its acting in this way; and the necessary stimulation may be more safely given by means of ether or ammonia. In many cases the subcutaneous injection of three or four drops of liquor strychniæ is a most valuable means of improving the heart's action.

Another matter of great importance is that the room should be kept cool and dark and quiet. The patient's friends, who commonly collect around him, must be sent away, one or two intelligent and able-bodied men being alone left to watch him. The lights are to be shielded, and in the daytime the window-curtains drawn. The patient often tries to get out of bed, and if his attendants can by persuasion, or by employing a little force from time to time, induce him to lie down quietly, that is no doubt to be preferred; but it is far better for him to be kept in a recumbent position by a sheet folded across his chest and tucked well in, than for him to be violently held down and restrained for any length of time by the hands of those about him.

The first point in treatment is to give a good purge, and to ascertain the condition of the urine; the second to sustain the strength with food, whatever can be taken; the third to procure sleep by quiet, or by such sounds as slow music, or by such drugs as chloral, bromides, sulphonal or chloralamide; and the last to watch the heart and pulse, and to use ammonia, ether, strychnia or digitalis, strophanthus or caffeine, to avert death by syncope.

Chronic alcoholic neuroses.—Quite apart from the action of alcohol upon the nervous centres in the acute disease just described, a long-continued state of nervous disorder is a more frequent effect of intemperance.

The most important of these disorders is muscular tremor. This is more commonly noticed in the hands, which are unsteady and shake; but Anstie found that in a majority of cases the lower limbs were really affected before the upper. The patient is often able at first to control the tremor by an effort of the will. It is generally more troublesome in the morning than at any other part of the day, and it may render him unable to do any work requiring nicety of manual adjustment, until he has taken a dram, or eaten some food. Even before marked tremor occurs, the action of alcohol is sometimes manifested by a peculiar restlessness; the limbs are apt to start involuntarily, and cannot be kept quiet except by an effort of attention. At the same time there is an irritable condition of the mind; the patient complains that though he may feel drowsy when he goes to bed, he cannot sleep, but keeps turning from side to side.

At a somewhat later stage other cerebral symptoms develop themselves. A buzzing or rushing sound in the ears may be heard; and with it there is often a dull diffused headache. Muscæ volitantes are complained of; flashes of light seem to pass before the eyes, especially at night, just when the patient is dropping off to sleep; and there are momentary attacks of vertigo.

The intellectual and moral powers gradually become impaired. All certainty of purpose is lost, and there is mental disquiet which makes it impossible for the patient to settle down to any occupation or to complete the tasks he may begin. He often has a vague feeling of dread for which he cannot account; or he may become subject to a delusion that some one is lying in wait to injure him. Anstie mentions as another symptom that the patient often has a vivid apprehension that he is in danger of falling down a precipice, even when walking on firm ground in broad daylight.

Another occasional symptom consists in pains in the limbs, especially around the wrists and ankles, as well as in the shoulders and down the spine. They are somewhat paroxysmal in their character, returning each day at about the same hour, most commonly towards night; and they are greatly aggravated by fatigue, whether of mind or body.

Impairment of sensation is also apt to occur, especially in the upper limbs; and the power of muscular co-ordination may be lost, so that the state of the patient closely resembles that produced by tabes dorsalis. Epileptiform convulsions sometimes show themselves, and they are of the gravest augury. The mental state often passes into mania or melancholia, and ultimately complete dementia.

Indications of gastric disorder are present at an early period. A common complaint is that of nausea in the early morning, or of actual vomiting, and this may recur day after day as regularly as during early pregnancy. Morning diarrhœa is another frequent symptom. There is generally a failure of appetite, particularly at breakfast-time; the tongue is foul, with a thick yellow fur; or, less frequently, red and glazed, and it is tremulous when put out. The breath acquires a peculiar fœtor, which is not that of any alcoholic drink, and which can hardly be described, although when once smelt it is unmistakable. The eyes are red and watery; the conjunctivæ are often slightly jaundiced; the features look flabby and expressionless; and the nose, cheeks, and forehead are reddened, with darker crimson points, called *stigmata*.

Sometimes the nose, cheeks, and chin are not only red but covered with papules or pustules, a condition known as "acne rosacea," or the nose may be enlarged, with pendulous outgrowths. However, it must not be understood that the appearances last mentioned are seen only in those who have indulged in alcoholic excesses; they are sometimes seen in persons who have been perfectly temperate. Acne rosacea—or gutta rosea, as it is more properly termed—is, in fact, a symptom of dyspepsia, most often, but not always, the dyspepsia of drink. Moreover in women it may be the result of disorder of the menstrual functions.

The limbs of a person suffering from chronic alcoholism are generally wasted, especially the legs. The abdomen, on the other hand, is often large or pendulous, and its parietes, as well as the contained viscera, may be loaded with adipose tissue. In those whose chief beverage has been beer, even the limbs may be covered with fat; and since the interstitial connective tissue of the muscles shares in the process, there is often apparent bulkiness of the fleshy parts of the arms and legs.

With regard to the chronic action of alcohol upon the nervous centres Anstie believed that, contrary to what is the case with the digestive organs, the effects depend almost solely upon the quantity taken, and not upon the form in which it is taken. That ardent spirits differ in their general effects from wine, and both from malt liquor, is a matter of common experience, as illustrated by Hogarth's print of 'Beer Street and Gin Lane;' but the effect on the nervous system does not seem quite the same; spirits lead not only to hepatic cirrhosis and ascites, but to delirium tremens; ale and beer to fatty liver, overgrowth of fat about the heart, often with hypertrophy as well, and rather to lethargy and inaction than to excitement and frenzy.

There are immense differences in the liability of different individuals to suffer from alcoholic excess. Some men can drink spirits freely for years, and seem to be none the worse for it; others break down in health under comparatively small amounts of drink. Women show little power of resisting its evil effects; perhaps because those who are intemperate pass all their time indoors.

Among the *antecedents* of intemperance must be mentioned, in the first place, those occupations in which persons are brought continually into contact with intoxicating liquors. A large proportion of the patients who present symptoms such as have been described are men employed in breweries or distilleries, public-house keepers and their wives, and travellers for wine and spirit merchants. Cab-drivers and hawkers, and others who are exposed to rough weather, are also tempted to intemperance; while for some persons, as cooks and shoemakers, it is admitted as an excuse that their occupation is monotonous, deprives them of proper exercise, and keeps them confined in close, ill-ventilated rooms. Depressing mental influences drive others to drink; poverty and misfortune make them eager for the oblivion of intoxication. Again, there are cases in which stimulants were at first taken for the relief of pain, but in which the habit of indulgence grows upon the patient until it becomes a disease. Anstie has rightly laid great stress on the responsibility of allowing women to take wine or brandy in order to render them less susceptible to neuralgia, or to the sufferings which are so apt to attend on the menstrual period.

Lastly, there is no doubt that proclivity to intemperance is capable of hereditary transmission. This tendency is by some writers regarded as itself a neurosis, to which they give the name suggested by Roesch, "Oinomania," more correctly "Œnomania." Whether acquired habits of drunkenness are hereditary is very doubtful, but the congenital tendency thereto may be transmitted like that to gout or any other disease. In such cases the dipsomaniac is seized every few months with a craving for drink, and for days together he behaves like a madman, taking long journeys without any purpose, eating little and drinking much; but when the affection passes off—at the end of a month or so—he regains his usual health. He then lives soberly and chastely, and manages his affairs with discretion.

The *morbid changes* that are found in the nervous centres of drunkards are essentially atrophic. The cerebral convolutions are wasted, and the cerebro-spinal fluid is in excess; the pia mater is opaque and thickened, and even the bones of the cranium are sometimes found denser than normal and without diploë.

The effects of chronic alcoholic poisoning in producing certain forms of spinal paraplegia, and the still more characteristic paralysis due to peripheral neuritis have been already discussed (p. 595).

The *diagnosis* of chronic alcoholism may present every degree of difficulty or it may be perfectly easy. Commencing general paralysis, locomotor ataxia, and hysteria are perhaps the conditions most likely to be confounded with it. Unfortunately one cannot place any confidence in the statements of the patient himself, however guardedly our questions may be put, for persons who indulge in secret drinking are always untruthful.

In the *treatment* of chronic alcoholism the most important point is that the patient should abstain at once and entirely from all intoxicating drinks. It is in such cases that large "hydropathic" hotels are of service, where regular habits and exercise in the open air are enforced, while an unstimulating diet is provided and all facilities for procuring drink are withdrawn.

Certain medicines may also be of considerable secondary service. Anstie recommends especially quinine, which (if there should be sickness) may be given in a state of effervescence. Marcet's favourite remedy was the oxide of zinc; he says that in doses of from two grains upwards thrice daily it has a powerful effect in inducing sleep. Bromide of potassium often does great good in cases of this kind. To relieve the imperious craving for drink, aromatic spirits of ammonia, tincture of nux vomica, and tincture of capsicum in a bitter infusion are useful drugs. According to Anstie, half-drachm doses of spirit of ether with the same quantity of tincture of sumbul may restore tranquillity to the nervous system and make sleep possible. He also recommends from a quarter to half a grain of the extract of Indian hemp. Chloral hydrate and hyoscyamus are likewise valuable hypnotics. If opiates are absolutely necessary, one should always employ the hypodermic injection of morphia (from one tenth to a quarter of a grain) rather than give any preparation of opium by the mouth, and this only when the urine is free from albumen and the patient has been purged. Even in advanced stages Anstie obtained striking benefit from the administration of cod-liver oil; and he says that when there is much tremor, strychnia is often useful in a dose of $\frac{1}{48}$ to $\frac{1}{32}$ of a grain three times daily.

As to *prognosis*, the symptoms of chronic alcoholism may last for years; and even the hardest drinkers sometimes reach old age. They are apt to become prematurely old, their hair turning grey and their arteries becoming rigid. Gout, Bright's disease, cirrhosis of the liver, or fatty degeneration of the heart may be developed, and cerebral apoplexy may suddenly cut them off. Such diseases as pneumonia or fever are ill borne, and are very likely to prove rapidly fatal, as are also accidental injuries and surgical operations.

NEUROSES FROM MORPHIA.—The evil which arises from the abuse of that invaluable drug, opium, has long been recognised. It is taken as a valuable medicine in malarial districts—in Cambridgeshire, for instance; and probably its abuse began in the same way in China. It produces loss of appetite, constipation, and emaciation, with the deterioration of the character which always follows indulgence in what is known to be an evil habit. Coleridge, after many years of uselessness and misery, emancipated himself from the thraldom. De Quincey has left in his 'Confessions of an Opium-eater' a monument of the pleasures, the pains, and (it must be added) of the incapacity to tell the truth which this habit produces.

Opium-smoking as practised among the Chinese is said to be even more injurious than opium-eating.

The useful and convenient practice of subcutaneous injection of morphia has of late years brought with it, in Western Europe and in America, a more widespread abuse than that of opium. The morphia-habit, as it has been termed, seems to be more prevalent in Germany than in England. It usually begins in the legitimate use of the anodyne to relieve pain, but the result is so rapid and effectual that the patient is tempted to make the punctures himself, and, once familiar with the practice, he continues it long after the original disorder for which it was prescribed has disappeared.

The patient suffers terribly from insomnia and restlessness, he (or she) loses appetite and becomes pale and sallow, sometimes constipated and sometimes troubled with frequent and irritable action of the bowels. Moreover, sooner or later some of the punctures are sure to suppurate.

The writer has only seen a few cases of this malady. The first was an old gentleman from America, who had long become accustomed to the habit and suffered comparatively little. The next was an elderly lady, who had begun the practice for facial neuralgia and succeeded in breaking it off. The third was a young American, who suffered from obscure pains in the region of the gall-bladder. A fourth was a maiden lady, who began the habit to allay the pain of a renal calculus, and who was cured of both at last. In a fifth case, where the arms were scarred all over with the marks of abscesses, abdominal pain had been the origin. A sixth occurred in a young man, the subject of severe and complicated caries of the bones and joints, and he more than once nearly killed himself with a large dose.

The prognosis is not worse than in cases of alcoholic intemperance. The patient must be encouraged to bear the pain, and when unsupportable it must be relieved by smaller doses of the remedy. Often a single drop of the officinal solution is sufficient, and not infrequently pure water has, by a pious fraud, been successfully substituted for the liquor morphinæ. Moreover, bromides, chloral hydrate, hyoscine, sulphonal or chloralamide may each be substituted with advantage in its turn.

NEUROSES FROM OTHER DRUGS.—The *bromides*, if long given in large doses, produce very unfavourable effects on the intelligence, of which an example which came under the writer's care is mentioned at p. 929.

Chloral hydrate produces a still more miserable condition of depression in some patients, and, valuable as it is, should never be used as an habitual hypnotic.

Arsenic is an occasional cause of paralysis, affecting the lower extremities more frequently than the arms. This, like lead-palsy, depends on peripheral neuritis.

While these sheets are passing through the press (Nov., Dec., 1900), the medical journals contain accounts of a widespread and often fatal epidemic of multiple peripheral neuritis in and about Manchester, which has been traced to arsenical poisoning. The vehicle of the poison was beer, and its origin was in the pyrites from which is manufactured the sulphuric acid used in malting.

The effects of *lead* on the nervous system are eclampsia, and sometimes mania or insanity, atrophy of the brain, and peripheral neuritis, chiefly affecting the upper limbs, either the extensors primarily or the shoulder-muscles, or the hand; but occasionally the peronei and other muscles of the leg and, still more rarely, those of the larynx (cf. p. 597).

*Mercurial tremors.**—This disorder, once common among looking-glass makers and other workers in quicksilver, has now happily become very rare.

The following is De Haen's account of the disease in one of the miners of Idria more than a century ago, as cited by the late Dr Sanders, of Edinburgh:—"Deaurator, quinque et viginti annorum, horrendo artuum omnium maxime superiorum vexatus, ita ut nec comedere, bibereve solus, neque loquens amplius intelligere potuerit. Nutriendus, vestiendus, et (infantis instar) alvum urinamque positurus, adjuvandus erat: dolorum cæterum immunis."

The first thing which a man notices when he is beginning to suffer from mercurial tremor is that he is no longer sure of his arm and hands. So soon as he attempts to take hold of anything they shake. When his arm is bent it is not by a continuous motion, but by fits and starts; his hand cannot be directed with precision to any object, and even when he has grasped it, he is often unable to let it go. Before long the lower limbs are affected; he may have to be led, and when he walks his limbs may tremble and dance, so that (as Watson says) they look as if he hung upon wires. His tongue becomes tremulous, and his speech hurried, jerking (or *staccato*), and at length unintelligible; this last symptom has been termed *psellismus mercurialis*. When the tremor has once been excited by exertion or emotion, the patient is unable for a time to stop it; but if he remains quiet for a little while it passes off, and does not return until he has to make some fresh effort, like the "intention-tremors" of insular sclerosis. Getting some other person to grasp the affected limbs sometimes controls their movements.

The onset of mercurial tremor is generally gradual, but sometimes almost sudden. It is said to be more apt to occur in the cold and damp of winter than at other seasons.

Salivation often accompanies the more severe forms of mercurial tremor. The teeth become loose, the gums are ulcerated, the breath is fœtid. The patient grows weak, anæmic, and thin. After a time he begins to suffer from headache and loss of memory, and at length the disease may end fatally by delirium and coma.

If he is withdrawn from the poisonous influence, the tremor subsides in two or three months; but sometimes the recovery is incomplete, and the upper limbs remain unsteady.

Mercurial tremor was formerly common in this country among "water-gilders," who employ for gilding an amalgam of mercury and gold, from which the volatile metal is expelled by the action of a charcoal fire; but water-gilding is now superseded by electro-plating. Another trade in which mercury is used is "silvering" mirrors; this, however, comparatively seldom gives rise to the tremor, probably because little of the metal is volatilised, since heat is not employed. But mercury does give off vapour to some extent, even at 68° or 70°, so that the disease may show itself in those who have to deal with the metal at ordinary temperatures.

In 1864 a man, suffering from an extreme form of mercurial tremor, was admitted into Guy's Hospital under Dr Rees. He had been packing up skins previously washed with an acid solution of mercury. He said that he had at one time been salivated, but some doubt would perhaps have remained as to the cause of his symptoms, which terminated fatally, had not

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* *Synonyms.*—Mercurial Shaking Palsy—The "trembles."—*Fr.* Tremblement des Doreurs (Mérat, 1812).—*Germ.* Mercurialzittern.

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Dr Swayne Taylor succeeded in obtaining mercury from the kidneys, and, in smaller quantities, from the brain and liver (‘Guy's Hospital Reports,’ Third Series, vol. x, p. 176). This man had worked at his occupation for three years before he began to suffer much. Watson relates the case of a man who had been a water-gilder for seventeen years before he was attacked.

It is possible that the disease is sometimes due, not to the inhalation of mercury, but to its absorption by the skin. Dr Mapother states that the anatomy porter of the Irish College of Surgeons, who at one time rubbed enormous quantities of mercury into patients affected with syphilis, was for thirty years subject to mercurial stammering.

The *diagnosis* of mercurial tremor is easy; but in its earlier stages it is liable to be mistaken for paralysis agitans, and in its later periods for chorea. Anstie (‘Lancet,’ 1872, i, p. 734) mentions the case of a man who was supposed to have died of delirium tremens, until it was discovered that in his trade he had been inhaling the fumes of the acid nitrate of mercury.

Prophylaxis and treatment.—Much has been done to prevent this disease in those whose occupations expose them to it. The workshops are freely ventilated and provided with flues or chimneys; and often a glass screen interposed between the upper part of the workman's body and the fire by which the mercurial vapour is set free. Personal cleanliness, frequent ablutions, and avoiding to take food in the room where the work is carried on, are important; and drunken habits are injurious, perhaps by favouring disease of the kidneys. Like other forms of tremor, that which is caused by mercury is often diminished for the time by a glass of wine or of some other stimulant. Sir Thomas Watson's patient said that when the disease was first coming on he found himself unable to get upstairs to his work until he had taken half a quartern of gin.

Among medicines iodide of potassium appears to be the most useful. It was suggested by Melsens on the same theory as for chronic lead-poisoning, and mercury is said to have been detected in the urine while iodide was being taken. Watson found iron more useful than any other drugs in the case which he relates, and Anstie saw great benefit from cod-liver oil in several cases. In a patient of Gull's, electricity appeared to be beneficial, sparks being taken from the spine; it was thus that De Haen's patient (whose case was quoted above) was cured.

The other effects of mercury are salivation and, probably, stimulation of the pancreas, liver, kidneys, and tubular glands of the intestine. It was proved by Rutherford that it does not increase the flow of bile in dogs with a fistula into the gall-bladder, but it certainly restores the colour to the fæces in man. The result of mercurial poisoning is to produce pallor by diminution of the hæmoglobin of the blood-discs; but it has been ascertained that this is not the effect of the drug in cases of syphilis; not only does the sallow, earthy hue of the disease give way to a healthy colour, but the actual amount of hæmoglobin is increased.

Tremors from zinc-poisoning.—There is a curious disorder to which brass-founders are liable; it was studied by Dr Greenhow in Birmingham, in 1858, and named by him “brass-founder's ague,” on account of the resemblance between its symptoms and those of a paroxysm of intermittent fever (‘Med.-Chir. Trans.,’ vol. xlv). It is in no way dependent upon malaria, but is caused by the dense white fumes of oxide of zinc, the result of deflagration of the metal, which at a certain stage in the formation of brass fill the

ing-shop, unless it is thoroughly ventilated. Exposure to these fumes is
owed by malaise and weariness, by a sense of constriction at the chest,
d sometimes by nausea. Afterwards shivering comes on, with chattering
the teeth; this is succeeded by a more or less marked hot stage, and the
ck ends in profuse sweating. Next day the man feels unwell, but not
ugh to be unable to work. There is no regularity in the recurrence of
attacks, which come on from time to time under the operation of their
ting cause, but especially when the patient has been off work for a few
s. They seem not otherwise to impair the health, and it is not known
they shorten the life of the patient. Drinking milk is said to be pre-
ive of the brass-founder's disease.

Dr Greenhow reported that, beside this affection, brass-workers at Bir-
am are very liable to shaking palsy. He could only find, however,
case in an old man. Dr Gee published in the 'St Barth. Hosp.
for 1889, p. 28, a case of a brass-founder, aged thirty-three, who
ed from muscular paresis, trembling tongue, and ankle-clonus, which
oms were ascribed to zinc-poisoning. See also a good account of the
ed "ague," the bronchitis (coniosis), and the nervous symptoms to
n the Birmingham brass-founders are liable, by Dr Robert M. Simon
rit. Med. Journ.,' April 28th, 1888). He is inclined, with Dr Hogben,
tribute part of the poisonous effects to the copper, rather than to the
of the compound metal.

HYSTERIA AND HYPOCHONDRIASIS

> **"Orandum est ut sit mens sana in corpore sano."**
> **JUVENAL.**
>
> **"In nervous diseases he is the best physician who knows best how to inspire hope."**
> **COLERIDGE.**

HYSTERIA—*Nomenclature—A real and distinct disease—Mental and moral perversions—Hysterical affections of sensation and of movement—Contractures—Globus hystericus—Flatulence—The hysterical fit—Ætiology and Pathology of Hysteria—Diagnosis—Prognosis—Prophylaxis—Treatment.*

Catalepsy — Trance — Sleep-walking — Ecstasy — Dancing mania—Hysterical mania—Epileptiform hysteria—Anorexia nervosa—Hysteria in men and boys.

HYPOCHONDRIASIS—*Distinctions from hysteria—The supposition of imaginary diseases—Distinction from melancholia—May mask real disease—Treatment—Prognosis.*

IN many of the diseases of the nervous system which have been described in the preceding pages, mental symptoms mingle with those in which consciousness is not involved. The coma of apoplexy, the delirium of poisoning by alcohol or belladonna, the temporary unconsciousness of minor epilepsy, the paroxysmal mania which follows major epilepsy, and many symptoms of "general paralysis" are mental. But there are disorders in which, instead of the mind being affected as the result of anatomical lesions or poisons or functional disturbance of motion or sensation, the mental aberration is primary, and various bodily disorders result therefrom; and yet the patients are not insane. These disorders will be treated in the present chapter under the traditional terms of Hysteria and Hypochondriasis.

HYSTERIA.*—This ancient name implies the erroneous theory that disturbance of the uterus is the cause of the curious and long-recognised paroxysmal attacks known as hysterical fits. The hypothesis was crude and almost baseless, and the term has been extended to many other conditions beside the "fit of the vapours" which it originally denoted. It is idle to quarrel with this extension of meaning—an extension not arbitrary or designed, but natural and common to all languages,—and it is pedantic to object to our speaking of male hysteria because men have no uterus. We might as well refuse to use such terms as pituitarium, rheumatism, or horea.

It must be admitted that, like some other diseases, hysteria is difficult to define. Since it is independent of any appreciable structural change, its definition would naturally be based upon either its causes or its symptoms. But the former are unknown, and the latter are singularly variable and inconstant. Moreover, the curability of most hysterical affections, and the fact that they are generally contrasted with organic diseases, have caused a tendency to apply the term vaguely to cases of unascertained nature, but which are regarded as of a trifling character, and likely to be of transient duration. In this way the words hysteria and hysterical have often become meaningless.

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* Ὑστερία (*i. e.* womb-sickness), from ὑστέρα, the uterus—Passio hysterica.—*Anglicè* its of the mother—The vapours.

There is, however, no question that most of the affections called hysterical are the genuine expression of a special morbid condition, for which the name of Hysteria is as convenient as any other, beside having the sanction of antiquity and established usage. For we find that two or more of these affections are frequently present in the same patient at the same time, or that a person who has suffered from one is afterwards attacked by others. Then, again, there are marked peculiarities in the age, sex, temper, and other habits of such patients. Above all, there is in most cases an intellectual and emotional state which can be recognised apart from other indications of the disease, and may be regarded as its fundamental character.

Mental condition.—The principal features of the mental state which characterises hysteria are a disturbance of the sensations, an exaltation of the emotions, a perversion of the will, and a loss of the balancing power of the judgment. The patient seems to have no power of controlling her own feelings, so that trifling occurrences excite her to rapture or plunge her in despair. She may conceive a violent affection for some persons, and an equally irrational aversion from others; or she perhaps shows great attachment to some pet animal, while others, no less harmless, excite repugnance and disgust; she torments all about her with her caprice and her selfish claims upon their attention; she appears unable to make up her mind to the smallest effort, and lies in bed all day from sheer want of energy to rise: and yet in some particular direction she will show the most obstinate tenacity of purpose. She craves for sympathy, and insists on her delicate health being recognised. In pursuance of this object she will apply corrosive acids to her skin, swallow needles day after day, or run them into all parts of her body, and make repeated, but always unsuccessful, attempts to poison or drown or hang herself. Her stoical endurance of pain is sufficient to show that a mere defect of volitional power cannot be the essential element of hysteria. As Reynolds (' Syst. of Med.,' vol. ii) put it, the will is no longer called into exercise by the judgment or reasoning faculties, but only by some one dominant idea or emotion. Thus, as Jolly observes (von Ziemssen's ' Handbuch,' vol. xii), if one can by stimulating a spirit of emulation supply a hysterical patient with a sufficient motive to undertake a good work, such as nursing, she will often carry it out with unusual perseverance and energy.

Moral perversion.—The moral tone of a hysterical woman is often seriously impaired, particularly in certain directions. Morbid sexual inclinations have in some cases a strong hold upon such a patient, and she will scheme to induce her medical attendant to make a vaginal examination. Watson relates a case in which a young woman contrived to make a hospital surgeon believe that she had a stone in the bladder; and the imposture was not detected until she had been tied up in the position for lithotomy in an operating theatre full of students. A patient who was supposed to have a hydatid in the liver, and who had been punctured without result, allowed the operation to be repeated, and almost immediately produced a piece of the stomach of a rabbit, which she declared she had vomited, and which she no doubt thought would be mistaken for a hydatid membrane; a few hours later she sent for her medical man again, to remove from her vagina another piece of the same substance. Others have been known to drink their urine and then to bring it up again, pretending that none was passed in the natural way, and the fact that such cases have occurred makes one hesitate to accept as authentic cases described by Charcot as hysterical ischuria; for there is often great difficulty in detecting impostures of this kind. Charcot himself relates a case of Boyer's, in which a woman pretended that urine came from her umbilicus, ears, eyes, and breasts, and that she vomited fæces; and it was not until she had been placed in a strait waistcoat that little balls of fæces ready prepared for swallowing were found in her bed. Hysterical young women also simulate phthisis and pretend to spit or vomit blood.*

Some of the factitious affections of the skin are by no means easy of diagnosis. In the museum of Guy's Hospital there is a model of the right breast of a girl, which is reddened and has on it a number of raised nodules, some of them larger than peas. The first time that patient was in the hospital the cause of the affection remained undiscovered, although it was evident that some irritant was being intentionally applied; but more than a year afterwards she was again admitted, and Mr Birkett, happening to visit the ward at an unusual hour, found a piece of lint, strewn with powdered cantharides, which she had put on the breast. Sir William Gull related a similar case in which he detected with a lens a glistening fragment of the same powder on the skin. Some years ago a girl was admitted into Miriam Ward who had on her chest and breasts several whitish gangrenous-looking patches, of irregular size and form; these successively shrivelled up into brownish scabs, which soon became detached, leaving the skin beneath reddened and rather scaly. On one finger, in the groove between the skin and the nail, there was found a bright yellow stain, which affected both structures to about the same extent, and which seemed indisputably to have been caused by a drop of nitric acid. So that perhaps the patches on her chest were likewise produced by the acid, although they did not show the characteristic yellow colour. It happened strangely enough that shortly before she came in there was in the ward another patient, a girl of eighteen, affected in exactly the same way with what there was no reason to doubt was spontaneous gangrene. The museum contains models taken from each of these patients (Nos. 296, 299).

Other forms in which hysterical patients exhibit their craving for notoriety are illustrated by the cases of the Welsh fasting girl, and Louise Lateau, the Belgian nun.

Among the many diseases simulated by hysteria, the following may be noted:—Paraplegia, Aphonia, Hæmoptysis and phthisis, Hæmatemesis and gastric ulcer, Dyspnœa and pleurisy, Caries of the spine and hip disease, Synovitis of the knee or other joints, Abdominal tumours, bleeding stigmata, Epilepsy, Hydrophobia, and Hyperpyrexia.

Perversions of sensations.—The hysterical patient sometimes complains of intolerance of light, and will have the room darkened. Reynolds relates the case of a woman who had for weeks been lying with her hands before her eyes to keep out the light of a dull London sky. When he brought a candle close to her in order to examine the pupils she shuddered, knit her brows, and held both hands between it and her eyes; but as soon as her attention was distracted to the state of her front teeth, the brows became relaxed, the hands were removed, and she bore the light without inconvenience. In other cases the patient is distressed by the slightest sound, and will allow no one to speak above a whisper; and yet will speak in a loud voice or make a noise in stirring the fire without seeming to mind it.

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* A remarkable case of this kind obtained some notoriety about fifty years ago by the publication of a book called 'The Female Jesuit in the Family.' The patient, a typically hysterical governess, imposed on the Protestant credulity of the household.

Jolly quotes from Amman the case of a woman who discovered the presence of some cherries in another room by their odour, and who could distinguish one person from another with her nose; and such persons often detect by their taste the presence of the minutest trace of any flavour that they dislike. It is perhaps a perversion of the gustatory sense that leads girls affected with hysteria to eat cinders, sealing-wax, lead pencil, and the like; this perverted appetite used to be called "*pica*." They are sometimes equally fond of repulsive odours or flavours.

The sense of touch, again, may be unduly acute. In almost every case of hysteria there is at some part or other of the body over-sensitiveness to painful impressions—*hyperæsthesia*, or rather, in strict language, *hyperalgesia*. Sometimes the patient complains bitterly if the skin over one or more of the spinous processes is pressed upon or even touched; sometimes she has extreme tenderness of the breast, or of the edge of the costal cartilages.

In some cases there appears to be an actual exaltation of perception: a hysterical girl may seem to hear through stone walls, but deception is often practised, especially the deception by which the patient deceives herself.

Another frequent effect of hysteria is impairment of common sensation, *dysæsthesia* or *anæsthesia*. This may either be limited to ordinary tactile impressions or it may include those of heat and cold; or it may concern only the perception of pain, *analgesia*. Gendrin went so far as to declare that sensation was more or less defective in every case of hysteria. This statement is too absolute, but the symptom is undoubtedly often present, and Lasègue and Charcot are probably right in saying that it would be more frequently observed if it were carefully looked for, since the patient may be unaware of local anæsthesia until her sensibility is tested. In many cases subjective sensations of tingling, pins and needles, are complained of by the patient, or numbness, or local acute pain. Nor is hyperæsthesia incompatible with anæsthesia. The sense of touch, of muscular contraction, or heat and cold, are each independent, and in disease may be separated from the totally distinct perception of pain (cf. *supra*, p. 632). Indeed, acuteness of discrimination of the tactile, muscular, or thermic perceptions is impaired by pain. So there is nothing incredible in an hysterical girl telling us that a certain part of the skin is exquisitely tender, and the seat of burning pain when touched; while yet at this spot the power of appreciating tactile impressions may be greatly diminished.

But the most characteristic sensory symptom is ***Hemianæsthesia***. Briquet stated that this occurs on the left side more often than on the right side in the proportion of seventy cases to twenty. The limitation of the parts in which there is loss of feeling is often remarkably sharp, corresponding almost precisely with the median line of the face, neck, and body. It differs from hemiplegia in its wide extent and in the greater implication of sensation than of motion. The mucous membranes are affected in a similar way; taste may be wanting in one half of the tongue, the sense of smell may be impaired, and there may be a considerable degree of amblyopia, with limitations of the visual field for the several colours—a symptom which Galezowski has named *Achromatopsia.* Occasionally we see complete hysterical hemianopia. According to Jolly, reflex excitability is sometimes wanting; sneezing cannot be induced by irritating the nose, nor retching by tickling the fauces on the affected side. Lastly, Charcot noticed that the skin of the paralysed side is cold and pale, and yields scarcely any blood to a needle's prick.

Apart from hemianæsthesia, loss of feeling in the mucous membranes is common. Anstie remarked on the frequency with which it may be found, if searched for, in the back of the pharynx ('Lancet,' ii, 1872), and believed that whenever a person who has not been taking a bromide can without retching let one pass the finger down to the epiglottis, the diagnosis of hysteria is probably correct. Reynolds mentions several cases, occurring in married women who were still bearing children, in which there was absolute anæsthesia of the vulva and vagina. Impairment of sensation in the bladder is supposed to be sometimes the cause of retention of urine in hysterical patients, but in other cases it appears to be due to a prurient desire to have a catheter introduced. Jolly relates the case of an hysterical patient who burnt herself terribly by taking live coals out of the fire and pressing them with both hands into her vagina, without showing any sign of pain.

Hysterical anæsthesia is not permanent. It may last for several weeks and even for months, but sooner or later it always passes off.

Motor paralysis.—Another manifestation of hysteria is paralysis of movement. Aphonia and dysphagia often result from loss of power in the laryngeal and the pharyngeal muscles respectively; and the former symptom gives great aid towards the diagnosis of doubtful cases. Hysterical aphonia is as common as hysterical aphasia is rare.

Not infrequently hysterical paralysis takes the shape of paraplegia or of hemiplegia. The former affection was described in a previous chapter (p. 642). The latter is less common; when present it more often affects the left side than the right; the proportion in Briquet's cases was as forty-six to fourteen. An important distinction between it and the hemiplegia due to an organic lesion of the brain is that in hysterical hemiplegia the side of the face and tongue remain unaffected. This was long ago remarked by Todd, who also pointed out that the patient in attempting to walk "drags the palsied limb after her, as if it were a piece of inanimate matter, and uses no art of circumduction nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks." Reynolds adds that a paralytic patient looks at her feet; an hysterical patient at the persons who are watching her.

Occasionally one limb is alone affected with hysterical paralysis; or, still more rarely, only part of a limb. The only muscle affected singly is the levator palpebræ; and hysterical ptosis is not uncommon.

Hysterical paralysis is often transitory, lasting only a few days or a few hours, though it may reappear in the same limbs or in others. But there are cases in which it persists for months or even years; and then it is often accompanied by rigidity (see next paragraph). The paralysis may disappear suddenly, under the influence of a sudden emotion or shock; or it may pass off very gradually. The removal of the patient to a hospital ward—away from sympathising friends and relations—is often followed by a more or less rapid recovery. The patient is told that she is expected to begin to move the paralysed limbs; day by day she is encouraged to do more; after a time she is taken out of bed, dressed, and put in a chair; and before long she walks as well as ever.

Contractions.—A remarkable effect of hysterical paralysis is a chronic spasm or rigidity of one or more of the limbs. Charcot published in his 'Leçons' several capital drawings illustrative of the distortions which it produces, and the French word *contracture* is often adopted for such cases. The affection may follow immediately upon a severe hysterical fit; and the paralysis and the rigidity then develop simultaneously. But in most cases, as in those of spastic paraplegia of organic origin, the contraction does not come on until there has been loss of power for a considerable time. In certain cases only one leg or one arm becomes contracted; but sometimes both legs are affected, sometimes both the arm and the leg on one side, sometimes three of the limbs, or even all four. When the affection assumes a hemiplegic type, and the rigidity follows the loss of power after some interval, it might be thought that there would be a difficulty in distinguishing it from the late rigidity of hemiplegia due to organic disease of the brain (p. 736); but in the hysterical affection the contraction as a rule comes on, not gradually, but suddenly, as the immediate result of a fresh hysterical fit. The arm is semi-flexed; and, according to Charcot, the leg always falls into a state of rigid extension, the adductors of the thighs are forcibly contracted, the knee and ankle straightened, and the toes stretched down to the utmost extent, while the sole is turned inwards; so that the condition of the foot resembles that of talipes equino-varus.

This condition is not without exceptions; Jolly relates a case in which one leg became powerfully flexed at the knee-joint; and in a marked case of hysterical paraplegia (which came under the writer's observation many years ago) the girl's legs were forcibly flexed at the knees and hips, so as to resist the utmost power that could be safely used to straighten them, until chloroform was administered. She afterwards recovered completely, and became an excellent nurse.

The rigidity in these cases is not relaxed during sleep; nor does it undergo any variations in degree at different times or periods of the day. The spasm is not confined to a single set of muscles, for one is not able to force the patient's arm into a position of complete flexion, any more than to extend it. By making the patient inhale chloroform, however, one can temporarily remove the spasm, so that the affected joints become perfectly supple.

The muscles remain well nourished, and retain their electro-contractility for a considerable time. But when a limb has been contracted for months or years without intermission, a little general wasting may occur, the reaction of the muscles to galvanism may be somewhat impaired, and in some cases it is said that the contractures no longer disappear under chloroform. The spastic paralysis of hysteria almost always ends in recovery; and in many cases this takes place abruptly. One of Charcot's is very instructive.

A woman was first attacked, at the age of thirty-four, with loss of consciousness after a moral shock; she then fell into the fire and burnt her face severely. After this she had several seizures, some of them hysterical in character, others rather epileptiform. Four years afterwards she had a very severe fit, attended with convulsions and followed by an apoplectiform stupor with stertorous breathing; this was at once followed by left hemiplegia. Rigidity of the left limbs set in abruptly after an interval. In the following year the right limbs also became contracted; and later still the jaws were fixed, so that an œsophageal tube had to be used. Her right arm, however, became free; and Charcot maintained that recovery was still possible. One evening, six years from the commencement of the contraction of the left arm and leg, she had an attack in which she imagined herself to be about to die. She cried out, became agitated, and with her right arm pushed aside those who held her. Her strong desire to get to the window for air was resisted; she became more and more angry; and first her right leg lost its rigidity, then her left leg, and finally her left arm. She got up and walked; and in eight hours her cure was complete. A slight crackling in the joints was all that remained of the complaint.

Such a case has an obvious bearing on mediæval stories of miraculous cures. Charcot quotes an article by Littré in which detailed accounts are given of certain persons who were cured at the end of the thirteenth century by pilgrimages to the tomb of St Denis, where the relics of Louis IX had been recently deposited. Three of these were young women who had been suddenly attacked with contraction of one leg or of the arm and leg on one side, and who also had anæsthesia. They were all cured suddenly, and in circumstances likely to act upon the imagination.

In some exceptional cases of hysterical "contracture" recovery never takes place. Charcot mentions several instances which he believed to be hopeless. One is that of a woman, aged fifty-five, whose legs had been contracted for sixteen years; under chloroform her knees could still be relaxed; but her feet remained in a condition of equino-varus. He made an autopsy in one case in which all four limbs had been contracted for ten years; and he discovered sclerosis of the lateral columns in nearly the whole length of the cord. The patient was a hysterical woman, and the rigidity passed off several times and returned again before it became permanent; so that there seems no reason to doubt that the case originally was only functional.

The points on which Charcot would lay stress as indicating an unfavourable prognosis in a case of long-standing contraction of a limb are that particular groups of muscles are wasted in an extreme degree and affected with fibrillary tremor like that seen in progressive muscular atrophy; that there is a great diminution in the contractility, as tested by faradisation; and that the rigidity remains to a considerable extent when the patient is under the influence of chloroform. On the other hand, he does not attach any importance to a little loss of substance in the affected limbs, or to a slight impairment of the electrical reaction of the muscles.

These exceptional cases of sclerosis of the lateral columns following a functional paralysis may be taken as indications that the most transitory physiological perversions of function are really accompanied by a transitory anatomical lesion. Again, they may inspire hope of ultimate recovery even when spastic hemiplegia or paraplegia is the result of demonstrable tissue changes.

Globus.—Among the most characteristic symptoms of hysteria is the so-called *globus hystericus*. It is difficult to say whether it is merely a morbid sensation, or depends upon spasmodic contraction in the œsophagus and pharynx, or on the sudden ascent of gas from the stomach. The former opinion was maintained by Eulenburg and Reynolds, the latter by Jolly. To the patient herself the globus is a sensation as if there were a ball or lump in the throat, which she cannot swallow or get rid of. She will sometimes put her finger into the pharynx, and make herself sick; or she will drink mouthful after mouthful of water to wash it down. The exact nature of the sensation varies. Some patients liken it to a bean sticking in the throat; others feel as if an animal were creeping about within the lump; while with others, again, it is rather a feeling of constriction about the neck as if the collar was too tightly fastened. The relation of such sensations to the state of emotional excitement which characterises hysteria is shown by the fact that a precisely similar sensation is felt by healthy persons when suddenly seized with grief or terror. Thus, children before a fit of crying experience a sensation of a lump in the throat which is probably identical with the hysterical globus. In some cases the lump is not merely felt in the throat, but seems to rise up from the chest, or from the abdomen.*

Flatulence.—Another frequent and probably allied symptom of hysteria is a rapid distension of the intestines with gas. The abdomen may suddenly swell up, so that the patient is obliged to take off her stays and undo all the fastenings round her waist. This form of tympanites seems to bear no relation to meals, and may return each morning before breakfast. The gas may ultimately escape either by the mouth or from the rectum. How such an enormous quantity of gas can be produced in the stomach and bowels is unknown. It is sometimes undoubtedly air which has been swallowed; sometimes probably gases, liberated from the food and expanded in accordance with physical laws by relaxation of the muscular walls of the stomach and bowels. The chemical character of the gas needs more exact determination, but it is believed to consist chiefly of carburetted hydrogen.

In some cases hysterical tympanites persists more or less for years. Bamberger alludes to a case of this kind, in which the patient passed through an attack of cholera without alteration of the size of the abdomen, but an abundant flow of saliva, coming on spontaneously, at once led to its temporary subsidence.

As Gowers remarks, the particular manifestation of Hysteria may often be determined by a real though trivial local ailment: as dyspnœa by bronchial catarrh, tympanites by flatulent dyspepsia, aphonia by a cold, spinal tenderness by enteric fever (as in Huchard's remarkable cases), blindness by amblyopia (as in a case we had lately in Mary Ward), and hysterical arthropathy by some slight injury to the knee.

The hysterical fit.—There remains to be described what was once regarded as the chief symptom of this disorder, the hysterical attack or "fit of the mother." In the majority of cases no such attacks occur during any part of their course; and when they do occur they present great variety of symptoms. Sometimes the patient has an unusually painful globus, and then begins to cry and sob, or breaks into violent laughter: and she may go on laughing and crying alternately, until she is exhausted. Such an attack, for which the vernacular name is now "hysterics" and was formerly "vapours," may occur singly, or may be repeated again and again. A nearly constant symptom is that when the fit passes off, the patient voids a large quantity of colourless urine, of low specific gravity.

In more severe cases a hysterical attack is accompanied by violent movements of the body and limbs. Often the patient screams, and then falls into a state of opisthotonos, supported for some minutes together on the back of her head and her heels. Or she may throw her limbs in all directions, beating them on the ground, or striking her own chest with her closed fists, tearing her hair, attacking those about her, and struggling to release herself from restraint. Or, again, she may thrust one arm high in the air: or forcibly bend her arm over the chest, and her thighs across one another.

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* So Lear:

> "Hysterica passio! down, thou climbing sorrow:
> Thine element's below."

some of the fingers and toes being at the same time stretched apart, while others are as strongly flexed. The respiration is quickened and noisy, but it is not stertorous. The features undergo the strangest contortions and grimaces; the eyes are generally fast closed; but the eyelids quiver, and the eyeballs are often turned upwards. It will be remembered that in an epileptic fit they are wide open, with pupils dilated, while in hysteria the eyes are shut and the pupils are of natural size. The more severe paroxysms approach those of epileptiform hysteria, miscalled hystero-epilepsy; but there is no real connection between hysteria and true epilepsy.

There appears to be little impairment of consciousness in these hysterical attacks. At the commencement the patient almost always knows what is happening, so that she is able to place herself out of the way of injury. Sometimes she keeps on screaming during the greater part of the fit, or shouts out "fire," "thieves," or "murder," or pours forth a continuous stream of foul language. Sometimes she has visual hallucinations, and raves at imaginary persons. Sometimes she seems to be quite unconscious; but by careful watching one may often make out that she really notices what is going on, casting occasional furtive glances at the bystanders from between the half-closed eyelids or changing her behaviour in accordance with their remarks.

Causes and nature of hysteria.—The very name of this disease involves the doctrine that it is the result of uterine disorder; and the ancient Greek writers—including Plato and Aristotle—actually supposed that the womb left its place in the pelvis and wandered about the body, setting up disturbances in different parts. From their time to the present there have never been wanting theories which have referred hysteria to a similar cause. The physicians who have held such views have generally been professed gynæcologists; but even Romberg, the great German authority on these disorders fifty years ago, defined hysteria as a "reflex neurosis proceeding from sexual irritation."

By different writers various disorders of the sexual system have been made the starting-point of hysteria. Some have endeavoured to trace it to unsatisfied desire, others have referred it to excessive venereal indulgence, and others again to the common menstrual disorders. Addison published a clinical lecture in 1830 to show that its phenomena depended upon "uterine irritation." By Dr Henry Bennett ulceration and induration of the cervix uteri were represented to be concerned in causing hysteria; and afterwards Dr Graily Hewitt assigned the principal place to flexions of the womb.

Charcot laid great stress on the frequency with which hyperæsthesia of the ovary is present. According to him the pain is sometimes very acute, and widely diffused over the abdominal surface, with special hyperæsthesia of the skin over an area two or three inches in diameter, corresponding with part of one iliac and of the hypogastric regions. In other cases no spontaneous pain manifests itself; indeed, the skin is anæsthetic, and the muscles can be pinched up without pain; but on making deep pressure towards the brim of the pelvis one can feel the ovary as an oval body, which slips beneath the fingers. Further pressure upon it gives rise, not exactly to pain, but to a peculiar sensation which the patient at once recognises, having felt it many times before as the starting-point of hysterical attacks; and this, passing up to the epigastrium, is followed by pain in that neighbourhood, and sometimes by nausea and vomiting. Next, if the pressure on the ovary be continued, the patient experiences palpitation of the heart with extreme rapidity of the pulse, a sensation of globus in the neck, a hissing noise in the ear of the same side, an impairment of vision, and then mental perturbation, ending in a regular hysterical fit. Charcot also believed that when there is hemianæsthesia, or paralysis, or contraction of the limbs on one side of the body, it is always the ovary on the same side which is hyperæsthetic; and that if such symptoms are bilateral, both ovaries are affected. Lastly, he found that when a patient is seized with a hysterical attack, even if epileptiform, it may be cut short by energetic compression of the ovary continued for three or four minutes.

Even among children, hysteria is far more common in girls than in boys. We have seen that this is the case with chorea likewise, and in both diseases the preponderance of cases in females is probably due to the same cause, their innate emotional susceptibility and want of power to resist external influences. Hysteria seldom shows itself in such women as would be called "masculine," whereas hysterical men and boys are those who are of a "feminine" disposition.

One influence which favours the development of the disease is want of a proper education. If a girl is morbidly self-conscious, she is too often allowed to indulge her vanity, and to nurse the belief that she is gifted and misunderstood. If she is studiously disposed, she is left to pore over books, without proper exercise in the open air. If she craves for precocious excitement, she is taken to balls and theatres when still young, she is permitted to sit up late at night, to lie late in the morning, and to feed her imagination with romances. The influence of example may also induce a liability to hysteria; the daughter of a hysterical mother is very likely to acquire the disease in her turn; so potent is this cause that there is no need to suppose that hysteria is transmitted by descent. In comparison with the emotional excitability in a girl, her intellectual capacity has little influence either in disposing her to hysteria or in guarding her against it. As regards bodily constitution, no doubt some hysterical women are robust, with fresh colour and a vigorous circulation, but by far the greater number are pale, weakly, and delicate.

The real relations between hysteria and affections of the ovaries and uterus are best elucidated and understood by the analogy afforded by some of the paroxysmal neuroses. We have seen that in many persons the attacks of megrim are constantly excited by disorders of the visual apparatus, but that in others this is not the case. We have found that vertigo is in some individuals brought on by diseases of the ear, and in some by diseases of the digestive organs, but that in others it is traceable to neither. So also it appears that hysteria does not always depend upon any one set of external exciting causes. In some cases it perhaps develops itself spontaneously. Very often anæmia or chlorosis may play an important part in its causation, and it has sometimes been observed as the direct result of deprivation of food. In other instances, probably in the majority, it is directly called forth by extraneous impressions on the nervous centres—not, as a rule, derived from the pelvic organs. Emotional influences—a sudden alarm, the shock caused by the death of a relative, or the unexpected loss of property—may bring on hysteria in a person who had before seemed perfectly healthy. A similar result may follow a gunshot wound (as has been observed by Weir Mitchell), or it may be brought about by a railway collision. In some cases a spirit of imitation suffices for its production; the disease has been known to spread like an epidemic from one woman to another among the inmates of a hospital or a workshop. In other instances, as observed by the late Dr Anstie, indulgence in alcohol seems to be the chief cause of hysteria.

Again, it often happens that several of the conditions which may bring forth manifestations of hysteria are present together. A person who is attacked after a sudden fright may before have had her health depressed by exhaustion of mind and body, or may have been labouring under religious excitement, jealousy, wounded self-love, or remorse. The liability to hysterical symptoms of all kinds is increased at the periods of menstruation, and in many women it is also augmented at the climacteric age.

No doubt there are many cases in which disorder of the generative organs is the main cause of the development of this neurosis, and it is practically wise to accept Addison's teaching that such disorder should be carefully searched for; and fortunately we can investigate the state of the ovaries by pressure above the brim of the pelvis without offence to a patient's delicacy. But writers are by no means agreed as to the frequency of ovarian irritation in hysterical women. Jolly disputes the accuracy of Charcot's statements, and quotes Scanzoni and Amann as having found among their hysterical patients as many as 19 or 20 per cent. in whom the pelvic organs were healthy. As he remarks, the fact that these observers were professed gynæcologists renders it certain that their figures in regard to this point are not too high. Speaking from his own impressions, he is disposed to state the proportion of hysterical patients free from these complaints at more than half. ('Ziemssen's Handbuch,' xii, 2, p. 461.)

We cannot accept Romberg's definition of hysteria, and it has led to much unjust suspicion, and to much ill-directed treatment. A right-minded woman is ashamed of giving way to hysteria, without our assuming a sexual origin for the disease. In the great majority of cases there is no such cause at work,—the disease is one of the nervous system and the mental condition. It occurs in girls under puberty, in men and boys; it is often cured by marriage, and still more often by maternity. Moreover, hysteria is very seldom present in males, at or after puberty, who are tainted by sexual vice; and it is said to be almost unknown among prostitutes.

Age and sex.—Hysteria is a feminine disorder, but there is no reason except arbitrary definition for refusing the name to similar symptoms in patients of the male sex (*infra*, p. 975). It is most common in girls between fifteen and twenty-five; or, as the same fact may be put, between puberty and marriage. It is infrequent in younger girls, and becomes less rare as they approach the period when menstruation begins. After marriage it often reappears during pregnancy, and still more frequently soon after childbirth; but, on the whole, it is less common than in unmarried women. About the climacteric, hysteria not unusually shows itself again. It is almost unknown before ten or eleven, and after fifty-five.

No race or country is exempt from hysteria, but it is far more common in Europe and America than among uncivilised nations, and among educated and delicate women than in those who have to work for their living. It appears to be more violent in its symptoms in France than in England or Germany. It is very rare in India, and Weir Mitchell states that it is far less common among the negro and aboriginal women than among the descendants of English colonists in America.

The vapours, with constant alacrity in swooning, were, to judge by the novels and letters of the Georgian period, far more common among the young women of the eighteenth century than at present. With more rational breeding, outdoor life, and active exercise, the girls of the Victorian reign are not only stronger and healthier than their grandmothers, but to a large extent have escaped from the tyranny of hysteria.

Diagnosis.—That this is often difficult must be sufficiently apparent, not only from what has been said in the present chapter, but from the repeated references to hysteria which have occurred in our discussion of organic diseases of the nervous system.* In all doubtful cases it is important to search for the indications of hysteria which have been described above. For example, anæsthesia limited to small portions of the cutaneous surface is probably present in many cases without the patient's knowledge, and its discovery may go far towards clearing up a doubtful case.

Hysteria not only mimics the symptoms of almost every disease of the nervous system, but also those of phthisis and of abdominal disorders. A woman, however, may be hysterical and yet be affected with organic disease of the brain or elsewhere. This is true even of cases in which all the symptoms of hysteria are present in the most marked form, still more if we include its more aberrant or doubtful manifestations. Sydenham went so far as to say that the majority of women were hysterical; and if the psychical characteristics of this neurosis are after all only an exaggeration of those which belong to the whole female sex, it is not surprising that no absolute boundary line can be drawn.

There is no proportion in the degree to which the different symptoms of hysteria are developed. A woman who has hemianæsthesia, or paralysis, or some local pain may give no indications of emotional susceptibility, may be quite free from uncomfortable sensations in her throat, and may never have had the slightest approach to a hysterical attack. Yet the whole course of the case may show conclusively that the complaint is of this nature. Indeed, when one has to deal with marked affections such as paralysis or contraction of a limb, if they are not due to organic disease, one is seldom wrong in making a diagnosis of hysteria, even without corroborative evidence.

So, again, one may usually attribute to the same cause all cases of hemianæsthesia if unaccompanied by motor paralysis, even when the loss of sensation is limited to a single limb. Sir William Gull, however, has left on record a case in which this rule misled him ('Guy's Hospital Reports,' Third Ser., vol. iv).

A nurse in the hospital, aged thirty-eight, complained of numbness in the left arm. Below the elbow sensation was completely wanting; but when the skin of different parts above that joint was carefully tested with the point of a needle there were all kinds of contradictions in her statements. She would one minute say that she could feel the needle at a certain spot, and the next minute that she could not feel it. Such observations led to the conclusion that the case was one either of feigning or of hysteria; but after the anæsthesia had existed for two years the limb became gradually paralysed. Two years later she died from the effects of an accident, and it was then found that the spinal membranes were thickened, especially in the cervical region, and that the posterior columns of the cord and the grey matter had undergone degeneration.

In practice we must distinguish not only between hysteria and organic disease, but also between it and malingering; for the two are far from the same. An hysterical girl is proverbially deceitful, but when cured of her hysteria she is cured of her deceit. A malingerer deceives others, but not himself, and his self-control is unimpaired.

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* See Dr Buzzard's valuable remarks on the "Simulation of Hysteria by Organic Disease of the Nervous System," 'Brain,' 1890, p. 1.

Prognosis.—The rule that hysterical fits are devoid of danger to the patient's life is not altogether without exceptions. Wunderlich met with a case in which a servant girl, aged nineteen, who had for eight weeks suffered from a succession of epileptiform fits of hysteria, unattended with fever, fell suddenly into a state of collapse, and died in two days with a temperature of 109·4°. Other cases have been recorded by Meyer in which excitement of a nymphomaniacal character was followed by cramps and spasms in the muscles, and led to collapse and death after an illness of some weeks.

Again, the more chronic forms of hysteria sometimes prove fatal by marasmus (cf. p. 971, *infra*). Wilks related in the 'Guy's Hospital Reports' for 1866 the case of a young lady who lost her sister rather suddenly by heart disease; whereupon she began to complain of palpitation, sickness, and pain over the heart, would take no food, declared that she had heart disease like her sister, and should soon follow her to the grave, and did actually die in a sort of hysterical convulsion exactly five weeks afterwards. He also gives the case of another girl who would eat nothing, but merely nibbled a biscuit and drank wine by drops. The bowels ceased to act, but masses of scybala were from time to time removed from the rectum. She became thin, and Wilks at length renounced the opinion that the complaint was merely hysterical, and thought that there must be some organic disease of the intestine. But when, at the end of more than two years, she died rather suddenly, the autopsy revealed nothing to account for her illness. Her body was then fatter than when he first saw her, and she had in fact taken rather more food towards the last. Of such cases the writer has only met with one, in which the patient, a girl of twenty-five, after long-continued and severe hysterical symptoms, died with extreme emaciation, and a careful autopsy showed the absence of any discoverable lesion.

Apart from these very exceptional cases, we may not only assure parents that there is no danger to life in the most severe case of hysteria, but that the patient will almost certainly recover completely. Nothing is more remarkable than the contrast between the patient and the convalescent. One of the gravest cases the writer has seen was in a girl who was only cured after prolonged seclusion under strict medical supervision. She is now again active, useful, and happy. The young woman who, as a cripple, was a burden to her mother, was transformed into a sensible and helpful sick-nurse. A third patient, who was called a maniac, and who twice attempted suicide, recovered, married, and is now the busy and efficient head of a large household.

Treatment.—When called to a patient who is actually in an hysterical attack, there are several methods by which we may succeed in cutting it short. One plan, suggested by the late Dr Hare, is to keep the patient from breathing for a minute or two by closing her nostrils and mouth; the vigorous inspiration which she makes, as soon as she is allowed, is often followed by relaxation of spasms and subsidence of the fit. Reynolds found this mode of treatment notably useful when an attack is so prolonged as to call for interference with the natural course of events. The old-fashioned treatment of a fit of hysterics was to "crumple up the patient's fingers," cut her stay-laces, and hold burnt feathers to her nose,—*i. e.* to let the fumes of ammonia produce a deep inspiration and strong sneezing. A rougher method, to the value of which many can testify, consisted in drawing the patient's head over the edge of the bed and pouring water upon it from a large jug; but giving the necessary orders to the patient's friends in her hearing was often enough to bring the fit to an end. An ancient hospital-sister in Esther Ward never failed to cut short an outburst of hysteria among her patients by threatening this homely treatment.

Compression of one ovary will sometimes arrest a hysterical fit, even of the epileptiform kind; Charcot speaks of one patient who learnt to apply the pressure herself. Another procedure, often adopted by the late Mr Stocker, consists in pressing upon the arteries and other structures on each side of the neck, in the same way as has already been described under the treatment of epilepsy (p. 927).

One must never forget that the patient is quite alive to all that is going on, although she may seem to be unconscious. A calm and quiet demeanour, the avoidance of all expressions of sympathy, and assurance of a speedy and harmless termination of the attack, may do much to shorten its duration; for if one is flurried or alarmed she is sure to notice it.

The earlier the age at which hysteria began to manifest itself, the more unfavourable is said to be the prognosis. It is therefore very important that a child who displays emotional excitability which seems likely to develop into hysteria should be placed under the most favourable moral and hygienic influences possible. Exercise in the open air, a plain nourishing diet, limitation of the hours of study, early rising, sea bathing, and healthy mental occupation are the best prophylactic measures; combined, in the case of children of over-anxious mothers, with what a wise and revered lady is said to have advised, "a little wholesome neglect."

When an hysterical patient is anæmic or chlorotic she should, of course, take one of the preparations of iron for a considerable time. In many cases it is advisable to prescribe an astringent vaginal injection, such as a compound alum lotion, containing a drachm of sulphate of zinc and a drachm of alum in half a pint of water.

In dealing with special symptoms one must never overlook the importance of treatment by removal from home (p. 960). This is often successful in the management of hysterical paraplegia—an affection which is apt to persist for months if left to itself. So, also, one can sometimes cure hysterical aphonia by confidently assuring the patient that she is able to speak. The writer has seen a case of this affection at once and completely cured by taking electric sparks from the larynx; or an interrupted current may be passed through the throat, or, in long-standing cases, galvanism may be applied directly to the vocal cords. Sometimes empl. lyttæ applied over the larynx has been successful when electricity had failed.

Paralysis of the limbs may be treated with advantage by faradisation, or by the application of a narrow piece of blistering plaster completely round the affected arm or leg.

Hysterical anæsthesia is sometimes benefited by the application of a faradic current by means of a dry metallic brush. Stimulating liniments may also be rubbed into the affected parts.

The remarkable effect on hysterical anæsthesia of the application of gold coins, metallic bracelets, and magnets, and even of pieces of wood and other indifferent objects, attracted much attention twenty years ago at the Salpêtrière Hospital (see the second Report by MM Charcot, Luys, and Dumontpallier to the Société de Biologie, 'Brit. Med. Journ.,' 1878, vol. ii, p. 548). It is difficult, if not impossible, to unravel the underlying facts from the tangled mass of hysterical perversions, physical and moral; but any good effect which "metallothérapie" may have by its action on the imagination of the patient is more than exceeded by the attendant evils (cf. Dr A. H. Bennett's case, 'Brit. Med. Journ.,' 1878, ii, 563; and Dr Donkin's paper, *ibid.*, p. 613).

At present metalloscopy and metallotherapy have receded whither the once famous "metallic tractors of Perkins" and the odic force of Reichenbach preceded them; and we hear more of "hypnotism," a revived mesmerism, and of "suggestion" in this induced hypnotic state.*

For rigid contraction of the limbs, the application of the continuous galvanic current is sometimes useful. But probably benefit more often results from straightening the affected joints under chloroform and placing the limb upon a splint, than from any other plan of treatment. Watson speaks of cases in which a stream of cold water directed upon the contracted part, and continued in spite of the patient's complaints of pain, led at once to the relaxation of the spasm. He also tells how Sir Charles Clark cured by the same method a girl who had hysterical trismus so that she could neither speak nor eat. He began to pour pitchers of water upon her face, but before he had emptied the second pitcher she began to scream and complain, with her mouth widely open.

When hysteria produces long-standing contraction of a limb, medicines are altogether useless; but for most of the other effects of the disease drugs are certainly useful. Most writers speak of assafœtida and valerian as owing their virtues chiefly to their disgusting taste; but many hysterical women actually relish assafœtida, and pills containing valerianate of zinc ought on this hypothesis to be almost inert, whereas there is no doubt that, in doses of gr. j—ij, they are often effectual in removing aphonia, hemianæsthesia, and hysterical hemiplegia. In 1874 a woman was in the clinical ward of Guy's Hospital who had paralysis of her left arm and leg, the latter being rigidly extended; she took large doses of assafœtida for some days without benefit, but was afterwards quickly cured by the valerianate of zinc, so that she walked out of the hospital within five days from the time when she first began to take it. In other cases equally good results have been obtained with five grains of assafœtida taken every three or four hours. Reynolds found bromide of potassium altogether ineffectual in any form of hysteria; and both he and Jolly recommend that opium or morphia should be given for the relief of pain and to procure sleep. But one must always keep before one's mind the evil results which may follow from the establishment of a morphia habit (see p. 951), and the continued use of chloral as a hypnotic is objectionable on precisely similar grounds. Yet perhaps more harm is done by alcohol than by any other drug. The late Dr Anstie was well justified in the strong protest he made against advising a hysterical woman to take a glass of wine or a teaspoonful of brandy, or even a dose of sal volatile to keep up her spirits whenever they are depressed, or to enable her to bear a frequently recurring pain. One should never sanction the employment of alcohol in any form by such patients, except perhaps a small quantity of malt liquor at meal-times.

The system of treatment introduced by Dr Weir Mitchell is of the greatest value in bad cases of hysteria, and particularly in Anorexia nervosa, and has undoubtedly saved many lives. It consists in separating the patient from sympathising friends, making her eat largely, and using massage to the limbs.

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* See the late Mr James Braid's excellent little book, 'Magic, Witchcraft, Animal Magnetism, Hypnotism, and Electro-biology' (1852); and Professor Heidenhain's 'Animal Magnetism,' translated by Dr Wooldridge, with a preface by Mr Romanes (1880). Mesmer was a Viennese physician who threw impressible patients into a *sommeil critique* by magetised water. The same thing is now done by means of a revolving kaleidoscope.

Maniacal excitement with hallucinations and delusions may occasionally complicate an hysterical attack. Such symptoms are, as a rule, transitory, and the patient becomes perfectly rational as soon as the fit passes off; but in certain cases they last for a considerable time, and then it may be difficult to distinguish between hysteria and insanity. Such doubtful cases find their way into lunatic asylums; and Dr Savage, in the twenty-first volume of the 'Guy's Hospital Reports' (1876), says that a large number of the female insane patients admitted into Bethlem Hospital have suffered from severe hysteria at former periods in their lives, and that many cases, at first regarded as examples of hysterical mania, end in death or dementia. Perhaps the best test of hysteria is that the mental faculties are found unenfeebled when recovery takes place; and thus many doubtful cases have at last been set down as hysterical because of the suddenness and completeness with which the patients have regained their senses.

Epileptiform hysteria.—In 1876 a girl was in Guy's Hospital whose main symptom was a rhythmical clonic spasm recurring at intervals of a few seconds in the neck, shoulders, and arms; she also had transitory fits in which she seemed to lose consciousness, while the jerking motions became intensified and succeeded one another with increased frequency.

In other cases of hysteria, fits occur which still more closely resemble those of epilepsy. The loss of consciousness is apparently complete; the eyes are widely open, the pupils dilated; the tongue is said to be sometimes bitten; the movements are rhythmical, clonic spasms, repeated again and again. Such cases have received the special designation of "hystero-epilepsy;" and there have been many discussions as to whether they are transitional links between the two neuroses, or instances of their co-existence in the same patient. But these attacks are proved not to be true epilepsy by their not recurring and ending in dementia, by their much longer duration and by their greater violence. Moreover, as Charcot himself observed, this so-called hystero-epilepsy does not, however closely the attacks may follow one another, entail danger to life, with the development of a high temperature, as in the status epilepticus (p. 918). He relates the case of a woman who for more than two months laboured under a constant repetition of epileptiform fits. Once they succeeded one another without a break from 9 a.m. to 8 p.m.; and then again from 9 p.m. onwards for an equal space of time. He estimates that she had from 150 to 200 attacks on that one day alone. As he remarks, if this patient had been suffering from epilepsy, she would soon have succumbed. The woman never passed urine nor fæces involuntarily, and got out to relieve herself in the brief intervals that occurred between the attacks. Her temperature rose occasionally for a short time to 101·3°; but the mean was 98·6°. As further distinctions between hystero-epilepsy and epilepsy Charcot observed that the former never assumes the type of the *petit mal*: and lastly, that, however perfectly epileptiform a fit may be, it can always be arrested by compression of the ovary.*

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* The extraordinary gestures of these unhappy patients have been carefully studied and named, as phases of "Ecstasie," of "Clownisme," and so on. See a report of a visit

Professor Mendel showed the Medical Society of Berlin in 1887 an almost unique and apparently typical case of epileptiform hysteria in the person of a man fifty years old.

Anorexia nervosa vel hysterica is a remarkable condition which was first described by Sir William Gull ('Clin. Soc. Trans.,' vol. vii). It is attended with extreme wasting, and has often been taken as the result of some latent tubercular affection. But the emaciation is actually too great for this diagnosis; since patients with organic disease seldom become so thin as this until they are no longer able to get about, whereas it is characteristic of hysterical anorexia that those affected with it display an excessive restlessness and bodily activity. In this complaint the pulse and the respiration are slow, and the temperature is generally below normal. Sir William Gull's patients were chiefly young women between the ages of sixteen and twenty-three. It does not appear that any of them showed definite indications of hysteria. Some of his cases terminated fatally, but in others a complete recovery took place, the main points in their successful management being the avoidance of fatigue, the administration of nourishing food at very frequent intervals, and the use of warm clothing.

It is in these cases that Dr Weir Mitchell's treatment by seclusion, with abundant feeding and regular shampooing or massage, is most successful.

The writer once saw a fatal case of this form of hysteria, with Dr Dabbs, of Newport. Extreme emaciation preceded death. At the autopsy there was confirmation of suspected vaginal irritation, but the organs were like those of starvation. The cord was normal to the eye, and histological examination revealed no lesion.

Catalepsy.—In some patients a hysterical attack is attended with singular symptoms, which have long been regarded as belonging to a special neurosis named catalepsy (κατάληψις = a seizure or arrest). Its characteristic features are that the patient, although deprived of sensibility and of voluntary motion, remains fixed in whatever position she occupied at the commencement of the fit. Yet her muscles offer no such resistance to external force as would prevent the limbs from being easily bent or extended by another person, or the body from being placed in any posture.

The condition of a cataleptic patient may be compared with that of a lay figure, such as artists use; if she is sitting up her arms can be put at the most awkward angles with the trunk, and will remain without falling, at least for a time; if she is recumbent her spine and pelvis may be bent so as to form an obtuse angle with the thighs, and will retain that posture. The name of *flexibilitas cerea* is sometimes given to this peculiar state of the muscles; they have been found by Rosenthal to have their electric sensibility and contractility either normal or increased. It is, however, a mistake to suppose that during the cataleptic state the muscles are capable of resisting the force of gravity for an indefinite length of time. On the contrary, the limbs, if extended, slowly fall again to the side; and Dr T. K. Chambers quotes a case in which an impostor was detected by attaching a weight to the extended hands. She supported it without moving, and ultimately she confessed the fraud.

The eyelids of a cataleptic patient may either be widely open or shut;

to Salpêtrière by Dr Gamgee ('Brit. Med. Journ.,' Oct. 12th, 1878); and details of cases in the same number, p. 561, with Professor Charcot's lecture, *ibid.*, p. 789. Some of the attitudes are shown to coincide remarkably with those of demoniacs in mediæval paintings.

in the latter case, if opened, they very slowly close again. The pupils may contract under the influence of light. After recovery, all memory of what occurred is said to be lost.

Catalepsy probably never occurs except in persons who either are obviously hysterical or at least disposed thereto. A case of the minor form of epilepsy was quoted above (p. 917), in which it assumed a cataleptic character; but fully developed catalepsy does not appear ever to accompany epilepsy in a man, or in a woman if she has no hysterical tendencies. The attacks seem almost always to be traceable, at least in the first instance, to a fright or to some other powerful emotion. Dr Chambers mentions the case of a girl, a patient in St Mary's Hospital at Paddington, who when Covent Garden Theatre was burnt down was awakened by the light flashing into the ward through the uncurtained windows; on the following morning she was attacked with catalepsy. The most striking instances of catalepsy which stand recorded are to be found in medical works of a time when the modern extended conception of hysteria was not thought of, and yet the reports of these cases often contain clear proof that the patients were in the highest degree hysterical. The celebrated history related by Dr John Jebb nearly a century ago is a case in point.

> The subject of it was a young lady who suffered from hysterical risings in the throat and flatulence, and who was highly susceptible to every change in the weather. Although she was prepared for Dr Jebb's visit when he first went to see her, she was seized with the disorder as soon as his arrival was announced. "She was employed in netting, and was passing the needle through the mesh, in which position she immediately became rigid, exhibiting in a very pleasing form the figure of death-like sleep. . . . The positions of her fingers, hands, and arms were altered with difficulty, but they preserved every form of flexure they acquired, nor were the muscles of the neck exempted from this law, her head maintaining every situation in which the hand could place it, as firmly as her limbs. . . . About half an hour after, the rigidity and statue-like appearance being yet unaltered, she sang three plaintive songs in a tone of voice elegantly expressive, and with affecting modulation." The seizures sometimes lasted as long as five hours.

Trance.—Similar attacks of much longer duration have received a different name. The condition of the muscles is not the *flexibilitas cerea* of catalepsy; either the limbs resist attempts to change their posture, or they are completely flaccid, falling into any position whatever by their mere weight. Such patients are said to be in a state of trance. It is stated on good authority that this condition may last for days and weeks, the face perfectly pale, the breathing so feeble as to be almost imperceptible, even the heart's pulsations and the pulse at the wrist scarcely to be felt. These are the cases which have led to the popular belief that death is sometimes only apparent, and that there may be a danger of persons being buried alive; and it cannot be denied that a patient in such a condition might easily be allowed to die by careless or ignorant attendants, or might be buried before death. The complete cessation of breathing and of pulse, the absolute coldness of the whole surface, the collapse of the cornea, and the absence of the conjunctival reflex are usually sufficient evidences of somatic death; but in a case lately seen by the writer, so possessed were the patient's friends with the danger of premature interment that they were not satisfied to let the body be placed in the coffin until the absolutely unmistakable sign appeared of incipient decomposition.

Sleep-walking.—Other curious nervous conditions are known as somnambulism, ecstasy, and hypnotism. The somnambulist, without seeming to wake from a state of sleep, walks all over the house and even out of doors. She balances herself without difficulty on narrow planks, and manages to avoid all obstacles in her path. She takes no notice of anyone whom she may meet, and if with much difficulty awakened, she is bewildered, and has no recollection of what she has been doing. Marvellous stories are told of the feats which are accomplished by persons in this strange condition, which has been happily compared to an acted dream.

Sleep-walking is most often seen between the age of five or six and that of twenty, and is more common in girls than in boys. In the case of children under puberty, the habit often seems due to over-excitement by studies or by games, and sometimes is accompanied by nightmare and other effects of indigestion.

The state of ecstasy is one in which a person becomes regardless of all external circumstances and engrossed with some particular emotion or idea. She remains motionless, with staring eyes and fixed expression, or she may repeat a few words with ceaseless monotony. After she returns to consciousness she remembers the vision she beheld in the state of day-dreaming.

A similar state can be produced by mesmerism (or " hypnotism " or " electro-biology "), and is called by the practitioners at Nancy in 1890, *somnambulisme provoqué.* When this condition frequently recurs we have as a result what is called double consciousness, of which marvellous examples are from time to time published. A careful investigation of such cases might, amid much credulity and imposture, possibly discover some facts of physiological interest. A typical example of this curious condition of "double consciousness" is given by Dr Proust, in the 'Revue de l'hypnotisme' for March, 1890.

Chorea major.—Mental excitement naturally finds vent in muscular action, and rhythmical movements increase and exalt the emotion which gives them birth. Religious dancing was common in antiquity, and survives in the minuet before the altar, which may still be seen in Seville Cathedral, and in the solemn gyrations of dancing dervishes at Constantinople.

In the Middle Ages there were epidemics of Dancing Mania, of which an interesting account will be found in 'Hecker's Epidemics of the Middle Ages' (p. 87 of Dr B. G. Babington's translation for the Sydenham Society). This Chorea major was also known as the Dance of St John the Baptist,* as the Dance of St Vitus (cf. p. 880), and in Italy as the Tarantula. It spread over Germany and the Netherlands in 1374, reappeared at Strassburg in 1418, and was not extinct till the seventeenth century.

Pearce, a traveller in Abyssinia, early in the present century, gave an account of a very similar dancing mania in that country, which was called " Tigretier," and Dr Davidson has described an epidemic among the natives in Madagascar which closely answers to the account of Chorea major in the Middle Ages. Similar cases in Scotland are described by Sir Walter Scott in his 'Demonology and Witchcraft,' and find a parallel in the religious dances of the " Jumpers " in the United States.

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* Possibly the connection with this saint was through the dancing of the daughter of Herodias, but more probably it was because the old pagan festival of dancing and leaping through the fire took place on St John's Day at Midsummer. St Vitus was the patron and help in need of those afflicted with dancing mania, as St Martin in smallpox, and St Anthony in erysipelas.

A remarkable instance of this saltatory mania was recorded by Mr Kinder Wood ('Med.-Chir. Trans.,' vii): and still more extraordinary movements were performed by a patient of Dr Abercrombie, who had suffered for two years from various nervous affections.

Mr Wood's patient, a young married woman, who had suffered severely with pain in one side of the face, began to be troubled with involuntary movements in the eyelids, which were opened and shut with excessive rapidity. After a time the hands were beaten rapidly upon the thighs, and the feet upon the ground. Then she became half raised from her chair and seated down again, these movements succeeding one another as quickly as they possibly could. Other modifications of her attacks occurred from time to time, until at last she took to skipping about the room, regulating her movements by a series of strokes on the furniture as she passed, or by movements of her lips, as though beating a tune. Someone thinking he recognised the air as "The Protestant Boys" began to sing it, and she suddenly turned and danced up to him, and continued dancing until she was out of breath. Then a drum and fife were procured, and she immediately danced up as close to the former as possible, and went on till she lost the step, or until the measure was changed, or was made so rapid that she could not keep up with it. It was presently found that a continual roll of the drum put an end to her movements; and thus the attacks were at length prevented. The explanation which the patient gave was that there was always a tune dwelling on her mind which at times irresistibly compelled her to begin the involuntary motions.

Abercrombie's patient, while lying perfectly quiet, would suddenly with her whole body make a kind of convulsive spring, by which she was jerked entirely out of bed. Or, if sitting or lying on the floor, she would fling herself into bed, or leap, as a fish might, on to a wardrobe fully five feet high. After a time the muscles of the back and neck became affected with a wonderful semi-rotatory movement, which sometimes went on without interruption night and day for weeks together. If the head or neck was touched, the motion was increased to extraordinary rapidity. She was cupped, and the affection suddenly ceased with a convulsive start of the whole body; but it returned again and again, and finally disappeared only when constipation and menstrual disorder were corrected.

Dr Fagge recorded a similar case which occurred some years ago at Guy's Hospital. A little girl, aged nine, had been knocked down by a boy five days before her admission; she was insensible at the time, and seven hours afterwards she had a fit. Subsequently she had nine other fits. They began by her making a low sighing or moaning sound, after which the upper extremity became contracted, the teeth were clenched, and with a sudden bound she threw herself completely out of bed. When she recovered consciousness she had no remembrance of what had occurred. On the day of her admission she had fourteen fits, and at one time she remained insensible for two hours. A day or two later she had two fits during the visit of the physician, Dr Owen Rees; in these she clapped her hands, and her face went through a most extraordinary series of contortions. In one attack she struck a part of her head to which a blister had been applied, whereupon she at once became conscious. After she had been in the wards six days one of the fits was commencing, when the nurse told her that she must be tied down: she immediately began to cry and recovered, saying that she would have no more fits. From that time she remained well.

For cases in which vertical movements of the arm are incessantly repeated, the special name of *malleation* has been invented. The writer once saw a boy of fourteen or fifteen who continued this action all day long. Sir William Gull, who was consulted on the case, recommended a flogging in the patient's hearing, and the malady was cured without the prescription being carried out.

Hysterical movements of the head or the body round and round have been dignified with the title of *rotation* or *gyration*. Several instances are quoted by Watson, of which the most striking, related by Dr Watt ('Med.-Chir. Trans.,' vol. v), occurred in Scotland in 1813.

A girl, aged ten, who had before exhibited other nervous symptoms, was seized with a propensity to turn round on her feet like a top, with great velocity, and always in one direction. This continued a month, and then passed off. Afterwards she began to roll over and over on her bed, moving rapidly from one end to the other, for six or seven hours every day. Having been taken into a garden she quickly rolled along the whole length of a gravel walk, and even when she was laid in the shallow part of a river she began to turn round as usual, until she seemed on the point of being drowned. She made little or no use of her arms in performing the rotations. After another month she began an entirely new set of movements. Lying on her back she would, by drawing her head and her heels nearly together, bend herself like a bow, then she would relax her muscles, and fall with considerable force on her seat. This she repeated ten or twelve times a minute for several hours daily. After a fresh interval she began to stand on her head; and then to let herself fall again, her knees striking the bed first. This movement also she continued for fifteen hours a day, at the rate of twelve or fifteen times a minute. Blistering, purging, and leeching were employed without result; but having been taken to Glasgow from her home in the country in an open chaise, and brought back again after three days, she was seized with diarrhœa, and soon afterwards recovered entirely.

Hysteria in male subjects.—As above stated, this remarkable neurosis or combination of neuroses is, in the great majority of cases, a disorder of the sensations, emotions, will, and bodily functions of women—particularly incident to the periods of youth and of the menopause, but possible at any time after the infant has developed into the girl. Nevertheless undoubted cases of hysteria occur in the male sex, just as undoubted cases of hypochondriasis occur in women. It is extremely rare in full-grown men, but is not very uncommon in boys from the age of eight or ten to puberty, and, less frequently, from that period till two or three and twenty.

In Wilks's lectures on 'Nervous Diseases' there are several cases recorded. In one the principal symptom was so-called laryngismus, in another aphonia, in another convulsions which had been ascribed to spinal meningitis, in a fourth the "malleation" or hysterical hammering referred to above. Sometimes the boy can be thrown into spasms like those of tetanus or of a frog poisoned with strychnia, as in a patient of Mr Holden's at St Bartholomew's Hospital, who was cured by removing a fatty tumour.

Dr Rühle, of Bonn, relates the case of a spoilt boy of fourteen, who had violent convulsive attacks ending with vomiting, and was cured by "removal from home, cold shower-baths, and the fear of the rod" ('German Clinical Lectures,' p. 449).

The writer was once consulted about a schoolboy, who had twice been laid up at home on account of "hæmorrhage from the bowels." He was ruddy and well nourished, and no disease of any organ could be discovered. A specimen of "melæna" was at last procured, and proved on examination to contain no blood, but a silver salt from a solution that he had used in photography. He then became violent and threatened to kill himself, but was soon cured by the wholesome discipline of a public school.

A naval surgeon gave a graphic relation in the 'Lancet' a few years ago, of a cabin boy of sixteen, who completely lost his voice and appeared to suffer from attacks of choking and suffocation. He was carefully treated for several days, until a sudden and unexpected pinch made him swear loudly, and thus revealed the imposture.

In 1888 we had in Philip Ward an apparently healthy young countryman, twenty years old, who, beside dyspepsia and hypochondriasis, suffered from "fits" of a decided neurotic character with well-marked globus hystericus. As with many hysterical girls, he had a good character and was anxious to get well.

The treatment for hysteria in males is the same as that above advised for females. It is, however, more usual for the element of malingering to be part of the complaint. Some of the cases just mentioned were treated and cured rather as faults than misfortunes, and this is often justified by the result. But in such cases it is well for the physician to use medical discipline such as blisters or the application of faradism with a dry brush, with a curative and not a penal intention.

In 1891 there was under the writer's care a tall, rather pale, but intelligent boy of twelve or thirteen, who is quick at his tasks, fond of healthy games, and to all appearance truthful and ingenuous. As the supposed effect of a slight blow on the head he gradually showed weakness in the legs, staggering, and then complete inability to walk. There were no cerebral symptoms and no pelvic symptoms, the reflexes were normal, and he could move all his limbs when lying in bed. We concluded that the paraplegia was functional or neurotic, assured him he would be cured with electricity, and after three or four applications of an induced current of moderate force he perfectly recovered.

HYPOCHONDRIASIS.*—From hysteria we pass to hypochondriasis by what seems a natural transition, for these two neuroses resemble each other in being essentially selfish and introspective, and also in their tendency to simulate organic diseases of various parts of the body. Indeed, some writers believe that hypochondriasis, which chiefly occurs in men, is in the male sex the representative of hysteria in the female; and the uncertainty which still exists as to the relative frequency of hysteria in men and boys arises mainly from the fact that cases which would be at once set down as hysterical, if they occurred in women, are often wrongly attributed to hypochondriasis because the patients are of the other sex.

But hypochondriasis may be seen in women, especially about the time of the menopause, without any admixture of the proper characters of hysteria; and we have quoted several characteristic cases of pure hysteria in men and boys. Moreover, there is an essential difference between the two diseases. As we have seen, the mental state in hysteria is characterised by exaltation of the emotions, and diminished power of the will to control the feelings. But in hypochondriasis the morbid sensations which the patient experiences are not dependent upon any external cause; they are created by the mind itself. Thus Romberg was not without justification when he called it the antithesis of hysteria.

Again, hypochondriasis is unattended with any of those nervous vagaries—convulsive attacks, paralysis, hyperæsthesia, anæsthesia—which are so characteristic of hysteria. Its predominant symptom is always of one kind; the patient believes, without cause, that he is the subject of serious bodily disease. Before long he becomes conscious of a pain in some particular region of his body, upon which he at once concentrates all his attention; and he is now a "hypochondriac."

The disease is one which has retained the same appellation since the days of Hippocrates and Galen. But they conceived the actual seat of the disease to lie in the viscera which are situated immediately below the diaphragm; and a similar hypothesis was intended to be conveyed by the equivalent English expression, "the spleen."† Indeed, it is the fact that dyspepsia is often present; the bowels are constipated, and gas is apt to accumulate, with flatulent belchings and noisy rumblings in the abdomen.

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[*] Ὑποχονδριακὸν πάθος—Passio hypochondriaca—Passio atrabiliosa—Melancholia (in part)—Humor atrabiliosus—Milzsucht—The spleen.

[†] The first to place hypochondriasis among nervous diseases was the celebrated anatomist Thomas Willis.

In all probability the starting-point of hypochondriasis is often real constipation with flatulent dyspepsia. Affections of the stomach and "liver" lead, as we shall find, to depression of spirits and irritability of temper. But what is peculiar to the complaint with which we are now concerned is the marked exaggeration of every local pain. Moreover, the painful region seldom, if ever, remains the same during the whole progress of the case. After having long dwelt upon a gnawing or burning pain at the epigastrium, which he felt sure must indicate cancer of the stomach, the patient at once loses this, and perhaps begins to suffer from symptoms which point to the throat. Or he is attacked with dyspnœa and palpitation of the heart, and feels convinced that he has serious cardiac disorder; or a slight cold leads to a cough, and he begins to collect the sputa, is certain that he is phthisical, and consults the physicians who have a reputation for curing pulmonary affections; or he finds himself giddy, experiences a sensation of weight and pressure in the head, and forthwith thinks of nothing but of the apoplectic fit which he believes to be impending. Perhaps the most miserable of all hypochondriacs are those who refer their sufferings to the genital organs. To this class belong most of those who complain of spermatorrhœa or impotence. They have dragging pains in their testes, which always hang too low; the urine is turbid, and they have emissions at night, during defæcation, and at other times. One sees at once that their whole attention and thoughts are concentrated upon the sexual function; and to make matters worse, they are sure to have studied vile pamphlets, written for the very purpose of stimulating their apprehensions, and of inducing them to seek relief from the writers.

Hypochondriasis is often hereditary. As to whether the disease tends to pass into insanity in the same individual there is much difference of opinion. Melancholia is the form which bears the closest superficial resemblance to it; but Romberg remarks that this is characterised by a tendency to self-negation, whereas in hypochondriasis the whole attention is concentrated upon personal feelings and sensations; and he goes on to say that one suffering from melancholia treats his medical attendant as an enemy, and avoids him; but the hypochondriac looks up to him as his guardian and saviour. He is constantly on the search for new plans of treatment, and expects that each fresh one will cure him. As Romberg puts it, "the more physicians, the better he is satisfied; he likes to change them as often as he would change a poultice." Yet, in spite of all, he is not unhopeful, and is never weary of life. Hypochondriacs do not commit suicide, and any doubtful case in which such an attempt is made may be safely set down as one of actual insanity. Probably the same may be said of every case in which the patient has distinct delusions, as (for example) that he is made of glass, or has a barrel organ in his belly, or that he has swallowed spiders. Griesinger, though so great an authority on mental disorders, seems to have entirely mistaken the boundary line between hypochondriasis and melancholia; the cases which he relates as examples of the former are almost all of them really instances of the latter disease (cf. p. 987). This perhaps explains how he found hypochondriasis "extraordinarily frequent in young people." Gull and Anstie express the general opinion when they say that it is scarcely ever seen before puberty, and very rarely makes its first appearance after fifty.

Prognosis.—A point which must always be kept in mind is that a person labouring under well-marked hypochondriasis may also have organic disease of the stomach, for example, or of the liver, or an aneurysm of the aorta. Many a patient has had his complaints made light of until the signs of one or other of these diseases have become too manifest to be overlooked, or until he has died suddenly, from rupture of a large vessel, or from angina pectoris.

In weighing beforehand the probable success of treatment in a case of hypochondriasis, one has to take into consideration not so much the apparent severity of the symptoms as the length of time they have lasted and the circumstances in which they began. The younger the patient the better the prospect of cure: particularly if the family history is good. It may be said that although cases occur in which it is as difficult to separate hypochondriasis from melancholia as to distinguish hysteria from mania, yet neither of these slighter affections are really premonitory of insanity. Occasional coincidence in the patient or in a family only shows that they do not exclude it. The most marked case of pure hypochondriasis the writer has had to treat began in a young man who is now so no longer, who is still subject to grievous fits of depression and irresolution, introspection and remorse, but who has justified the prognosis given when he married, that he would often cause anxiety but would never go mad.

The *treatment* of this disease is difficult and unsatisfactory. One should never try to make light of the patient's sufferings, but should show oneself to be really interested and anxious to relieve them. Active exercise out of doors should be taken daily, and mental occupation should be provided. Sea-bathing, the cold water cure, the warm baths at Gastein, may sometimes be prescribed with advantage; but the benefit is due to change of air and scene, and alteration in the habits of life. The digestive organs must be carefully watched. The continual exhibition of laxatives is injurious: it is better to depend on diet and exercise to procure a healthy state of the bowels. But an occasional dose of calomel and Epsom salts is valuable in many cases, and may be repeated once or even twice a week. Valerian is said to be sometimes useful, and so, with some patients, is tincture of sumbul. Neither quinine, strychnia, nor phosphorus is generally of service, nor iron, unless there be marked anæmia. Anstie rightly laid stress on the importance of not prescribing alcohol for hypochondriacal patients, even in the form of medicinal tinctures, lest it should lead to habits of intemperance.

Sydney Smith's prescription for hypochondriasis was rational and manly religion, large fires, and the society of those who think well of us. To this may be added open air and active exercise—turning, gardening, riding, and such country pursuits. "Throw but a stone, the giant dies," is Cowley's dictum, and he knew the malady better than its cure. As to drugs, we may be allowed for once to borrow from Hahnemann, and prescribe blue pills to cure the blue devils.

PSYCHOSES

OR

DISORDERS AFFECTING THE MIND

(*By G. H. SAVAGE, M.D.*)

> "Mentem sanari, corpus ut ægrum,
> Cernimus et flecti medicinâ posse videmus."—LUCRETIUS.

Insanity as (1) *the expression of disease of the brain ;* (2) *an expression by the nervous system of bodily disease ;* (3) *disorder of functions of brain—Classification—Causation.*

Acute delirious mania—Acute and other forms of ordinary Mania—Melancholia with bodily complaints (*Hypochondriasis*) *; with mental complaints* (*true Melancholia*)*—Stupor, active and passive—Delusional insanity—Hallucinations of the senses—States of mental weakness—by defect—by instability. Specially named varieties of insanity : puerperal, alcoholic, moral, syphilitic, gouty, plumbic, febrile, phthisical, asthmatic, cardiac, renal and diabetic—Epileptic insanity—Paralytic and tabic insanity.*

Testamentary capacity—Certificates and other medico-legal points.

Idiocy and imbecility.

IN approaching the study of insanity, it is first of all important to remember that we have not a definite disease, such as typhoid fever or phthisis, to investigate, but that the condition or state called insanity depends, not only upon the evident symptoms, but upon their bearing on the social environment of the individual. There is, in fact, no absolute standard of sanity, and the divisions of the condition called insanity are to a great extent arbitrary. Insanity will have to be here considered chiefly from its clinical or medical aspect, but yet its social and its legal aspects must not be entirely neglected. Though there is no standard of sanity, yet it is found that disorder of the nervous system expresses itself along more or less definite lines, and we shall have to point out the fairly regular association of symptoms which occur in the different groups of insanity.

First we must recognise that while some cases of insanity depend upon cerebral disease or degeneration, others depend upon diseases of the body, which are referred to the nervous system ; so that there is in the latter cases a mental aspect to a bodily disease. Thirdly, we shall have to consider the most difficult of all cases—those in which there is disorder of function without any visible pathological change in the nervous system. So, then, we have (1) disease of brain with disorder of its function; (2) disorder of some function or functions of the body with insane or nervous interpretations; and (3) disturbance or disorder among the functions of the brain leading to social unfitness.

While recognising *forms* of insanity, we must remember that these forms merge insensibly into one another, that no symptoms are absolutely characteristic of any one form of mental disorder. The *forms* are, then, arbitrary, but more or less convenient, groupings of symptoms. In considering the forms of insanity, it will only here be necessary to make the most characteristic divisions, such as the following:

Melancholic states, in which there are mental and bodily weakness and excessive and painful self-consciousness.

Maniacal states, in which there is weakness marked by loss of control.

Dementia, in which there is weakness with more or less evident defect in the mental functions.

It is necessary also to refer to cases which are associated with disorders of the senses such as hallucinations and illusions, which are placed together under "Delusional Insanity."

The above are the more common groups into which symptoms of insanity form themselves. But these symptoms may occur in two distinctly opposite pathological states. They may occur as symptoms of disease of the brain, as seen in the degenerative process called "general paralysis of the insane," or they may occur in what we describe as the second and third groups of insanity. In fact, mental disorder, whether it occurs with brain disease, or bodily disease, or only as a disorder of mental function, has similar symptoms. Just as one may meet with cough which is due to disease of the lungs, cough which is due to a spasmodic condition, as in asthma, and cough due to defects in cardiac circulation, so we may have delusion due to organic disease of the brain, to bodily disease, as bad supply of blood, or to disorder of function, as seen in false sensory impressions.

Insanity occurs among the civilised and the uncivilised, but in its most marked and serious forms it is specially a disease of the more highly cultivated, and I shall point out that the most fatal forms of brain decay increase directly in proportion to the nervous strain and modes of life met with in city-dwellers.

Insanity is steadily increasing in England and Wales. We see this from the statistics of the Commissioners in Lunacy. In 1875 there were per 10,000 of the population, 24·75 males and 28·43 females reported as insane; in 1885, they were 26·61 males and 30·80 females; and the 1897 report gives 30·04 and 33·84 respectively.

The proportion of insane to sane in 1859 was 1 to 536, in 1897 it was 1 to 313. Finally taking the admissions of fresh cases, in 1875 these were per 10,000, 5·36 men and 5·18 women; in 1897 the numbers were 6·02 and 6·04 respectively.

Though, therefore, it is evident that there is a steady increase in the numbers of the insane in England, yet it is evident that, as this increase occurs chiefly among the poorer classes, there is not much ground for alarm. for the insane being better cared for live longer, the struggle for making a living being more keen, more who are helpless are sent to asylums who in former times would have been kept at home; and there seems to be with knowledge a greater belief in asylum treatment. Hence much of the increase in numbers is due to the insane being more frequently placed under control.

The brain and nervous system, like the lungs, stomach, and other viscera, have their special ways of expressing disease; the mental symptom may be similar though the causes may differ.

We must look for the symptoms of insanity in changes due to defect or disorder of the factors of mind.

The factors of mind, for our present purpose, will be considered to be, first of all and perhaps most essential, the inherited nervous system, the nervous basis which is added to by the experience gained through the senses—special and common—the resulting perceptions being bound together and arranged by memory, the resultant being mind, which reacts according to will and emotions.

In every case of insanity it is well to examine carefully into the inheritance, then into the working of each factor of mind, the whole being tested by the reaction of the person to his surroundings.

In considering the so-called *causes* of insanity, the student must be warned that it is extremely rare to find a single cause producing an attack of mental disorder, and it is therefore better to speak of *conditions* than *causes* of mental disorder. Though it is perfectly true that certain vital conditions, such as race, stage of civilisation, climate, and the like, deserve to be considered as conditions predisposing to insanity, yet, in a short chapter such as the present, it is beyond our scope to consider more than a few of the general conditions which give rise to mental disorder. The conditions may be divided for convenience into those acting chiefly upon the mind, and those acting chiefly through the body—the so-called *moral* and *physical* causes. These again can be divided into *predisposing* and *exciting;* and though some of these act distinctly as moral causes, yet it will be found that in the majority of cases one cause may be both moral and physical, and may also act as a predisposing and as an exciting cause. Take, for example, alcoholic stimulants, which act primarily as a physical and predisposing cause; but the associations of the drunkard are likely to lead him into fresh troubles, which again react, and a fresh outbreak of drink and excitement may be the real exciting cause of the outbreak of insanity. The most common *moral* causes are mental anxiety, worry, overwork, money losses, religious excitement, love affairs, domestic trials, fright, and occasionally joyful emotions. Among the *physical* causes, intemperance, organic disease of the brain, injury to the head, venereal excesses, masturbation, epilepsy, pregnancy, parturition, and lactation are more common causes assigned in England, and in addition to these, one of the most important conditions of insanity is *heredity.*

It is extremely difficult to be sure of the part played by inheritance, yet it is sufficiently certain that a large proportion of people are insane because of the insanity of their parents. The insanity which is passed on from parent to child has special peculiarities, which will have to be referred to more in detail later on. There seems in such cases to be liability to disorder of function rather than to disease of the brain itself, and there also appears a greater predisposition to recurrence in cases with strong neurotic inheritance. Besides direct inheritance we have to remember that insanity is often developed in families where epilepsy, hysteria, hypochondriasis, some spasmodic nervous affections, and the like, occur; and it is probable that if to these conditions in one parent be added any special cause of organic degeneration in the other, the offspring will be rendered more unstable, that is, more liable to fall out of step with the other members of a civilised society.

Causes of insanity act directly, *i. e.* immediately, only in very rare instances. The mischief is much more commonly the result of frequently recurring disturbance; and therefore in considering any case of insanity with reference to causation, it is important that changes in temper, disposition, and habit should be carefully investigated. It must be understood that true causes in very many cases cannot be traced, and in many instances the supposed *causes*, such as sleeplessness and dyspepsia and even alcoholism, are really the earlier *symptoms* of the disease.

It is necessary to refer to a few of the general conditions in more detail. In cities degenerative neuroses and general paralysis are most common, whereas the latter is almost unknown in peasant races, such as the Celtic population in Ireland, Wales, or Scotland. Education is a cause of insanity if ill suited to the nature of the person, but over-education alone is very rarely the cause. More women than men become insane, but more men have general paralysis and more women recover from insanity. Puerperal and climacteric conditions affect women. Any form of insanity may attack patients of any age, but in childhood the tendency is to idiocy, in early youth to imbecility, in youth to weak and impulsive mental states; in middle life the greatest amount of acute insanity occurs, as well as most general paralysis, at the climacteric melancholia and delusional insanity are frequent, and as age increases the tendency is to dementia.

Solitude and sedentary occupations followed apart are very dangerous conditions; the isolation of widowhood is often dangerous. Domestic worry chiefly affects women, and business worry and anxiety men. Love affairs and sudden shocks, whether painful or pleasant, affect mostly women and very young people. All causes producing excessive nervous exhaustion affect the young most; as do also the abuse of stimulants and chronic poisoning, such as that by lead.

Injuries are not frequent causes of active insanity, but lead to degenerative changes. Tumours and coarse lesions of the brain, syphilis excepted, are also rare causes. Religious and political excitement act mostly as exciting causes. Fevers and any causes of delirium may start insanity. Influenza has been particularly injurious.

Bodily diseases causing pain or weakness may set up nervous disorder. Insanity may depend on bodily disease, or on physical disfigurement and its moral effects. Insanity may alternate with other nervous diseases, such as epilepsy or hysteria, or it may alternate or replace other nervous or bodily diseases, such as asthma, gout, rheumatism, eczema.

ACUTE MANIA.—By acute mania we mean a state of mental weakness with marked loss of self-control. Under this head we place a variety of disorders extending from delirium to simple hysteria, and it will be convenient to make two distinct groups of the maniacal cases.

There is a well-recognised and well-understood condition—delirium; and formerly it was insisted that there were essential differences between mania and delirium, but it is certain that there are connecting links. There are cases in which there is some delirium but much maniacal excitement, and others in which there is little maniacal excitement and more delirium. It is well, then, to remember that when speaking of patients suffering from mania, we may have to do either with those in whom there is great bodily and mental disturbance, or those in whom there is a great mental disturbance with little or no bodily disease.

Acute delirious mania is a term used to express the symptoms occurring in the cases of mania in which the delirious element is most marked. Typhomania, brain fever, and other terms have been used, but it seems that we still are in want of some general descriptive term which will include all the cases which deserve to be grouped together, because, though most of these delirious cases are more or less maniacal, yet there are examples in which all the other bodily symptoms are present, but instead of mania there is either melancholia or partial weak-mindedness, or at least mental confusion. Acute delirious mania is a disease most commonly met with in young people of both sexes. It is common in the highly nervous, the accomplished and educated. It is generally ushered in by a slow process of change in temper, or by general feelings of uneasiness, restlessness, malaise, or hypochondriacal symptoms. It is common to meet with it in young women who have had some disappointment in love, some shock, or who have suffered from some physical disorder, producing nervous weakness or anxiety; they complain of sleeplessness and headache, loss of appetite, amenorrhœa, and general inability to apply themselves to ordinary work. This may be the early stage of acute delirious mania. Acute delirious mania may follow shock, fevers, such as scarlet fever, measles, and the like; poisoning, as from belladonna, from intemperance in alcohol or ether, and in my experience it may also follow the inhalation of an anæsthetic. It is not confined to young people. The period of depression may be followed quite suddenly by one of excitement, exhibited by voluble talking, exaltation of ideas, or not uncommonly by a rather sudden and startling development of erotic excitement. Within a few days the delirium has reached its height: the patient then has the appearance of one suffering from typhoid fever, lying on her back with a tongue dry and brown, or thickly coated, with sordes on the lips, the eyes suffused, the cheeks either sallow or (during periods of excitement) brightly flushed, the lips moving without any audible sound, the hands twitching or picking the bedclothes, the voice, if heard, harsh and raucous, the skin moist, often with slight increase of temperature; then rapid emaciation, tendency to bedsores, and excitement recurring irregularly, but most marked at night, with little or no reaction to the surroundings. Hallucinations of sight are specially common. There may be periods of tranquillity and apparent recovery followed by others of excitement. There seems to be no memory of the period of excitement. Patients recovering have little or no recollection of what has happened during their illness. It is almost impossible to say how long the period of excitement will last, but from a few weeks to two or three months is the limit, and it must be remembered that patients suffering from acute delirious mania are passing through a process in which exhaustion is the chief characteristic, and the excitement is invariably followed by a more or less prolonged period of depression, the depression not being that of melancholia, but rather that of inability to will or to desire, so that they are in a will-less, childish condition. From one third to one fourth of the young cases suffering from acute delirious mania die, and of the others probably a full third remain permanently weak-minded. Some rapidly gain flesh, but remain dull, apathetic, and unlike their former selves. In others delirious mania is a stage of general paralysis.

The chief points in *treatment* are to feed often and abundantly. It it is well to begin with a free purge by means of calomel or croton oil, and then to follow up with beef-tea, mutton broth, milk, brandy, and eggs. It is almost certain that artificial feeding will be required. Feeding by means of a nasal tube—or better, by putting a funnel in one nostril, and, while controlling the other, slowly pouring the fluid food down the nostril—will be found of great service in these cases. The feeding by a stomach-pump, by the rectum, or by the nose, must be repeated every three or four hours, and it is of the utmost importance that stimulants should be given without stint. In old days it was feared that with such apparent excitement of the brain, alcohol would have an injurious effect, but modern experience shows that stimulants, instead of causing excitement in these patients, are more likely to produce rest. In many cases the excitement is so extreme that some form of restraint, medical or mechanical, must be used. Chloral hydrate, in doses of from fifteen to thirty grains at night, and repeated in ten-grain doses two or three times a day if necessary, may be tried, but it is better to do without any narcotic if possible. In strong, young, active people the wet pack, *i. e.* the rolling the naked patient in a sheet wrunk out of tepid water, and then rolling one or two blankets outside this sheet, and placing the mummified patient on the bed for from one to three hours, will produce rest and quiet, but this treatment should be carefully watched by a skilled nurse, and during the process food and stimulant should be given. In some cases, in the earlier stages, warm baths with mustard in them will produce a stimulating effect upon the skin and secure a good night's rest. The great point to remember is that patients suffering from acute delirious mania are suffering from a disease which, as a rule, either rapidly kills or rapidly passes off, leaving the patient much exhausted, and as soon as the acute attack has passed it is of the utmost importance that they should have careful nursing, without much change in their surroundings. After the illness rest and quiet in the country or by the seaside is preferable, in the writer's opinion, to travel; and it should be remembered that in such cases intellectual work should not be undertaken for a twelvemonth from the onset of the disease.

Complete or only partial recovery may take place; the patient may be left more or less morally or intellectually crippled, and in some cases not only is there defect of mind, but, following the acute delirium, some more or less permanent wasting with contraction of the lower extremities may occur, and the writer has met with cases of delirious mania in which general paralysis has followed and made rapid progress.

There is no very marked tendency after complete recovery to the recurrence of attacks of delirious mania. In fact, delirious mania differs in that as it does in other respects from many forms of ordinary acute mania.

Ordinary Acute Mania.—In this condition, unlike the last, we have more organised expression of delusion, and more marked loss of self-control with less bodily disturbance. The forms under which maniacal excitement appears are endless, depending much upon the inheritance, education, race, and other peculiarities of the individual. We meet with every variety, from simple uncontrolled lust to violent homicidal destructiveness, and no one description can fully cover the whole class. Mania may occur as only part of a morbid mental process; it may follow a melancholic stage, or epilepsy; it may be part of *folie circulaire*, or of recurrent attacks of insanity; it may be one of the symptoms in general paralysis of the insane or of dementia due to age or definite brain disease. But in all cases the symptoms are sufficiently alike to allow one description.

The *causes of mania* are in no way special. Mania may follow from moral or physical causes, may follow injury to the brain, direct or indirect, or may follow upon bodily disease or disorder. A shock may produce either mania, melancholia, or dementia.

Nearly all cases of mania begin with uneasy feelings about the epigastrium, occasionally with feelings either of emptiness or fulness of the head, rarely with headache or photophobia. Sleeplessness and fear of impending ruin are common, anorexia, constipation, restlessness, inability to apply themselves to work, irritability, and emotional weakness. This period may last from a few weeks to several months, and may become extremely marked and profound. After the period of depression the patient may almost suddenly grow more restless, more excited, and markedly loquacious. He may say that he has been ill, but that now he is quite well, and he may become extravagant, boisterous, and over-generous. He may discover that he does not require to sleep so much or to eat so much as he did, and he may be full of ideas that he will be able to make a fortune by some invention. Any interference by his friends is resented, and in many cases such early interference precipitates the attack of mental excitement. Sleeplessness of a restless type is marked, appetite is variable, a craving for stimulants may occur. The bowels as a rule are confined; the skin is often sallow, but of normal temperature; hallucinations of sight and hearing are not uncommon in the earlier stage; delusions as to property, wealth, position, and also as to the actions of friends, are common. Will as a rule is unstable, so that the patient is incapable of persistent effort. He is often emotional and irritable, but his memory is so far good that he remembers what is said or done, though things appear to him different from what they would in health. Sexual desire is frequently great, but there is probably no increase of sexual power. The general bodily condition is one of weakness, and the general mental condition is one of instability and mobility.

The two things that one has to guard against chiefly are the reduction of strength and the infliction of injury to self and others; the real question of treatment depends almost entirely upon the question of danger to the patient or his friends. Mania may end fatally from sheer exhaustion; this occurs mostly in elderly patients, or in people exhausted from some general bodily disease. A large number of maniacal cases get well, but a certain number remain either permanently weak-minded or permanently unstable, so that they are constantly liable to recurring attacks of insanity. Of those who do not sink in the earlier stage the period of excitement may last for weeks or months; probably four or five months is an average period for acute maniacal excitement to persist. During this period there will almost certainly be intervals during which the patient is more quiet and apparently improving, and it is noteworthy that after a night's sleep, produced either by narcotics or as a result of exhaustion, the patient may appear to be improving; but we must be prepared for remissions and relapses in the course of ordinary acute mania. During the maniacal excitement destruction of clothes and filthiness of habits are very common and distressing. Each individual case exhibits peculiarities in its course, symptoms, and termination. Patients often suffer from some bodily ailment before the mental storm begins to pass; one will complain of headache, another of neuralgia, one of rheumatism, and another of restless sleepless misery connected with some trifling bodily ailment before the disappearance of the excitement. As a rule the symptoms disappear slowly, it being noticed that the patient is getting more self-control, has greater desire to see friends or relations, writes letters home, is anxious to see his business, and very probably may be discontented with all restrictions. Discontent in persons suffering from mania is equivalent to the returning consciousness of a man who has been stunned; and just as in the latter case there is for the patient himself a painful consciousness, while for the onlooker there is the satisfaction of sensibility returning, so also discontent in the maniacal patient means returning self-consciousness. In proportion as the patient has been excited will there be depression or mental exhaustion; and it is pretty certain that a prolonged attack of mania will be followed by a prolonged attack of mental exhaustion or depression, during which there may be more or less sleeplessness and loss of appetite, with general mental weakness; but in these cases change of surroundings and as speedily as possible a return home will be found the best treatment.

From a practical point of view, all powerful narcotics and depressants are merely means to keep a patient within control, and not means of really curing our insane patients. It will be necessary in some cases to try chloral, bromide of potassium and the like, and it undoubtedly in a few cases will be found that bromide of potassium alone or combined with chloral will lessen the excitement and give time for the patient to recover. But the most important consideration is that, whatever treatment is followed, we must be sure not to affect the appetite or we shall have cause to regret it. Abundant nourishment, with or without stimulants, associated with exercise in the fresh air and as much freedom as is possible, should be the treatment for such cases. Sulphonal is useful and appears to be harmless. Paraldehyde in drachm doses repeated every one or two hours till six doses have been given, is useful in many violent patients. Trional acts more rapidly than Sulphonal, leaving no after-effects, but its effect is less lasting. Hydrobromate of Hyoscine given *sub cute* in one hundredth of grain doses is very useful in very emotional and noisy patients, but should not be repeated more than thrice in one day.

Before leaving the consideration of this group, we must mention the patients who have suffered from acute mania and who never permanently recover. Some, as already stated, remain ever after liable to recurrence of insanity, others remain weakened in some special way so that they are unable to fulfil their duties as before. Thus, one individual will be morally weak and is called either a kleptomaniac or a drunkard, another becomes emotionally weak and takes to ambitious schemes for reforming his fellows or to spending day and night in religious exercises, a third becomes volitionally weak and allows himself to be looked after and attended to by his friends without any desire to help himself.

It is only necessary to add that, if the antecedent stage of melancholy be long, the maniacal one is likely to be prolonged also.

After epilepsy very violent mania is frequent, and chloral given by the mouth or the rectum may check the mania if administered early after the fit. Cases in which the mania depends on senile changes are less favourable but not hopeless. In such cases chloralamide is often useful.

Maniacal excitement due to general paralysis generally passes off in a few weeks. Mania due to alcohol is very variable, but may last for weeks or months.

MELANCHOLIA.—By the term *melancholia* we mean a state of unreasonable mental depression; grief without a real cause, depending rather upon a physical than a moral determinant.

Melancholia may be but part of the disordered process or may be the whole of the morbid state. In most cases of acute mania and in most cases of general paralysis of the insane there is a stage of melancholia which ushers in the acute symptoms. Melancholy as a form of mental disorder occurs in all ages, though probably it is most commonly met with in and after middle age. It is most common in the dark complexioned. The symptoms of melancholia may depend on general depression of the vital powers, or it may depend upon some special diseased process, as, for instance, heart disease. The most characteristic symptom at the onset is extreme self-consciousness. This develops into grief which is variously expressed; passing into irritability of temper, sleeplessness, loss of appetite and suicidal tendencies. Melancholia has been divided into many varieties. The chief methods of division depend upon the outward expression of the morbid grief. In some the symptoms are *active* and in others they are *passive*,—in the former the patient restlessly wringing his hands and openly complaining, while in the latter he is struck dumb by his misery. Melancholia is divided not only by its general but by its special aspects; in one the explanation of grief is in relationship to the bodily functions, while in the other the mental symptoms predominate. The former may be called *hypochondriacal melancholia*, and the latter *true melancholia*. Every shade of melancholia may be seen in relationship to hypochondriasis, but in an asylum we see only extreme cases which must be secluded in consequence of their desire for death or refusal to take food.

Hypochondriacal melancholy.—There are four distinct groups of cases which come under this head. First, those patients whose one complaint is that they are dying and that nothing can be done for them, though they may make no definite complaint of any special disease.

Next we have a large group of cases in which the symptoms are referred to the *head*. Thus young patients will tell you that their brains are hot or wasting or hollowed or melted, and women about the climacteric will complain of opening and shutting of their brains, of "something coming away" when they move their necks, or the like. Some older patients complain of adhesions between the brain and the skull, or of miraculous removal of brain and nerve power.

The third group includes those who believe that some terrible calamity has befallen their *digestive tract*. One patient believes that his throat is stopped: these symptoms are allied to exaggerated hysterical globus. Another is sure that his stomach is ruptured or imperforate, or that some onnection exists between the stomach and the circulatory system. A few patients imagine that the rectum is permanently closed. All these cases have to be looked upon as exaggerations of hypochondriasis, cases in which every hypochondriacal symptom is greatly developed.

The last group of insane hypochondriacs contains those who believe that some trouble arises from their *reproductive organs*. Middle-aged men believe that they have been rendered impotent, that their testes have withered, or that by some evil habit or by some course of medicine they have become emasculated. Sexual hypochondriacs are more common among men than among women. It is comparatively rare in asylums to find women with uterine and ovarian hypochondriasis. There are many young sexual hypochondriacs whose disorder is partly due to masturbation, and these have varying disorders of the brain.

Beyond the above groups there are endless varieties; in fact, any part of the body may have morbid sensations giving rise to hypochondriacal interpretations.

In all cases of hypochondriasis the one object of *treatment* must be the withdrawal of the attention as much as possible from the sensitive part by occupation, while the general health is being improved. Judicious change of air, change of scene, travelling and mechanical occupation, with withdrawal from old associations and companions, are of the utmost value. Such cases recover in a fair proportion, but if past middle age, or if the development of morbid ideas has been slow, and if there be no definite physical illness associated with the mental disorder, the prognosis is bad. Such patients rarely become weak-minded, but persistently hold to their hypochondriacal delusions, which may remain for many years, the rest of the intellectual life of the patient being normal.

Ordinary melancholy.—Ordinary melancholia consists in emotional depression, in which the explanation of the feelings is moral and not physical. It may be the result of shock, or grief, or bodily illness. In certain predisposed persons any cause which reduces the vital standard below a certain point seems to be able to produce melancholia. The first symptoms are, as a rule, mere uneasiness, restlessness with inability to take interest in surroundings, tendency to tears and emotional disturbance, dread of some impending calamity, with undue sensitiveness to the conduct and the remarks of others. These symptoms are associated with loss of appetite, sleeplessness, and feeble circulation, as seen in winter by chilblains and cold extremities, together with loss of appetite, constipation, and amenorrhœa in women. These evidences of reduced power are all part of the process, and may rapidly pass into most pronounced mental depression. Up to this time there has been a feeling of misery, but there has probably been little or no explanation as to its cause. Later hallucinations of the senses or delusions may arise, and these depend to a great extent upon the education, age, and immediate surroundings of the individuals, so that the young woman thinks that her virtue is called into question, while the old man thinks he is going to the workhouse. It is common to hear patients in this condition refer to their past wickedness, and to say their souls are lost, that they are possessed by the devil, that they are "the Scarlet Woman," that they are metamorphosed into beasts, or are unnatural, and that they ought to be dead. At this time they will be sleepless, and probably will refuse to take food voluntarily. There will be complete inaction of the bowels and general apathy and listlessness; suicidal tendencies become marked, and it is important to remember that patients as a rule select a special form of suicide which they prefer, and will wait for an opportunity rather than seek death by any other means. Suicidal attempts are mostly to be feared when patients believe themselves to be impotent, when they believe themselves to be injuring their nearest relations, or when they dread being tortured "to make them confess," or when they believe themselves to be followed and dogged about. Patients who "hear voices" are also very liable to suicide. The melancholic process is, as a rule, longer than the maniacal one, and an average of from six to eight months is required for recovery. The symptoms, after slowly advancing, may slowly recede, the prognosis depending really upon the steadiness of the improvement both in mind and body. In some the cure is sudden, after some marked change in general health, such as return of normal sleep, digestion, or menstruation. Perfect recovery may take place, or there may be a slight excess of excitement after the melancholia has passed away. There is a great tendency to relapse in melancholic patients, especially in those belonging to an insane stock. A certain number of patients die from melancholia, but as a rule the cause of death is secondary to the mental disease, there being some congestion of the lung or extreme exhaustion and emaciation depending upon bad nutrition or insufficient feeding. In cases of chronic melancholia wasting of the brain is well marked, and the pia mater is sodden and easily separable.

The *treatment* of melancholia naturally divides itself into preventing harm and doing good. The first essential is to prevent suicide, and this is only to be done by constant watching, by the utmost care in the selection of nurses, and by insisting on the patient having his rooms on the ground-floor. Rest in bed, especially during the colder months, is valuable; warmth and food, with stimulants, being very important. In some cases of active melancholia morphia is very useful. When patients are treated at home, it is necessary to give narcotics, the rule being not to give them continuously, to avoid giving them in increasing doses, and to vary them as much as possible.

The next most important thing is the method of feeding. Patients who refuse their food absolutely may be fed by the rectum, by the mouth, or by the nose. As a rule, patients who resist should not be fed by enemata. In feeding by the mouth, if sufficient care and time be taken in nearly all cases food may be given in small quantities by the spoon being introduced by the side of the teeth. But if resistance be great it is better to pass a nose-tube or the stomach-pump. The nose-tube is very easily passed, and nurses can be trained in a few lessons to do it without danger; a large-sized soft catheter fixed on the end of a small glass funnel is all that is required, the tube being passed down one nostril, while the other nostril is compressed by the finger. Three to five pints of milk, six eggs, two pints of beef-tea or broth, and four ounces of brandy, may be given in twenty-four hours.

Among the forms of melancholia the most important are *simple melancholia*, that is, melancholy without delusions; *melancholy with stupor*, to which we shall refer again; and *active* or *restive melancholia*, in which an active expression of grief occurs. There is no special form of melancholia deserving the terms "religious" or "suicidal."

Simple melancholia is often present in men of middle age and of active intellectual habits. Misery without cause, self-accusation about trifles, sleeplessness and loss of appetite, inability to apply the mind or the attention, are the chief symptoms.

A month's rest under supervision, away from wife and friends, followed by restful and recreative travel, are all that is needed; but probably twelve months' rest from mental labour will be beneficial. This state may occur in much-examined youth, in much-worked or worried manhood, or may appear at the climacteric or decline of life. Suicide is common. There are other varieties of melancholia associated with bodily disease. Thus, undeveloped or suppressed gout may be represented by melancholia, and phthisis may show itself only as a progressive weakness and refusal to take food, with suicidal impulses. Melancholia, if of an active type, often benefits if morphia be given, and in young cases shower-baths are of service. In gouty and simple cases a course of Turkish baths will often work wonders.

STUPOR, INCLUDING ACUTE PRIMARY DEMENTIA AND MELANCHOLIA WITH STUPOR.—Under this one head two very distinct groups of cases are included, but in both of these the characteristic symptoms are the stupor, the silence, and the abstracted appearance of the patients. According to some there is no such thing as acute primary dementia without delusions, all these patients owing their mental attitude to some dread or terror. In fact they believe such patients to be in a kind of chronic panic, but it will in any case be necessary to distinguish between two groups occurring under this head, and for convenience we will call them the active and passive groups.

Active stupor.	*Passive stupor.*
Common in adolescents, who have a terrified expression, are wasted and thin, with skin purple and cold, they resist or are cataleptic, sleepless, wet, and dirty, have distinct delusions of dread, retain memory through the attack, refuse all food and require abundant forced feeding with stimulants; electricity and massage may be useful.	The passive form occurs more frequently as the result of some cause of exhaustion in young or middle-aged people, who have a silly aspect, muscles are flabby, skin greasy, limbs relaxed; they eat what is given them, sleep well, are clean if watched; there is no evidence of the existence of delusions, little or no memory on recovery; and for treatment they require baths, exercise, and general tonics.

The above groups resemble one another in history and in progress to a great extent; most of them occur in young adolescents. It seems as if in certain weak-minded persons there is energy enough for boyhood or girlhood, but when manhood and womanhood comes on it fails. With such cases shock, grief, masturbation, disappointment, overwork, unhealthy occupations, fevers, intemperance, rapid childbearing, or the like, may suffice to produce the breakdown. When once the condition of stupor has been established, as a rule it persists for several months, though occasionally there are recurrences of stupor between intervals of sanity. Little or no good comes to these cases unless the general health can be markedly improved. The patient, if in active stupor, is probably more or less cataleptic, and has to be forcibly fed. He wastes, and is wet and dirty, is very liable to secondary inflammations, and often has chilblains. He takes no notice of his friends. The stage of stupor is of very variable duration, and may end in death, in slow or sudden recovery, or in partial weakness of mind.

The state of mere passive stupor may be uniform and persistent, but commonly it is associated with periods of temporary recovery or of periods of excitement, or with sudden outbreaks of impulsive violence. This passive state frequently results from or follows an acute stage of mania, or develops after some severe and exhausting bodily illness.

Feeding, warmth, and watching are of great service. One danger in such cases is death from some secondary complication; local inflammation or phthisis may develop and terminate rapidly. After recovery there is frequently a period of exaltation during which sexual desire and tendencies to vice and intemperance may appear. The prognosis depends greatly upon the family history. If neuroses are common in the family, and if the patient is very young or badly developed, or if he have a badly shaped head, or any tendency to phthisis, the prognosis becomes absolutely bad. If a patient recover from stupor it is very necessary that for some years he should be carefully watched, and he should not return to any occupation which involves severe strain either of mind or body, so that it is much better after recovery from stupor to send the patient for one or more sea voyages, or, if possible, to let him follow some mechanical or outdoor life.

Delusional Insanity.—So far we have considered states in which there have been perversions of the whole mental functions associated with more or less bodily disturbance, but under this head we have to consider cases in which the perversion of the senses is the chief symptom. The expression "out of one's senses" is recognised as meaning madness, but only some patients are definitely out of their senses. Such are the cases now to be described. It can readily be understood that as our intellect depends to a great extent upon the impressions we receive and store up from our senses, if our senses mislead us the intellectual result will differ greatly from that of the ordinary standard. The cases which I have to describe depend chiefly upon hallucinations, which are sense impressions not depending upon any external or objective impressions. Thus a person who in the stillness of the night hears "*voices*" has hallucinations of hearing, and the person who, similarly situated, has *visions* or smells *poisons* or tastes *filth* is suffering from hallucinations of the senses of sight, smell, and taste respectively. Hallucinations of hearing are the most common, next in order we have hallucinations of sight, then in order those of touch, taste, and smell. Nearly all hallucinations may be reckoned as painful nervous impressions. The explanation which is given of the sensation depends to a great extent upon the education and occupation of the individual.

Patients suffering from *delusional insanity* are generally past middle life, single or widowed, often of insane family, leading solitary lives or filling positions which are uncongenial to them in one way or another. Hallucinations have been frequently traced to solitary confinement in prison, and I have met with several cases in which they have developed in men living isolated lives in the wilder parts of India and other English dependencies. As a rule the earliest symptom complained of is nervousness and nervous weakness. Patients become excessively sensitive, and this leads to a condition in which everything seems to point to or to affect the individual; his mind is like the tender corn to the walker, and just as every movement seems to affect the latter, so every other action of others seems to be directed against the former. He becomes suspicious, jealous, bad-tempered, more and more solitary, and this increasing solitude further develops the ideas of *suspicion*. He believes himself to be *persecuted* and *followed about*. He may think that his thoughts are read by others or appear aloud to himself and to others so that they read his secrets, he hears people coughing, jeering, or making allusions to him or to his family. He may hear words such as "devil," or worse, constantly whispered in his ears; he may then take the law into his own hands, and assault some one whom he believes to be his persecutor. The ideas of interference may become more organised, so that he is convinced that bodies of men, such as the Jesuits or the Freemasons, are interested in his ruin. Under these circumstances the patient becomes exceedingly dangerous, and, unfortunately, it is very hard to break through the hallucinations and to appeal to him by means of any other ordinary sense impressions which might in time loosen the bands of the false sense impressions. If these symptoms have developed slowly for twelve months the prospect of recovery is very slight. Patients who believe themselves to be followed or persecuted should be most carefully secluded or watched with the utmost care, as they are both homicidal and suicidal. They are sometimes so exceedingly suspicious that they will deny their delusions so as to mislead those of whom they are afraid. Besides those who believe that everything refers to them, are others who believe that the whole of their sense impressions are "symbols"—that the world is a kind of play which is being acted before them.

The general *treatment* of cases suffering from delusional insanity must depend very greatly upon the nature of the delusions, and the length of time which they have existed. Medical treatment is practically useless, but in some cases persuasion, associated with change of surroundings, works wonders. One young man believed that his relatives jeered at him from the roof, and that others made use of foul terms against him and them. This patient was excessively violent; yet by employing him, amusing him, and bringing distinct irrefutable evidence that these people did not exist on the roof and did not accuse his friends of iniquity, we slowly brought him to be more amenable to discipline, and at the end of twelve months he was discharged so far recovered that he has since emigrated and is now in perfect health.

CHRONIC WEAK-MINDEDNESS.—Loss of faculties, more or less general, may result in such extreme mental weakness that it is unsafe for the patient to be at large, as he would on the one hand be a tool for the wicked, and on the other a prey to the vicious. Chronic weak-mindedness, as seen in an asylum, may result from age, apoplexy, or it may follow nervous exhaustion, general paralysis, brain tumour, epilepsy, or any other conditions of nervous or mental excitement.

Simple *weak-mindedness* is *gauged* by *loss of memory* and *loss of control*, and every variety may be seen, from the weak-minded imbecile to the lustful and dangerous, demented lunatic. The general course of ordinary weak-mindedness due to age and the like is as follows:—Progressive loss of control of the emotions, loss of memory and tendency to collect articles of no value on the one hand, while there is thoughtless and reckless extravagance on the other, a redevelopment of lust and a tendency to intemperance, all which lead to a wastful expenditure of the reduced nervous capital. Often patients who have led reputable lives up to sixty may pass through every phase of moral degradation before it is discovered that their immorality was the result of arterial degeneration. Such cases require to be recognised and to be rather nursed at home than sent to asylums, if the means are sufficient, because the probable termination of such cases is an apoplectic fit or permanent childishness.

Among the subjects of chronic weak-mindedness we must include a large number of patients who, having suffered from acute attacks of mania or melancholia or stupor, instead of recovering their old mental balance, remain either *permanently crippled* or *permanently unstable*. The permanently crippled may exhibit their weakness by loss of one or more of the higher faculties, so that one becomes impulsive and homicidal, another ceases to respect truth and honesty and is called a kleptomaniac, another disregards decency and becomes altogether brutalised. In some a single faculty, such as memory, may seem to be affected out of all proportion to the others. The most characteristic loss after all is that of the highest social relationships: patients who have not perfectly recovered from acute attacks of insanity generally lose their affection for near relations and friends, and pass into a condition of philosophical calm or social indifference. Besides the loss of faculty already described, there are cases of permanent instability. Every asylum contains patients who are subject to recurrent attacks of mania or melancholia, patients who during twenty, thirty, or forty years periodically have recurrent attacks resembling in every particular the previous ones, with the probable exception that each attack is rather longer than the former, and leaves some more or less well-marked scar upon the intellect. Among these we have to recognise cases of chronic recurrent mania and of chronic recurrent melancholia.

Another group remains, that of those cases, who, after an acute attack of insanity, have a few symptoms surviving, organised as it were, so that the patient becomes automatic, with certain peculiar actions or modes of thought fixed. Thus one patient for sixteen years sobbed out hour by hour, "I don't know what to do;" and yet there was no sorrow in the later years, the lamentations having become purely automatic, and the patient eating well, sleeping well, and becoming fat. In other cases with chronic chattering incoherence, there may be persistence of some hallucinations, so that the patient is living in two mental worlds, or perhaps it were better to say between them; hearing faintly the real messages from the one, and more clearly the false impressions from the other. Many such cases, though violent at first, may be treated with sufficient safety at home, as is evidenced by the existence in so many country places of feeble-minded villagers, ready to run an errand or enter into boyish games. Such weak-minded persons may be trained to perform mechanical tasks sufficiently well, so that the simple drudges of an asylum are often patients suffering from chronic feebleness of intellect.

So much for the permanently crippled. The permanently unstable are those who, though fairly reasonable during most of their lives, are constantly subject to fresh attacks. In some, the recurrences are at long intervals, and are comparatively harmless to the nervous system; in others the frequency or severity of the attacks causes progressive destruction of mind.

The symptoms may be maniacal or melancholic. Each attack as a rule resembles its predecessors in mode of development, symptoms, and mode of termination, but each attack may further impair the stability.

The patient may have an attack of acute mania and recover, a second attack in four years, a third in three, a fourth in two years, and after that one or more attacks may occur yearly till the patient either passes into chronic mania or into hopeless weak-mindedness. In cases of recurrence, if maniacal symptoms are present, powerful drugs such as hyoscyamine in one hundredth of a grain dose, *sub cute*, may check the outbreak or the recurring habit, especially if the drug be given when the earliest sign of disturbance is noticed.

In recurrent cases of melancholia with suicidal ideas, with each recurrence suicide must be guarded against. Therefore such cases are always anxious ones if allowed to be at large.

With the unstable cases hereditary tendency is common. Such cases may live to an ordinary age. In some cases a weak-minded state gives place periodically to outbursts of violence.

Puerperal Insanity.—There is no special form of insanity deserving the name "puerperal," yet, as the puerperal conditions are not infrequently causes of insanity, the term Puerperal Insanity is convenient.

Insanity may be developed during pregnancy; it may be but an exaggeration of the longings of pregnancy, and on several occasions a distinct connection has been traced between some absurd or morbid appetite and a neurotic tendency.

Beside the mere longings of pregnancy and their insane exaggeration, one meets with true insanity occurring early in pregnancy. This is most common in women who have already suffered from some form of insanity. Thus, a woman who has had one or more attacks of puerperal insanity is liable to a development of this disorder with a succeeding pregnancy. Insanity of pregnancy may occur in the earlier months, and may pass off spontaneously about the fourth month; or the insanity may develop during the later months of pregnancy, and in that case will probably not pass off till long after delivery. As a rule, the insanity of pregnancy is of a depressed type, the patient being irritable, suspicious, jealous, refusing food, and dreading poison. Sleeplessness, refusal to take food, and tendency to suicide or infanticide, are the most dangerous symptoms, and those which have to be guarded against. The induction of premature labour is generally useless; and the best treatment is to improve the general health by change of scene, removal from home, or at least removal of children from home, withdrawal of husband's society, and careful watching.

The second group of cases of insanity with pregnancy contains those that have become insane after the fourth month. Such patients have frequently suffered much from exhaustion, due to frequent childbearing, repeated lactations, or vomiting and pregnancy. They may be maniacal, but usually suffer from melancholia, with ideas of unworthiness, and are strongly suicidal, and often infanticidal; some suffer only from simple nervous exhaustion and stupor. In treating these cases, rest, and abundant suitable food, and generally removal from home suffice.

It is rare for the insanity to pass off spontaneously with delivery. In some cases during the pains of labour, and immediately after, there is a temporary improvement, but as a rule the sanity following labour in such cases is temporary.

The next class to be considered contains those who become insane soon after delivery. Puerperal insanity occurs in a very large proportion among women with distinct insane inheritance. The causes are of long standing, rather than sudden; repeated pregnancy, poverty, exhaustion from sickness during the pregnancy, or several of the above causes combined, and though there are few noteworthy symptoms till after delivery, yet we should be prepared to find, on careful investigation, that there have been threatenings and warnings which have been neglected.

Within two or three days of delivery occasionally there is a temporary or transitory maniacal attack allied to the febrile disturbance associated with the oncoming of milk, and, like that, the transient mania may be relieved by a free purge. It is noteworthy that in this transitory mania infanticide or suicide may occur.

Insanity occurring after labour is divided into that which befalls the puerperal woman at once, and that which develops within the first two months after delivery. The former contains the greater number of maniacal cases, and the latter the more melancholic. Puerperal insanity does not follow instrumental labours in any great excess. Quite natural and easy labours are frequently followed by puerperal insanity in predisposed subjects. Puerperal insanity has undoubtedly a great tendency to recur in the same individual, but need not recur although she become again pregnant. Puerperal insanity may be characterised by either maniacal or melancholic symptoms, or from the first there may be stupor. The onset of the disorder is generally as follows:—Sleeplessness for several nights, with irritability, loss of appetite, and querulousness, especially against the husband and child; then increased irritability or anxiety, with ideas of poisoning and refusal to take food; afterwards appear hallucinations of the senses, violent outbreaks occur, or profound melancholy may develop, in the one case the woman being destructive, and in the other suicidal. During this period the milk and lochia may be natural; on the other hand, one or both may be absent. From a practical point of view it is necessary to attend carefully to the milk, and to follow the old wives' simpler methods of "dispersing the milk" rather than making use of belladonna and other poisonous drugs. Friction with castor oil or the application of hot salt is useful.

An attack of puerperal insanity usually lasts for several months, and requires most careful watching and care. The patients rapidly emaciate, often neglect themselves, and develop some secondary disorder which often proves fatal. There is no more special danger in the insanity than there is in the puerperal condition, but the two combined require double care in their treatment. Abundant feeding is the rule, with removal from home, and the sparing use of chloral or bromide of potassium.

The period of excitement may be unduly prolonged, so that in some cases patients are maniacal for eight or ten months before there is any sign of abatement of the disorder. Among the most characteristic signs of puerperal insanity in its later stages is the patient's discontent and jealousy of her husband, her tendency to mistake those who are about her, and the appearance of a weak-minded stage which is extremely difficult to treat. Following an acute attack of puerperal insanity we must expect to have a prolonged period of depression; and as soon as the patient has ceased to be actively antagonistic to her husband and home, and especially as soon as she expresses a desire to return to her family, it is better to make some movements in that direction. It will be found at this period that the patient gets stout, indolent, sleepy, and sometimes is too contented with an asylum; amenorrhœa also exists, and frequently this amenorrhœa and general condition of apathy are only relieved by return to home and domestic cares.

In such prolonged cases a second pregnancy is particularly hazardous; and in all cases of puerperal insanity it is of the utmost importance to impress upon the husband the danger of another pregnancy occurring within at all events two years.

Seventy-five per cent. of puerperal cases recover, about 5 per cent. die, and 20 per cent. remain chronic. Of those who recover, the majority recover slowly; mental and bodily health improve together. Of those who die, some die from septicæmia, but more from exhaustion due to the weak bodily condition associated with the mental excitement. Of those who are uncured, a large proportion have had many previous attacks, but a certain number of patients do not recover from their first attacks of puerperal insanity, and it is therefore well to be guarded in the prognosis even from the first.

Beside puerperal insanity, we have a form of mental disorder associated with *lactation*. From two or three months after delivery patients become suspicious, sleepless, irritable, and inclined to wander from their homes. In some cases there is amenorrhœa, but more often menorrhagia, due probably to incomplete involution of the uterus. These patients are sallow and anæmic, with bad appetite and constipated bowels; they frequently suffer from hallucinations of smell, taste, and hearing.

As a rule they require merely tonic treatment. They recover in the course of two to six months if removed to healthy surroundings, and away from home cares and worries. They require warmth, food, stimulants, fresh air, and, in the summer, baths, and the seaside.

Alcoholic Insanity.—Although no one special form of insanity depends upon drink, yet there seems to be some relationship between alcoholic intemperance and the variety of insanity. It is therefore well to recognise the chief forms assumed by insanity depending upon drink.

Sudden outbursts of drinking in nervous subjects will lead to delirium tremens, and delirium tremens, instead of passing off as it ordinarily does, assumes in such cases more or less the character of a chronic delirium. With some patients acute delirious mania follows an acute alcoholic debauch. In such the removal of stimulants alone will not suffice to cure at once, and the persistent use of opium is rather injurious than otherwise. Hallucinations of sight and hearing similar in all respects to those met with in delirium tremens occur, but are more persistent; refusal to take food is constant, emaciation takes place rapidly, and, unless the patients are fed abundantly with milk and nutrient soups, a fatal issue may be expected. If the patient be young and strong, a purge by croton oil and an emetic of sulphate of zinc may be found the best treatment to start with. If the excitement seems likely to wear out the patient, chloral alone or with bromide of potassium may be given at night, but it is best not to give these drugs continuously or in too large doses. Peraldehyde in doses of one to two drachms is useful.

Beside the acute mental disturbance produced by alcohol, we meet with every variety of delusional insanity, patients believing that their food is poisoned, that they are being watched, spied upon, and the like. Jealousy with vindictiveness is very often met with. Insane interpretations of morbid cutaneous and muscular feelings also occur; thus patients will complain of being galvanised, mesmerised, or "interfered with." The prognosis in those suffering from delusional insanity due to alcohol is not very hopeful, and in any case they should be kept under observation for many months before they are discharged as *well*. In some cases the morbid sensations persist and the morbid interpretations remain, and yet these cease to be active agents upon the patients' will, so that we meet with chronic cases of alcoholism, with chronic delusions, who are able to be at large without danger to themselves or friends. Chronic alcoholism probably tends in some cases to the production of general paralysis of the insane; it is much more certain that it tends to senile dementia, and in the cases of senile dementia connected with alcohol there is a great tendency to apoplexy, hemiplegia, and paralytic weakness of mind.

It is legal to send a patient suffering from alcoholism into an asylum if he cannot be controlled, or if he be dangerous to himself or others; yet we ought to be extremely cautious in signing certificates for persons suffering from chronic alcoholism with symptoms of insanity, for though seclusion will restore them to their former mental state, that is not a healthy or normal one, and the prospect of legal proceedings is in such cases considerable.

MORAL INSANITY.—It may seem unphilosophical to describe a form of insanity as belonging to the moral apart from the intellectual side of man, but the term will perhaps appear clearer to some readers if moral insanity be defined as a perversion of the nervous system by which the man or woman is rendered "anti-social." To give an idea of what is meant I would say there are two distinct conditions under which moral insanity may exist: first, the children of insane or highly neurotic parents seem in many cases to come into the world unable to grow up into full intellectual and moral capacity; secondly, there are certain diseases of the brain or disorders of the mental functions which, having upset the nervous balance, leave a fresh moral standard far below what was the normal and healthy standard of the individual. Thus there are some patients who are unable to develop into their moral and social places, and there are others who having filled their proper positions are, as the result of disease, reduced to a lower moral rank.

First as to the young patients, the offspring of parents who have been drunken, epileptic, or insane, not uncommonly are morally rather than intellectually insane. They are perverse and incapable of being trained into their duties to society. They are frequently sexually precocious and vicious, they are cruel, mischievous, untruthful, thievish, and at times fire-raisers. At the same time these persons may have one or two special gifts. Thus, they may be extremely rapid calculators, they may be able to reproduce musical combinations, or they may have wonderful memories for isolated facts. As a rule such patients require isolation. As they grow older they become more dangerous from their sexual passions and from their destructive tendencies. They often pass into eccentric, weak-minded drudges in asylums.

Beside these we have to consider those who are morally weak as the result of acute attacks of insanity. After acute mania some young patients become kleptomaniacs, others lose all self-control and become amorous and impulsive, others cease to be truthful and in one way or other show marked anti-social qualities. In these cases there is little or no prospect of recovery,—they are like people who have been scarred by the smallpox in whom nothing will ever efface the marks.

Not only after mania but after other forms of mental disorder, moral defects may become manifest; and similar changes occasionally follow fevers; temporary, and in a few cases permanent, moral changes have been observed to follow an attack of typhoid or rheumatic fever, especially with hyperpyrexia.

Moral perversion may occur as an early symptom of general paralysis of the insane; it is common with alcoholism and is frequently seen in the earlier stages of senile dementia. In some cases the faults or vices of old men are really the result of early degeneration of the brain.

Moral insanity is scarcely capable of medical treatment. In the case of many young and middle-aged persons the best and indeed the only course is to let the delinquent receive the due punishment of his faults. The discipline of the jail has undoubtedly done good in some cases. Such young persons will ruin themselves and their friends, and if steps are taken to send them to asylums, they often retaliate and cause endless legal trouble.

Syphilis may give rise to various forms of insanity. The moral effect of the consciousness of transgression may set up hypochondriasis with syphiliphobia, and this may end in general paralysis of the insane. Syphilis may produce local brain lesions with various degrees of mental weakness: or syphilitic epilepsy may lead to the same result. Syphilis may give rise to arterial changes which may lead to dementia, or to paralysis ending in dementia. In the majority of cases of general paralysis of the insane there is a history of syphilis.

It is not uncommon to find patients who have suffered from syphilis and who have recovered from secondary symptoms, which have been called Pseudo-General Paralysis, pass into weak-mindedness or into stages hard to be distinguished from general paralysis. In a certain number of weak-minded children and idiots, and in some elder patients, congenital syphilis has produced sensory or intellectual weakness or developmental General Paralysis. In any case of insanity in which syphilis has previously occurred, anti-syphilitic drugs should be used; but in most cases resembling general paralysis or with marked mental defect no good result will follow such treatment.

Gout may occur in neurotic families and may be of special importance. Before and after gouty attacks there are often marked mental changes in patients, and in some cases of so-called suppressed gout most profound and suicidal melancholia may occur only to pass off when acute gout appears. Gout may seem to alternate with insanity as with asthma or eczema. Gouty degeneration of arteries is not uncommonly associated with signs of senile decay and dementia.

Lead-poisoning may produce maniacal excitement or it may end in dementia. The same poison may produce all the symptoms met with in general paralysis, and may almost certainly give origin to that disease. It also leads to epilepsy and its results.

After *fevers* it is not uncommon to meet with more or less mental weakness, which does not depend much upon the severity of the fever. This is not uncommon after typhoid fever, and may range from simple loss of memory to loss of control or mania, with emotional disturbance. In persons of highly nervous temperament, the delirium of any fever may set up mental disorder, which may appear as acute delirious mania or, more commonly, simple acute mania.

Phthisis is related to insanity in many ways. The insane are more than usually liable to phthisis. Insanity in one parent and phthisis in the other gives rise to a very unstable type of mind. Many patients who recover from severe attacks of mania or melancholia die a year or two afterwards of phthisis.

A form of insanity called phthisical insanity has been described. The patients are suspicious, and refuse their food because they believe it to be poisoned; they often have hallucinations of taste and smell, and of hearing; they rapidly waste, without having any cough or expectoration; the temperature is more or less like that of phthisis, and dulness is found if sought for under the clavicles. These patients require forcible feeding with stimulants and cod-liver oil. Hæmoptysis with temporary relief to the mental symptoms is not uncommon.

Spasmodic asthma often occurs in neurotic families, and it may occur in persons who have had attacks of insanity. In some cases the spasmodic attacks disappear and the patient becomes melancholic, only being restored to his right mind when the asthma returns.

Heart disease may act as a cause of insanity; mitral incompetence being most frequently associated with feelings of distress, anxiety and melancholia, while aortic lesions, and at times mitral contraction, have been met with associated with mania and great excitement.

With *renal disease* the tendency is toward mental depression.

Diabetes is not uncommon in neurotic families, nor is it very rare in insane persons; but often with the onset of insanity the diabetes disappears.

Epilepsy and insanity are allied by origin. Epilepsy occurs in neurotic subjects, and epilepsy in infants causes idiocy or imbecility. Frequent recurrence of epileptic fits, whether of the major or the minor form, leads to weak-mindedness. Hallucinations of the senses may precede the fits; and after them periods of unconsciousness may follow, during which highly complex acts may be performed, the patient being in a state allied to somnambulism. Epileptic fits may be followed by fury of the most violent kind, during which brutal and bloody deeds may be done (p. 918).

Convulsions may possibly be replaced by mental disorder—" l'épilepsie larvée." In this the patient after a sudden arrest of the work in hand goes through more or less highly organised acts unconsciously; these acts in each recurrence are usually alike; and just as each epileptic fit is a repetition of its predecessors, so is each mental attack like its forerunners.

As a rule these automatic states follow some epileptic seizure.

Jacksonian epilepsy is not so frequently followed by mental disorder as is the idiopathic epilepsy which occurs in neurotic subjects.

Paralytic insanity.—After apoplexy there is a great tendency to weak-mindedness. This is not always enough to deprive the patient completely of self-control, but it often leads to loss of memory and to a certain want of self-control, manifested in erotic lust, in emotional weakness, and in a tendency to be easily influenced by others. It commonly passes from the slighter to the graver forms of dementia, especially if fits recur.

Tumours of the brain generally tend more or less certainly to loss of some of the functions of the mind.

In *locomotor ataxy* we frequently meet with a neurotic history, and in many cases ataxy is an early symptom of general paralysis. But besides, with locomotor ataxy there may be outbreaks of mania, generally of a suspicious, jealous nature; or the symptoms of the disease may be falsely interpreted by the patient: he may say that his legs are electrified and his sexual power removed by his enemies, or he may accuse people of twisting his bowels or destroying his sight.

If the symptoms are part of general paralysis, the prognosis is bad; but if due to locomotor ataxy, the prognosis is much better as far as length of life is concerned.

ADMINISTRATIVE CAPACITY.—A most important duty of the medical man is to learn to judge of the mental capacity of a patient who may have had an attack of insanity or other nervous disturbance. It must be remembered that some persons who have been insane recover completely, and may be able to transact business as well as ever; but that the prospect of recovery and of mental capacity is lessened with the increasing number of attacks and with increasing years. Wills may be made by persons who are chronic lunatics, and yet the wills may be so reasonable that judge and jury will support them.

To upset testamentary capacity it must be shown that the patient had no knowledge of what he was doing, or that he had mistaken ideas about what he was effecting, or that his judgment was biassed by delusions of one kind or other, or that, being weak, he was unduly influenced. Defective memory is a thing to be specially noted. If it can be shown that a man when making a will did not remember the number of his children or whether they were living or dead, there would be ground for disputing his will. If, on the other hand, it can be shown that he was emotional and easily led by others, even though the memory was not very defective, yet it might be shown that he was too readily influenced at the time he made his will by those who were near him. If it can be shown that he had a causeless antipathy—the result of a delusion—to his direct heirs, it is only necessary to prove the nature of these delusions and their existence about the time the will was made. Apoplexy probably gives rise to weak-mindedness more frequently than any other disease of the brain, and the mental weakness produced by apoplexy certainly gives rise to the greatest number of trials in the probate courts. A man may have one or more attacks of apoplexy and yet may remain of "disposing" mind; but it is well to remember that after fits of apoplexy memory is very frequently affected, emotional disturbance is readily raised, and other signs of weakness and change of character are generally present.

There may be complete testamentary capacity without capacity for speech, in fact aphasia and weak-mindedness are not necessarily related; but it may be found difficult to get a jury to understand that a person unable to give the name to the simplest object may yet be able to dispose of his property. This difficulty will be greatly increased if agraphia also is present.

THE CONFINEMENT AND RESTRAINT OF INSANE PATIENTS.—One of the most difficult points for a medical man to decide is as to whether a patient should or should not be sent to an asylum; and having decided on sending a patient away from home, the next difficulty arises as to where the patient should be placed.

For the reception of persons of insane mind there are houses receiving one patient only; this is called *single care*. These houses are without any licence, and no certificate is necessary for many patients so placed. But it must be understood that, even though a patient be placed in "single care," it will be necessary to have the ordinary medical and other certificates in due and proper form, if he is so insane as to need his actions to be controlled.

Beside houses which receive only one patient there are others, *licensed houses*, for the reception of two or more, and these merge insensibly into the private *asylums* which are to be found in most counties of England. Next there are registered *hospitals* into which patients are received at various rates, the principle of the hospital being that while some patients are received free or for small sums, others pay more than their actual cost, and thus help to pay for the treatment of the poorer. There are also the large borough and county *asylums*, expressly intended for the reception of those who are not sufficiently wealthy to pay for private asylums, and who, from one cause or another, are ineligible for hospitals. In some of these paying departments at low rates have been established. Lastly, a certain number of quiet harmless lunatics are confined in the *workhouses* and workhouse infirmaries.

If a person of unsound mind is to be removed to the borough or county asylum, it is necessary that notice should be given to the relieving officer of the parish, who in due course reports to the medical officer of the same parish or some other medical man; and the patient being brought before a magistrate, and the medical man having certified that he is of unsound mind and unfit to be at large, the magistrate signs an order for the transference of the patient to the county asylum. If a person of unsound mind be found wandering about the streets, he may be given in charge of the police, and, being taken before the magistrate, a doctor being called by the magistrate, may give evidence which satisfies the magistrate that the person is of unsound mind and requires detention; and then he may, on the magistrate's order and the medical man's certificate, be sent to the asylum. No patient can be kept or retained in a medical man's house for profit except under full legal certificates, unless he be a near relative, it being manifest that no one's liberty of action must be restrained to the advantage of another without legal authority.

There must now be *two medical certificates* on separate sheets of paper (one of these being by the regular medical attendant), a *statement* of particulars in the form given, and a *petition* to a magistrate or judge specially named for the duty; these must all be presented to the magistrate, who then satisfies himself of the correctness of forms and the necessity for the detention of the patient—he may see the patient or not as he wishes,—and signs *the order* for his reception into any hospital or private asylum or private house. A patient may be sent to any asylum on one medical certificate and the statement, provided that at once steps are taken to get the petition and other certificates after his admission (see *Urgency Form*). Patients may place themselves in asylums voluntarily with the consent of the Commissioners.

FORMS 1 AND 2.

Form of Urgency Order for the Reception of a Private Patient.

I, the undersigned, being a Person Twenty-one years of age, hereby authorise you to receive as a Patient into your (a) ______________

(b) ______________

as a (c) ______________ whom I last saw at ______________

on the (d) ________ day of ______________ 19 .

(a) House or hospital, or asylum, or as a single patient.

(b) Name of patient.

(c) Lunatic, or an idiot, or a person of unsound mind.

(d) Some day within two days before the date of the order.

I am not related to or connected with the Person signing the Certificate which accompanies this Order in any of the ways mentioned in the Margin (e). Subjoined [or annexed] hereto is a Statement of Particulars relating to the said ______________.

(e) Husband, wife, father, father-in-law, mother, mother-in-law, son, son-in-law, daughter, daughter-in-law, brother, brother-in-law, sister, sister-in-law, partner, or assistant.

(Signed)

Name and Christian Name at length ______________

Rank, Profession, or Occupation (if any) ______________

Full Postal Address ______________

How related to or connected with the Patient ______________

[If not the husband or wife, or a relative of the patient, the person signing is to state as briefly as possible—1. Why the order is not signed by the husband or wife, or a relative of the patient. 2. His or her connection with the patient, and the circumstances under which he or she signs.]

Dated this ________ day of ______________ 19 .

To (f) ______________

____

(f) Proprietor or superintendent of —— house, or hospital, or asylum (describing house or hospital or asylum by situation and name).

[In cases of urgency a lunatic person may be placed under care and treatment upon an Urgency Order made (if possible) by the husband or wife, or by a relative (i.e. a lineal ancestor or lineal descendant, or a lineal descendant of an ancestor not more remote than great-grandfather or great-grandmother), accompanied by a Statement of Particulars and one Medical Certificate. If a Petition for an Order for Reception of the patient has been already presented to a Judge, Magistrate, or Justice, a copy of the Urgency Order must be sent forthwith to such Judge, Magistrate, or Justice. An Urgency Order will remain in force for seven days from its date, or if a Petition for a Reception Order is pending, then until such Petition is finally disposed of.

The Urgency Order, Statement of Particulars, and Medical Certificate must be sent to the Superintendent or Proprietor of the Asylum, Hospital, or House where the patient is to be received.]

Lunacy Act, 1889.

(52 and 53 Vict., c. 41.)

This, accompanied by the *Statement*, will suffice to remove a person of unsound mind to an asylum, when the further certificates can be obtained.

STATEMENT OF PARTICULARS.

Statement of particulars referred to in the annexed petition.
ollowing is a statement of particulars relating to the said

(*Name of Patient in full*) ________________

N.B.—*If any Particulars are not known the Fact is to be so stated.*

Name of Patient, with Christian Name at length . . . ________________

Sex and Age . . . . ________________

Married, Single, or Widowed . ________________

Rank, Profession, or previous occupation (if any) . . ________________

Religious Persuasion . . ________________

Residence at or immediately previous to date hereof . . ________________

Whether First Attack . . ________________

Age on First Attack . . . ________________

dates. WHEN and WHERE previously under care and treatment as a lunatic, idiot, or person of unsound mind . . . . ________________

Duration of existing Attack . ________________

Supposed cause . . . . . ________________

Whether subject to Epilepsy . ________________

Whether Suicidal . . . ________________

Whether dangerous to others, and in what way . . . ________________

Whether any near Relative has been afflicted with insanity . ________________

Names, Christian Names, and full Postal Addresses of one or more Relatives of the Patient . ________________

Name of the Person to whom notice of Death to be sent, and full Postal Address if not already given . . . ________________

Name and full Postal Address of the usual Medical Attendant of the Patient . . . ________________

How many previous attacks?

Has the Patient been of Sober Habits? ________________

Number of Children? ________________

Age of youngest? ________________

Degree of Education? ________________

Signed ________________

When the Petitioner or Person signing an Urgency Order is not the Person who signs the statement, add the following particulars concerning the Person who signs the statement.

Name, with Christian Name at length . ________________

Rank, Profession, or occupation (if any) . ________________

How related to or otherwise connected with the Patient . ________________

–The Patient must be received into the Hospital before the expiration of *n clear days from the date of the Judge, Magistrate, or Justice's Order.*

ORDER.

Order for reception of Patient, to be made by the Judge of County Courts, Stipendiary Magistrate, or Justice appointed under the Lunacy Act, 1890.

I, the undersigned (*Name*)______________________________
being the Judge of the County Court of__________________________
***or* the Stipendiary Magistrate for_____________________________**
***or* a Justice for______________specially appointed under the Lunacy Act, 1890, upon the Petition of (*Name of Petitioner*)____________**
of (*Address and Description*)_____________________________
in the matter of (*Name of Patient*)__________________________
a person of unsound mind, accompanied by the Medical Certificates of (A)______________________and (A)________________ hereto annexed, and upon the undertaking of the said (*Name of Petitioner*)______________________to visit the said (*Name of Patient*)______________________personally or by some one specially appointed by the said (*Name of Petitioner*)____________________ once at least in every Six Months while under care and treatment under this Order, hereby authorise you to receive the said (*Name of Patient*)______________________as a Patient into your Hospital. And I declare that I have [*or* have not] personally seen the said (*Name of Patient*)______________________before making this Order.

(A) Names of Medical Practitioners signing Certificates.

Dated____________________

Signed____________*Judge of the County Court of*__________
____________or *Stipendiary Magistrate,*
To the* ________________or *a Justice for*____*appointed
Resident Physician,* *under the said Act.

N.B.—*Under all circumstances, if possible, the "PETITION" and "STATEMENT" below to be filled up by the Patient's Relatives. If no Relatives, by the nearest Friend.*

PETITION FOR AN ORDER FOR RECEPTION OF A PRIVATE PATIENT.

In the matter of (A)____
a person alleged to be of unsound mind.

(A) Name of Patient in full.

To His Honour the Judge of the County Court of
or To________________Stipendiary Magistrate for______________
or To________________a Justice of the Peace for______________
The Petition of (B)______________________________________
of (1)____
in the County of____
1.—I am (2)______________years of age.
2.—I desire to obtain an Order for the reception of (*Name of Patient in full*)______________________as a person of unsound mind in ____

(B) Name of Petitioner in full.

(1) Full Postal Address, and Rank, Profession, or Occupation.

(2) At least twenty-one.

3.—I last saw the said (*Name of Patient in full*) ______
at ______________ on the (3) ______ day of ______ 19 .

(3) Some day before the date of the presentation of the Petition.

Here e the con- tion or tionship h the ient.

4.—I am the (4) ______________ of the said (*Name of Patient in full*) ______________

If the Petitioner is not connected with or related to the patient, state as follows:—

I am not related to or connected with the said (*Name of Patient in full*) ______________

The reasons why this Petition is not presented by a relation or connection are as follows:—

____

____

____

The circumstances under which this Petition is presented by me are as follows: ______________

____

5.—I am not related to or connected with either of the persons signing the Certificates which accompany this Petition as—

(*where the petitioner is a man*) husband, father, father-in-law, son, son-in-law, brother, brother-in-law, partner, or assistant;

(*or where the petitioner is a woman*) wife, mother, mother-in-law, daughter, daughter-in-law, sister, sister-in-law, partner, or assistant.

6.—I undertake to visit the said (*Name of Patient in full*) ______ personally, or by some one specially appointed by me, at least once in every Six Months while under care and treatment under the Order to be made on this Petition.

7.—A Statement of Particulars relating to the said (*Name of Patient in full*) ______________ accompanies this Petition.

If it is the fact, add—

8.—The said (*Name of Patient in full*) ______________ has been received in the ______________ under an Urgency Order, dated the (5) ______________

) Here in- rt date of rgency rder, any.

The Petitioner therefore prays that an Order may be made in accordance with the foregoing Statement.

Full Christian and Surname of Petitioner ______

Dated ______________

N.B.—If neither of the Practitioners signing the Medical Certificates be the usual Medical Attendant of the Patient, the reason of this must be stated in writing to the Judge, Magistrate, or Justice by the Petitioner.

.B.—Medical Certificates of Patient's Examination, and the Signatures, are required by the above Statute to be dated *not more than Seven clear Days* previously to the date of the presentation of the Petition.

edical Practitioners signing the Certificates must not be in Partnership, nor one the Assistant of the other; nor must they be related to one another, as father, father-in-law, mother, mother-in-law, son, son-in-law, daughter, daughter-in-law, brother, brother-in-law, sister, sister-in-law, partner, or assistant; nor must they themselves sign the Petition or Urgency Order, nor must they be related to the Petitioner in any of the ways specified in the Petition.

One of the Certificates shall, *whenever practicable*, be under the hand of the usual Medical Attendant, if any (being a medical practitioner), of the alleged lunatic. They must use the terms specified in the Statute. (*See* marginal notes.)

By Order of the Commissioners in Lunacy.

1.—It is absolutely necessary that the Medical Men should write their Certificates legibly, so as to afford the opportunity of an exact copy being made.

2.—"All alterations in the original Certificates, *unless by the certifying Medical Men*, invalidate them; and *the initials of the latter* must be placed to every change or addition made."

CERTIFICATE OF MEDICAL PRACTITIONER—Schedule 2, Form 8.

In the matter of (*Name of Patient*)______________________

(1) of______ ______ in the______ (2) of______________

(3)______ ______ an alleged lunatic.

I, the undersigned (*Name of Practitioner*)______________

do hereby certify as follows:—

(1) *Insert* RESIDENCE *of Patient.*

(2) *County, City, or Borough, as the case may be.*

(3) *Insert* PROFESSION *or* OCCUPATION (*if any*).

1. I am a person registered under the Medical Act, 1858, and I am in the actual practice of the medical profession.

2. On the_____day of___________, 19 , at (4) ______________ in the (5) ______ of ______, separately from any other practitioner, I personally examined the said (*Name of Patient*)_________________and came to the conclusion that he is a person of unsound mind, and a proper person to be taken charge of and detained under care and treatment.

3. I formed this conclusion on the following grounds, viz.:

(*a*) Facts indicating Insanity observed by myself at the time of examination (6), viz.:—

____

____

____

____

(4) *Insert* PLACE *of examination, giving the* NAME *of the street, with* NUMBER *or* NAME *of the house, or should there be no number, the* CHRISTIAN *and* SURNAME *of Occupier.*

(5) *County, City, or Borough, as the case may be.*

(6) *If the same or other* FACTS *were observed previous to the time of examination, the certifier is at liberty to subjoin them in a separate paragraph.*

(*b*) Facts communicated by others, viz.: (7) (State the NAME IN FULL of the person giving the information, with the address and description.)

____

____

____

(7) *The* NAMES *and* CHRISTIAN NAMES (*if known*) *of informants to be given, with their* ADDRESSES *and* DESCRIPTIONS.

4. The said (*Name of Patient*)______________________ appeared to me to be (*or* not to be) in a fit condition of bodily health to be removed to__________________________.

5. I give this Certificate, having first read the Section of the Act of Parliament printed below.

Dated___________19 . Signed ______________________

Full Postal Address______________________________

Extract from Section 317 of the Lunacy Act, 1890.

"Any person who makes a wilful misstatement of any material fact in any Medical or other Certificate, or in any Statement or Report of bodily or mental condition under this Act, shall be guilty of a misdemeanour."

N.B.—By Section 28, "Every Medical Certificate made under and for the purposes of this Act, shall be evidence of the facts therein appearing, and of the judgment therein stated to have been formed by the Certifying Medical Practitioners on such facts, as if the matters therein appearing *had been verified on oath.*"

In signing certificates it is well to remember that the most important points are the facts observed by one's self, and these facts bear much greater weight if they can be shown in brief to be associated with a development of nervous disturbance. Thus "sleeplessness, restlessness, refusal to take food, threats of suicide, and a tendency to wander from home," would suffice, but would be strengthened by the fact communicated by A— B—, the nurse, that the patient tried to throw herself from the window, and said she was possessed by the devil. Whenever the certificate is strengthened by the addition of facts communicated by others, the name in full of such person must not be omitted. Certificates in England only hold while the patient is in England, and are broken by escape to the Continent, to Scotland, or to Ireland; but if a patient escape from an asylum, and remain in England, the same certificates hold good for fourteen days, during which period he may be recaptured.

No fresh licences for the reception of lunatics into private houses are granted by the Commissioners. Thus no fresh private asylums can be started. Patients under certificates are in direct relationship to the Commissioners, who require a complete copy of the certificates upon which they have been received, as well as a statement within a week of the reception of the patient as to his bodily and mental condition. They also require that a medical visitation book of a prescribed form should be kept and entries made from time to time of the visits paid to the patient by some independent medical man. The frequency of these visits will be decided upon by the Commissioners themselves. All letters written by the patients to the Commissioners in Lunacy and to certain other specified persons, must be forwarded unopened; all other letters of patients must either be forwarded to their address, or, being initialled, must be kept for the inspection of the Commissioners on their visit, or of some other constituted authority.

Patients can be transferred from a private asylum into a private house, or from a hospital to a private house, or the reverse, permission having been sought and granted by the Commissioners in Lunacy. When a patient is sent from one establishment to another, the order of the Commissioners, with a copy of the original order, statement, and certificates, must be forwarded with him. Patients cannot be transferred from a private asylum or private care to a county or borough asylum, or the reverse.

Though a patient is of unsound mind and under certificates, his friends and relatives have no right to transact business in his name even though they have recently obtained a power of attorney from him, for the signing the certificate of lunacy practically cancels the power of attorney. In some cases where the power has been given for a fixed time before the patient has become irresponsible, it has been allowed to have force.

If it be necessary to transact important business connected with a person of unsound mind, the only legal method is to place the affairs in Chancery, there being a special department of the Court for the purpose. Of Chancery cases there are two groups. The first includes only small properties, that is, under £2000 capital or £100 a year income, and for these the procedure is simple and rapid. It is as follows:—Two affidavits have to be drawn up in a form to be presently referred to, stating in detail the facts of the patient's insanity and his inability to transact business. These affidavits having been laid before the Judge or Master in Lunacy, with certain other facts as to his affairs and his relations, by the family solicitor, an order may be given by the Court of Chancery for the administration of this small estate; and two people, respectively called the committee of the person and the committee of the estate, will be appointed.

The following is an epitome of the second and longer process, which is costly and difficult to set aside, and therefore not to be undertaken without grave consideration: for the Court of Chancery will almost certainly realise the property of the lunatic, and a man's property may be realised at a very great loss while he is of unsound mind. It is therefore best to avoid placing any acute and presumably curable case under the Court of Chancery. When, however, it is necessary to put the affairs of a patient under the care of the Court, the family solicitor generally asks the medical man in charge of the case, or the asylum superintendent, to make a draft affidavit, and he also probably gets some independent medical expert in lunacy to draw up a second affidavit. These affidavits differ entirely from the ordinary medical certificate, there being no restriction as to consultation or separate and independent examination of the patient, and it is best to fill the affidavit up in the following way:—First, state your name, your medical qualifications, your qualifications to judge of the sanity of the patient, more especially your opportunities of having seen him; then give your judgment as to the form of disease from which he is suffering; give in the next two or three paragraphs a statement as to his general behaviour, his intellectual capacity, the delusions from which he suffers, the tendencies to suicide or destructiveness which he may have, and finally give your judgment as to the improbability of his speedy recovery; or, if you believe him to be fatally or incurably insane, give your judgment that he is so, and is unfit to take care, first of himself, second of his affairs. These two affidavits having been presented, in due course notice will be given to the patient that an inquiry or so-called inquisition or commission in lunacy will be held. The patient may then instruct counsel himself, or he may demand at the inquiry to be defended by counsel and to be present himself. Or he may demand that he may be tried before a Master in Lunacy with a special jury. In one case at least a judge of the Court of Chancery took the position of the Master in an inquiry. In some cases where the patient is extremely suspicious, there may be considerable difficulty in persuading a jury that he is of unsound mind; and in nearly all cases where there is likely to be opposition of this kind, where in fact the patient is still so reasonable as to be able to defend himself, it is better to take any other means that are possible. If the patient be found insane by inquisition the Court of Chancery in due time appoints one person to look after the affairs of the patient, who is called the committee of the estate, and another to look after the person of the lunatic, who is called the committee of the person. The Master may decide that the alleged lunatic is only unfit to manage his affairs, and he is left at liberty.

A Chancery lunatic remains so though he travel or though he escape to the Continent; he can be transferred from one asylum to another or from an asylum to a private house without any order of transfer from the Commissioners, all that is required being a signed order from the Court of Chancery and the committee of the person.

If a patient recover after he has been made a Chancery patient, it is necessary, before he can have his freedom and control of his property, that the whole of the proceedings should be set aside by a similar process. Two medical men (preferably those two who have signed the first affidavits for the reception of the patient) should draw up two affidavits, stating the grounds for their belief that the patient is now fit to manage his affairs.

The county and borough asylums are intended only for pauper lunatics, but a large number of patients have to pass from private care to these county asylums because their friends are unable to pay the charges, which range from one guinea per week up to almost any sum. Therefore chronic patients have to be provided for by the parish, the friends being called upon to pay for their maintenance in proportion to their ability and to the cost in the asylum. A certain number of county asylums at present receive paying private patients for about one pound a week, and recent lunacy legislation now enables all county and borough asylums to receive such paying patients. Patients may, under certain conditions, place themselves in registered hospitals or private asylums as voluntary boarders.

Idiocy and Imbecility.—These two are but grades of the same want of intellectual development, "idiocy" ranging from almost complete absence of intelligence, whereas "imbecility" may approach very nearly to mere eccentricity and oddness.

Idiocy depends in the majority of cases upon structural defect in the brain. A certain number of idiots are the offspring of insane parents; certain others owe their condition to injuries received in early childhood; while certain forms of physical and mental disorder occurring in weakly or nervous children may arrest mental development at a higher or lower point, leaving as the result an idiot or an imbecile. The brain is so delicately constructed that a slight damage done to it during its period of growth interferes with its permanent and normal development.

In looking at the pathology of idiocy, we have to recognise the fact that some children are born with defective brains, so that in some there is very little more than the ganglia at the base: a brain which suffices for the nutrition of the body, but is unequal to the development of the mental functions. In other cases important parts of the brain are absent, its commissural fibres or the radiating fibres being deficient; and in the same category are to be considered the changes produced in the brain by defect of one or more of the senses. The child who comes into the world without sight and without hearing stands a very great chance of being an idiot through the sensory starvation of the brain. In idiots we meet with every grade of defective brains, from the anencephalous idiot, whose brain weighs but a few ounces, to the hydrocephalic idiot, whose brain is deficient though his head is enormous. In certain idiots there is a general deficiency more than deficiency of any one part of the brain. All parts are there, but they are not sufficient. The convolutions are found to be broad and few, the grey matter is either not fully developed or is defective in quantity, or there is an enormous overgrowth of the connective tissue, so that in some idiots there are not only enormous heads but weighty brains. Beside all these, there are some children who in aspect are healthy, but who are idiotic or imbecile in consequence of some ill-understood defect in the power of the brain to work. If one may use the term, there are some "functional" or "neurotic idiots" who have fairly sized brains with fairly developed convolutions, and yet with inability to make use of the brain they have. Some of these belong to the nervous stock generally, and others seem to have had their capacity for intellectual development arrested by some acute disorder of infancy.

Most idiots are short-lived; many are defective not only in mind but in body. Many of them have malformations of limbs or a peculiar stunted aspect, with awkward, ungainly walk. Most of them are restless and irritable. Although generally slow of comprehension and dull as far as education is concerned, yet there are a certain number of so-called *idiots-savants* in whom some special faculty of the mind seems to grow at the expense of all the rest. Just as one may meet with a morally imbecile person with certain intellectual activities fully developed, so the learned idiot may have musical, mathematical, or mnemonic ability of a high order.

Idiots have been divided according to their *capacity for speech*, the very lowest being unable to speak, the next group being able to articulate or make use of simple words, a higher grade being able to comprehend short sentences; while the lowest imbecile, somewhat in advance of the highest idiot, would have still greater power, and a higher imbecile would be able to learn and repeat many things, slowly perhaps, but yet surely. Other varieties have been based on the *facial peculiarities;* some idiots having extraordinarily receding foreheads have been compared to fish, and others have the aspect of a Chinaman, while some seem to resemble other races or even some of the lower animals; and when one takes up a book in which the physiognomy of man is compared with the physiognomy of brutes, one is struck with the examples which have evidently been taken in many cases from idiots and imbeciles.

For our convenience here it will be well to take the groups of idiots as arranged by Dr Ireland.

(1) *Congenital idiocy.*—The first class contains those cases which start in fœtal life; there is general weakness as a rule, mental and bodily, and the prospect is altogether unfavourable. The same holds good equally with the *microcephalic* idiot (2), whose head, according to Dr Ireland, generally measures less than seventeen inches; there being a distinct and absolute want of brain the prospect is bad. (3) In the third class of eclampsic idiots are placed those children who have ceased to develop in consequence of *convulsions* which have occurred during early childhood. Probably, however, a very large proportion of these come of a nervous stock, and are thus already unstable when some such exciting cause as teething suffices to start convulsions which so modify the brain in its development that it never assumes full vigour.

(4) The next is a nearly related group containing the *epileptics*. Fully one third of the idiots in the Metropolitan Idiot Asylum at Darenth are children whose idiocy is at all events accompanied by epilepsy. As a rule the epilepsy weakens both mind and body, and in many cases seems to be associated with great moral perversity, the epileptic idiot being hard to manage, and in very few cases suitable for home treatment. A certain number of epileptic idiots outgrow their epilepsy, and though mentally defective may be developed to a moderate intellectual standard. Such patients should be tried with bromide, but cautiously; food, fresh air, discipline, and association with healthier children are the best treatment.

(5) With *hydrocephalus*, idiocy is not uncommon, and if the hydrocephalus be great it is almost certain that there will be intellectual defect; but the majority of cases with considerable hydrocephalic and mental weakness die, so that the number seen in a large idiot asylum is not great. A good many of these cases by careful treatment improve, and from idiots may be trained into the class of useful imbeciles, to a certain extent trust-

worthy, but having organic deficiency of brain which prevents them becoming more than useful drudges. Beside the hydrocephalic idiot, we have to recognise a group of *paralytic* idiots (6), in whom some local defect of brain occurring in early life has given rise to paralysis of one or more of the extremities, and has left a mental as well as a physical scar upon the brain. In these cases again we can expect but little improvement from treatment; the weakened limbs may be galvanised, friction and passive movements with baths may be used, and some power may be gained in them, just as some slight power may be gained in the faculties. (7) *Cretinism* will be considered in the next chapter.

(8) The next group contains the *traumatic* idiot. A number of idiots are found to be males that are firstborn, and a certain number of others are found to have had injuries to their head during parturition, and it is therefore well to remember that the delicate brain, too severely pressed upon by a narrow pelvis, or interfered with unnecessarily or severely by instruments, may be so permanently injured that idiocy results. Injuries before birth, at birth, or soon after birth occurring to children, will frequently prevent development; and there is no doubt that falls and similar injuries in early infant life give rise to a considerable proportion of idiots. Many of these are rather imbecile than idiotic; for the brain has been so interfered with that it only develops partially, and with it the mental faculties also are impaired but not wanting.

(9) *Inflammatory* idiocy is closely allied to the last group, but under this head have been placed the idiots with hypertrophy of the brain (p. 850). Here there is excess of connective tissue which interferes with the proper development, or at all events the proper function of the grey matter. Beside these, there are also the cases in which some disease of the nose or ears has set up inflammatory changes about the base or sides of the brain and interfered with its general development in a more or less mechanical way. The grades of idiocy from this cause vary greatly.

(10) The only other important group is that associated with *deprivation of the senses*. It is quite certain that among the lower orders a considerable amount of weak-mindedness was formerly, and to a certain extent is still, due to the want of special education for the deaf and dumb. Deaf and dumb children if not educated will from the deprivation of external impressions be but partially developed; and some, who would be weak-minded at the best, are by want of communication with the outer world left imbecile or idiotic. The same thing is still more true of those who happen to have loss or impairment of more than one sense: to them the portals of knowledge are closed. Nevertheless if these patients are taken early and trained specially, a large amount of instruction may be given them, and great amelioration of their condition will occur.

Idiots, if in the lower grades, should almost always be sent away from home. If there are other children, the influence of the weak-minded child upon them is distinctly injurious.

CRETINISM AND MYXŒDEMA

"La vallée sans soleil donne le crétin."—BALZAC.

Cretinism as an endemic form of idiocy—Its anatomy—Endemic bronchocele—Sporadic cretinism in England—Relation of goitre to cretinism—The cretinoid condition in adults (myxœdema)—The condition which follows removal of a bronchocele in man—and the effects of removal of the thyroid in animals (cachexia strumipriva)—Treatment of myxœdema.

CRETINISM is an exotic disease, and we therefore have no English word for it. The word is adopted from Switzerland by the French (as *crétin*) and by the Germans (as *Kreidling*). Its origin is uncertain: some would derive it from *chrétien* (in the sense of "innocent"); others from a Romänsch word, *cretira;* and others from *creta*, in allusion either to the chalky complexions of those who suffer from the disease (Littré) or to the calcareous formation of the districts where it prevails. The German name points decidedly to some connection with *Kreide* (creta, chalk).

The mental symptoms of Cretins do not appear to differ from those of ordinary idiocy, as described in the last chapter, but there is in addition a characteristic malformation of the skull and body generally, and an absence of the thyroid gland. They show all degrees of defect of intelligence, so that a Royal Commission on Cretinism which reported to the Sardinian Government in 1848, divided them into three classes, for which they proposed the names of "crétins," "semi-crétins," and "crétineux."

Endemic cretinism.—The most marked form of this curious disease occurs endemically in the Alps, and here cretins are well known to differ from ordinary idiots. They are seldom more than four feet and a half in height, and often below three feet. They have large brachycephalic heads. The features are broad and ugly; the eyes wide apart; the nose flat at the root, with widespread alæ; the lips thick; the mouth large and wide open, so as to allow the saliva to escape. The forehead and cheeks are wrinkled and the skin is coarse and rough. The hair grows low on the forehead, and is coarse and bristly. The chest is narrow, the belly large, and the limbs crooked. The hands are clumsy and broad, with short fingers. All cretins look old alike, and one of twenty is like one of forty.

The admirable investigations of Virchow have shown that all the peculiarities in the configuration of the cretin's skull and face are the result of premature ossification of the base of the skull. In a cretinous fœtus he found complete synostosis of the basi-occipital and basi-sphenoid bones, with a continuous layer of diploë from one to the other, so that no trace of the original separation could be discovered. The basi- and pre-sphenoid bones were also fused together; but this is less important, because their union usually takes place soon after birth, whereas the basilar process and the sphenoid bone should remain separate until at least the fifteenth year. The premature completion of this "tribasilar synostosis" (as Virchow calls it) puts a stop to further growth of the basis cranii in an antero-posterior direction, and gives rise to a marked deformity in the shape of the base internally. The sella turcica is very narrow, and the parts in front of the posterior clinoid processes form a greater angle than normal with the clivus or basilar process.*

Laterally, the cretinous skull doubtless expands abnormally, so as to compensate for the deficiency in its antero-posterior diameter, and this gives to the features of the cretin their peculiar breadth. The sunken root of the nose is probably due to deficient forward growth of the vomer.

The configuration of the head and face in cretinism is, then, directly attributable to premature synostosis of the basal sutures; and probably, as Griesinger suggested, the shortening of the fingers and other parts of the limbs may depend on too early union of the epiphyses.

Clinical relation to bronchocele.—The most important fact in the ætiology of cretinism is the close relation which it holds to *endemic goitre.* In this country as well as in others, goitre prevails in districts where cretinism is not met with. Indeed, the only place in England where endemic cretinism has been observed is the village of Chiselborough in Somersetshire; and Dr Petherton, who in 1847 discovered it there, lived to see it almost die out. On the other hand, there is probably no district in the world where endemic cretinism occurs without goitre being still more common. Moreover, it has been noticed that when a family migrates into a place where both diseases are met with, goitre is the first to appear; it is only in the second or third generation that cretins present themselves. These facts seem to point to the conclusion that both diseases are allied in origin.

Many cretins are also goitrous; but others, instead of an enormous bronchocele, have no thyroid at all, and these are usually the worst cases. According to the Report of the Sardinian Commission, goitre is absent in one third of the cases of cretinism.

Sporadic cretinism.—It is remarkable that scattered cases of cretinism sometimes occur in England, and in these the thyroid is deficient. In papers in the 'Medico-Chirurgical Transactions' (1871) and the 'Pathological Transactions' (1874) Dr Fagge originally described these cases as Sporadic Cretinism. In this form of the disease there seems never to be a bronchocele; but in the case of a boy who came to Guy's Hospital from Halden, in Kent, a sister had goitre. In most cases the thyroid is entirely absent, and no trace of it can be discovered on dissection, a fact first pointed out by Mr Curling in 1850. In most respects sporadic cretinism is identical with the endemic form of the disease. Some of the figures which illustrate the above communications on the subject (figs. 1 and 3, pl. iii, 'Med.-Chir. Trans.,' vol. liv, and fig. 2, pl. xii, 'Path. Trans.,' vol. xxv) show how close is the resemblance between the two.

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* Virchow found that the slope of the clivus was unduly steep in one fœtus. But at an early age a steep clivus is normal; and Nièpce found that in the cretins whose bodies he examined this part was horizontal; an observation which receives confirmation from a dissection of my own. It would therefore seem that the premature closure of the spheno-occipital suture, although it arrests growth, does not prevent some of the changes in the base of the cranium which should take place during childhood.—C. H. F.

One patient affected with sporadic cretinism, at the age of sixteen years and a half was only two feet eight inches high; another, when twenty years old, was only two feet four inches high. They have the same broad, square hands and short fingers, the same dry hair, the same rough scurfy skin as we described in Swiss cretins. Their heads are large and broad; their noses are flat at the root, so that the distance between the eyes is increased; and their mouths are large and gaping, with thick lips.

In the only case in which an autopsy was made (a patient of Dr Grabham, of Earlswood) the base of the skull was much altered in shape; the posterior clinoid processes were at a higher level than the anterior, and the sella turcica was very narrow. The clivus was horizontal, and its position seemed to be part of a general elevation of the occipital in relation with the other bones, for the cerebellar fossæ were exceedingly shallow. The patient was twenty-one years old at the time of death.

The intelligence of all those affected with sporadic cretinism is more or less imperfect, and many of them present an extreme degree of idiocy, so as to be deprived of the power of speech. Their disposition is generally mild and tranquil, and in this respect they seem to differ from the subjects of the endemic disease; for Griesinger says that the latter are unsociable and repugnant to each other. Probably, however, much depends on the conditions in which such patients are placed, and the amount of care and interest bestowed upon them.

Isolated cases of cretinism have been reported in England at Oldham, at Crompton, Duffield and Matlock in Derbyshire, and in more than one place in Scotland. Langdon Down recorded twelve and Beach five English cretins. A typical one is described by Dr F. N. Manning, of Sydney, in the 'Trans. Med. Congr. of Australia,' 1889 (p. 834); and Dr Stirling (*ibid.*, p. 840) records six cases from South Australia, with characteristic photographs.

Dr Anderson, of Cape Town, informed the writer in 1894 that he has only seen one case of cretinism and none of myxœdema in South Africa.

A curious feature of sporadic cretinism is the presence on each side of the neck in the "posterior triangle," outside the sterno-mastoid muscle, of a soft, lobulated, and moveable lipoma. A characteristic case of sporadic cretinism is figured by Dr Fletcher Beach in the 'Trans. Intern. Med. Congr.,' 1881, vol. iii, p. 627, in which there was no thyroid and two large supra-clavicular masses of fat. In one of Dr Fagge's patients these supra-clavicular swellings were much larger than hens' eggs. Their size seems to be influenced to some extent by the state of the general health: thus in another case of Dr Beach's ('Path. Trans.,' xxv) they were well marked when the patient first came under observation, but disappeared entirely before death, which was caused by protracted diarrhœa.

These fatty tumours are sometimes found in adults who otherwise appear to be in good health. They were very large and well marked in a man weighing fifteen stone, who consulted the writer in August, 1887, for obesity and symptoms of alcoholic excess.

Sporadic cretinism does not depend on any external cause. It has more than once been seen in several children of the same parents. Thus, in a family of twelve, three children were cretins of an extreme type, while the rest were healthy, and one was an accomplished oarsman; they were all born in London, and their parents were living in comfort.

One of Dr Fagge's cases was that of a girl, who was stated by her relations to have been perfectly healthy until she was eight years old, when she fell ill with what was supposed to be a second attack of measles, and kept her bed for a fortnight. After her recovery her physical development underwent a remarkable change. Her features were previously well formed; they now acquired the cretinous configuration. Her hair, once black and abundant, became light coloured, dry, crisp, and scanty. She ceased to grow; at the age of sixteen years and three quarters she was only four feet one inch in height. Her hands and feet were not larger than those of a child six or seven years old. She had a fatty tumour, the size of a hen's egg, above each clavicle; and no trace of the thyroid could be discovered in front of the trachea.

Sporadic cretinism very seldom develops itself so late as this, and the endemic disease is congenital. But taken with the fact that the thyroid is congenitally absent in most cretins, it certainly suggests that the febrile illness at the age of eight years led in some way to atrophy of that organ, and this to supervention of the cretinous state.

The following case shows that congenital cretinism may be cured by the same means which we shall presently describe in the case of acquired cretinism.

In the American 'Archives of Pædiatrics' for April, 1895, Dr West, of Ohio, reports and figures a case of sporadic cretinism in a female child, the only case in a healthy family in a district where goitre was unknown. She took half a grain of dried powdered sheep's thyroid daily, and afterwards glycerine extract from the time she was a year and six months old. After six months she had the appearance of a healthy and intelligent child, with her hair grown, her colour natural, and able to sit upright.

Dr Anderson, of Cape Town, found that the administration of thyroid tablets produced marked improvement in a case of sporadic cretinism in Cape Town, and the same method has been successful in Europe.

Ætiology of goitre.—Bronchocele is not always a real increase of thyroid tissue, and may be compared to pseudo-hypertrophy of muscles, the bulky lungs of emphysema, and the large white kidney. At all events, we have seen that it is connected with cretinism geographically and by descent.

Goitre usually occurs in the valleys of a limestone formation; but drinking-water impregnated with lime salts, magnesia, or ferrous sulphide cannot be its true cause. Bronchocele is common in many mountainous countries, not only in the Alps ("Quis tumidum guttur miratur in Alpibus?" asked Juvenal), but also in the Pyrenees, in Southern Germany, in the Peak district, where it is known as "Derbyshire neck," and in elevated regions in China, in the valleys of the Himalayas and of the Andes ("Mountaineers dewlapped like bulls"). It does not occur endemically in hot, flat, or low-lying districts, or near the sea-coast.

Cretinism is far more restricted in range; but beside the sporadic cases above described, it occurs endemically in the goitrous regions of Switzerland, the Engadine, and the Tyrol; also in the Pyrenees, in Cashmere, and in Peru. It is found among the inhabitants of valleys, only because the valleys are the parts inhabited; among those who drink snow-water, only because snow-water is drunk among the mountains—for there are no cretins among the Russians, Norwegians, Icelanders, or Greenlanders,—and it is found among a goitrous population, because some unknown cause produces both diseases, the one directly, the other by successive generations gradually enhancing its effect.

Two views have been taken of the relation of cretinism and goitre. One was put forward by Dr Fagge in 1871, namely, that they are both antagonistic effects of the same cause, and that goitre is protective against cretinism; that when the cause begins to act, or acts with but little intensity, the sole effect is goitre; but that if it acts with great intensity, or upon successive generations, it at length produces cretinism as well as goitre. When goitre has existed in a family for two or three generations, the structure of the thyroid may undergo increasing deterioration in some of the succeeding generation; until families in which cretinism has prevailed undergo complete extinction within a very few years. According to the Sardinian Commission it is rare for any family residing in the Valtellina to reach a fifth generation; so that but for immigration into this valley it would become altogether depopulated. Large goitres are frequently present in the non-cretinous brothers and sisters of cretins.

The other and now generally accepted explanation of the relation between goitre and cretinism is that they are not antagonistic, but consecutive effects of the same unknown cause; that endemic goitre is not a true hypertrophy, but a degeneration of the thyroid, and that although a local disease, yet when inherited, or when supervening early, or when, most of all, the thyroid is congenitally absent, it produces widely spread and serious disturbance, so as to affect the nutrition of the whole body. We shall presently see that this view is supported by the results of experimental and surgical removal of the thyroid. But before mentioning artificial and traumatic cretinism it will be well to describe an allied condition, not endemic but sporadic, not congenital but acquired, and sometimes not developed till long after adult life is reached.

Cretinoid Condition in Adults.*—Sir William Gull first described in 1873 ('Clin. Soc. Trans.,' vol. vii) a remarkable condition which he termed *cretinoid*, and which occurs in adults—most frequently in adult women. It is characterised by a change in the features, which become broad and flattened; the eyes appear too wide apart, the alæ nasi become thick, the lips large, the connective tissue below the eyes loose and baggy, and that under the jaws and in the neck thickened. The tongue is so large as to embarrass articulation, and interfere with wearing false teeth. The hands are broad and "spade-like." The texture of the skin becomes smooth, and the hair thin, scanty, and coarser than before. The general hue is pale, sometimes of a dirty white, recalling that of the cachexia caused by syphilis, or by lead, sometimes of a clear lemon-yellow like that of Addison's anæmia; but the cheeks are most commonly rosy, not only from dilated veins, but with a diffused red as if from rouge. At the same time the disposition of the patient undergoes an alteration, activity of mind giving place to a gentle placid indifference. The temperature is almost always subnormal, and there is frequent complaint of feeling cold.

A striking case of this malady was under the writer's care in 1885. She had the coarse scanty hair, the sallow complexion, with spots of bright red on the cheeks, looking as if she painted, the subconjunctival œdema, the broad, clumsy hands, thick blubber lips, slowness in answering, and tranquil temper. The contrast with a photograph taken years before was most remarkable. Though the "bladders" under the eyes looked ready to burst, acupuncture failed to bring out a drop of serum. Another woman was in another division of the same ward under Dr Taylor, and the resemblance of the two was remarkable. In fact, the physiognomy when fully developed is unmistakable.

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* "A cretinoid state supervening in adult life in women (Gull)."—*Synonyms.*—Myxœdema (Ord), Cachexie pachydermique (Charcot), Cachexia strumipriva (Kocher), Myxœdème primitive et opératoire (Reverdin)—Acquired or adult cretinism.

The year before (1884) there was a man in Philip Ward in an early stage of the same condition. His deliberate answers, heavy aspect, and placid, ox-like demeanour were already characteristic. Afterwards œdema, spade-like hands, and the other features above described developed into a typical picture; so that when he was subsequently in St Thomas's Hospital, and was shown by Dr Stone at an examination (1887), every candidate recognised the disease at once, even though he had never seen a case.

The uniformity of aspect of this affection, whether occurring in women or more rarely in men, is not the least remarkable of its characters.

In 1892 we had in Philip Ward a well-marked case of myxœdema sent up from Twyford by Dr Fredk. Young. The patient was a man of thirty-six, and the contrast between a photograph he brought with him, taken ten years ago, and one taken since his admission was most striking. His hair was coarse and thin, the forehead bald; the whole face, neck, and body swollen and thick, but not pitting (*œdema durum*); the eyes looked small and the lips blubbered; the cheeks were of a bright red colour; the tongue and mucous membrane of the mouth little altered. The hands were characteristically broad, coarse, and short. Speech was deliberate, but not so slow as in many other cases. The temper was placid, easy, and satisfied. The thyroid cartilage, cricoid, and trachea were readily felt, and moved naturally in deglutition, but there was no thyroid to be felt.

An elaborate report on 109 cases collected from various sources was published by the Clinical Society as an appendix to the 21st volume, and forms the most complete account of the disease that exists.*

This disease differs from the sporadic cretinism above described in the absence of deformity of the bones, and in the slight degree in which the mind is affected; both these differences no doubt depend upon its late development. But it clearly depends on the same essential pathology, and chiefly differs from congenital cretinism in the fact that the thyroid is affected when the stature, the ossification of the bones, and the mental development are already completed. Accordingly we may speak of endemic and sporadic cretinism in the child, and of acquired sporadic cretinism or "cretinoid condition" in the adult; or we may (as was once proposed with the air of a great discovery), instead of calling the condition described by Gull acquired cretinism, call cretinism congenital myxœdema.

The early cases of this disease were sometimes mistaken for examples of chronic Bright's disease; in many patients there is albuminuria, and in some the other signs of granular degeneration of the kidneys, but in others equally marked the urine is perfectly normal. The apparent œdema is not ordinary anasarca, for serum does not exude on puncturing the skin. In a patient who died at St Thomas's Hospital, excess of mucin was found in the œdematous tissues after death; accordingly the name "myxœdema" was proposed by Dr Ord for this remarkable cretinoid condition in adults (1880). The condition which suggested it is not constant, for no excess of mucin has been found when sought for in other cases ('Clin. Soc. Report,' 1888, pp. 47—54), but it appears to be present in the cases to be next considered of traumatic myxœdema.

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* Among the early cases observed in England since Sir William Gull first drew attention to the subject the following may be mentioned:—Dr Ord ('Med.-Chir. Trans.,' 1878; 'Clin. Trans.,' xiii, 1880), Duckworth ('Clin. Trans.,' xiii, 1880), Cavafy (*ibid.*, xv, 1882), John Harley ('Med.-Chir. Trans.,' 1884), Drewitt ('Clin. Trans.,' xvii, 1884), Nixon ('Dublin Quart. Journ.,' Jan., 1874). Cases have also been recognised in France; and in Germany by Riess, Erb, Senator, and Landau ('Berliner klin. Wochenschrift,' 1886, 1887).

Some cases of myxœdema have been confounded with acromegaly (cf. p. 548); but the two conditions are perfectly distinct.

Artificial cretinism.—Before myxœdema was recognised on the Continent, a remarkable condition which has been called "cachexia strumipriva"[*] was observed as the result of extirpation of bronchocele by Swiss surgeons, Dr Reverdin, of Geneva (in 1882), and Dr Kocher, of Berne. After some months the patients who have survived the operation begin to show mental hebetude, pallor, œdema, and some other of the characters of an adult cretin ('Arch. f. kl. Chir.,' 1883, p. 254).

In the following year Schiff and Wagner successfully removed the thyroid in dogs, and found that the operation was followed by cerebral disturbance, tremors, and convulsions, at first clonic but then tetanic, ending in death by coma.

The same operation was innocuous in rabbits owing to the parathyroids in these animals being separated from the principal thyroid. Internal parathyroids are also present in dogs.

The most important results for human pathology are those obtained on monkeys by Mr Victor Horsley ("The Brown Lectures," reported in the 'Brit. Med. Journ.,' Jan., 1885). He found that, usually within a week after the operation, fibrillary tremors appear in the limbs, which, like those of paralysis agitans, cease on voluntary movement. The monkey becomes anæmic, with increase of leucocytes as well as diminution of red discs. It sits moping and imbecile. The eyelids and abdomen swell. The temperature falls below normal, all tremors disappear, and the animal dies comatose in from five to seven weeks.

Two remarkable conditions appear to justify the application of the word myxœdema to this condition. One is great swelling of the submaxillary and parotid glands, with changes in the latter, which bring it nearer a muciparous gland; and the other is the great increase of mucin in the connective tissues, especially the tendons and superficial fascia, and its appearance in traces in the blood. These facts rest upon analyses made by Dr Halliburton, and on similar results obtained by Professor Welch, of Baltimore ('New York Medical Record,' 1888, p. 368).

The agreement of these results with those observed in human beings after thyroidectomy, and of both with the cretinoid condition described by Gull, and with sporadic and endemic cretinism itself, go far to prove the view above taken of the relations between cretinism and goitre.

But if this is admitted and we deduce all the symptoms of congenital (endemic or sporadic) myxœdema (cretinism), of the cretinoid condition in adults (acquired myxœdema), and of cachexia strumipriva (traumatic myxœdema), alike from the absence of the thyroid secretion—there still remains the question how this deprivation can so profoundly alter the nutrition of the skin, the hair, the connective and adipose tissues, the brain, and even the skull.

We can only compare it to the wonderful and widespread effects produced by castration in animals, particularly on the skin and its appendages, on the production of adipose tissue, and on the habits and disposition. A further parallel is furnished by the remarkable fact, now established, that disease or removal of the pancreas may cause diabetes mellitus, and by the effects of destruction of the adrenals in Addison's disease. Yet another analogy is presented by Dr Bradford's remarkable observations of the far-reaching changes in metabolism produced by removal of a certain proportion of renal tissue in dogs. All of these are examples of Paget's doctrine, that every organ is in some sense excretory to other organs.

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[*] *Struma*, as the equivalent of *scrofula*, meant originally a swollen neck, and was long applied indifferently to goitre and to swollen cervical lymph-glands. In Germany it has been restricted to the former meaning, so that "struma" means a bronchocele.

The most probable hypothesis is that an "internal secretion" is furnished to the blood by some of the "ductless glands," and also by the testes, the pancreas, and possibly by the kidneys, and that the absence of this disturbs the economy so as to produce the effects described.

A point in favour of this supposition is that if a small portion of thyroid is left behind, myxœdema does not ensue; and the same applies *mutatis mutandis* to the pancreas and the kidney. Another is what we shall presently relate as to the cure of myxœdema.

The exact constitution of the secretion is at present unknown, but it appears to be contained not in the blood or in the nucleo-albumin derived from the epithelium lining of the thyroid vesicles, but in the colloid material which fills the vesicles, and is probably secreted by the epithelium. A compound containing phosphorus and iodine has been isolated, and has been found therapeutically and prophylactically active in the same way as thyroid extract.

The relation of the symptoms of exophthalmic goitre, with an enlarged and highly vascular thyroid, to those of myxœdema, will be discussed in a future chapter.

Treatment.—No drugs have any influence on the cretinoid condition in adults, though pilocarpine and other medicines used to be given in hopes of benefit. The cure of the disease depends on supplying the place of the missing thyroid. This was first proposed by Dr Murray, of Newcastle; and extract of healthy thyroid was injected under the skin, or fragments of sheep's thyroid were grafted into the peritoneum (1892). But the astonishing discovery was soon after made that when eaten and digested, thyroid glands still retain their efficacy, and the consequent treatment, first proposed by Dr Hector Mackenzie, of St Thomas's Hospital, has been found to be perfectly successful. Like others, the present writer has seen the most remarkable results from the treatment by dried thyroid extract, and has not yet met with a failure. The only drawback is that if the remedy is left off, the disease before long returns.

DISEASES OF THE

PHYSICAL SIGNS AND GENE

> **"Who knows but that, as in a watch we may bear t moving of the wheels, and the striking of the hammers multitudes of other noises—who knows, I say, but that motions of the internal parts of bodies (whether ani sound they make; that one may discover the works p shops of a man's body, and thereby discover what instr**

Percussion—History—Methods—Physical qualiti
Resonance and dulness — Tympanitic reso
Amphoric and cracked-pot sounds—Diagnost
Auscultation—History—Methods—The respirato
tubular breathing—Physical qualities—Râl
of the voice—Vocal resonance in health—
Pectoriloquy—Ægophony.
Palpation—Tactile vibration—Inspection and mea
Pulmonary symptoms—Dyspnœa—Varieties—p
Cheyne-Stokes breathing—Cough: reflex, f
Sputa—Pleurodynia.

The modern era in medicine was begun in its theoretical side by Harvey, on its clinical anatomy and physiology languished from the the third decade of the nineteenth century, uncertain progress until the close of the wa Revolution. Indeed, certain facts known to unknown at the beginning of the present cen

In the fifty years between 1815 and 186 made in the clinical investigation of disease auscultation by Laennec, the recognition of Br tion of the ophthalmoscope and laryngoscope. added the discovery of the reaction of degener electricity to the diagnosis of nervous disorders that was much earlier and far less important,— the Röntgen rays to physical diagnosis.

Of these advances the earliest and the mo duction of the physical examination of the che

This was not entirely unknown to the ancients, and Auenbrugger had made important steps in this direction; but the chief merit of introducing auscultation as a means of diagnosis is undoubtedly due to Laennec.

The mere invention of a mode of listening to sounds within the chest might have remained a curiosity, and has more than once led to learned trifling; what made it fruitful was its association with morbid anatomy. The great merit of Laennec (as of Bright afterwards) was that he constantly followed up his researches in the wards by dissection in the deadhouse. Hence, notwithstanding his premature death (by one of the diseases which he elucidated), he left the main outlines of the detection of pulmonary diseases with few omissions to supply and fewer errors to correct.

Percussion.—This method was discovered in the middle of the last century by a Viennese physician, Auenbrugger, who published in 1761 his '*Inventum novum, ex percussione thoracis humani, ut signo, abstrusos interni pectoris morbos detegendi.*' The new method, however, seems to have been adopted by Stoll alone among contemporary physicians, and it had passed into complete oblivion when Corvisart, in 1808, brought out in Paris a translation of Auenbrugger's work, with commentaries of his own, based upon extensive practice at the Hôpital de la Charité. Percussion was adopted by Laennec, and introduced into this country in 1825 by Sir John Forbes.

The way in which Auenbrugger performed percussion was by striking the chest directly "with the tips of the fingers, brought close together and stretched out straight." He also directed that either a glove should be worn, or that the patient's shirt should be kept on, so as to avoid a slapping noise. Even now the bent fingers of one hand, with their tips brought to a level, are sometimes used to ascertain roughly the state of the back of the lungs; but for more delicate percussion such a method is inadequate.

In 1828 Piorry, who was afterwards physician in the Charité at Paris, published a work upon what he termed mediate percussion. This consisted in the use of a thin plate of ivory called a *plessimètre*, to be held by the left hand in contact with the surface of the chest, while a tap was given to it with the tip of the right forefinger, or with the tips of the fore and middle fingers. A hammer, which is known as a *plessor*, was afterwards added. On the Continent these instruments are still more or less employed, and they are sometimes useful for teaching a class without hurting the patient. But in this country the usual practice is to simplify Piorry's method by using the left fore or middle finger in the place of a plessimeter. It is curious that in his original work Piorry himself speaks of that plan as having been already adopted by certain English and American physicians who had attended his lectures.

It must not be supposed that percussion is easy. On the contrary, much pains and long practice are necessary to obtain correct results: and most clinical clerks continue to make blunders in percussion long after they have mastered the difficulties of auscultation. The stroke should come from the wrist; it should be short and sudden, so as not to damp the sound which is produced, and the fingers at the moment of striking should be as nearly vertical as possible. After the stroke, a sufficient pause should follow before the next is given, to enable the ear to appreciate the qualities of the sound produced.

The amount of force that should be employed, and the extent to which the finger should be raised before striking, vary with the thickness of the soft tissues over the part of the chest which is to be struck; and every physician unconsciously modifies his manner of percussing in different patients and upon different regions of the chest as experience has taught him, in order to elicit the best possible sound under various conditions. As a rule, percussion can be practised with skilled hands without causing any painful sensation; but in delicate women, and even in some very thin men, the sternum and the ribs may be so tender that a satisfactory sound can only be elicited with difficulty. Sometimes cough is excited by every attempt at percussion, and a forcible tap may even lead to blood-spitting, so that it is well to abstain from this method of examination when there has been recent hæmoptysis. In such cases, and generally with children, it is better to begin with inspection and auscultation.

Physical characters of percussion-sounds.—It is important to know what characters or qualities of sound can be appreciated by the ear; for with hearing, as much as or even more than with sight, our supposed immediate perceptions are really judgments, the result of experience, and not direct apprehensions of the physical properties which we gradually learn to infer.

We can immediately tell little of the direction and nothing of the distance of a sound. "Distant bronchial breathing" expresses a mental conclusion; the physical sign is "faint bronchial breathing." Phrases like *large* and *small*, *moist* and *dry sounds*, are mere figures of speech, and must not be used without this proviso. Other terms are comparisons more or less apposite, which must also be adopted with caution.

The qualities or categories of sound are, so far as we are here concerned, the following:

1. *Loudness or faintness.*—This quality depends on the amplitude of the sonorous vibrations, on the excursion of the waves of sound, or, as it is sometimes put, on their height. But the comparison to the waves of water or the circles or ripples which attend the casting of a stone into a pond is misleading, for here we have a bounded plane surface from which the waves rise; whereas in the case of sound the vibration has no reference to a plane. The vibration of a tense string, as the cord of a violin, is therefore a better comparison.

Loudness in a sound then answers physically to the quality of brightness, strength or intensity in light, a quality which depends on the amplitude of vibrations of ether.

We may, if we please, distinguish between mere loudness of a single sound and the effect produced by a great number of noises, none of which by themselves are loud. It is the same distinction as is familiar in the case of visual vibrations between a single brilliant flash of sunlight and a widely diffused illumination of low degree, as broad daylight on a cloudy day. This latter condition may be represented by such terms as voluminous or massive, and is characteristic, for example, of the healthy respiratory murmur, which is the result of very slight sounds heard from innumerable points. But here pitch and duration are combined with faintness to make a composite impression.

2. *Duration.*—This character applies in two ways. A sound may be prolonged because the movement which produces it is long, as the whistle of a steam-engine; but it may also last beyond the time of the disturbance which caused it, as the sound of a bell which is heard after the stroke of

the clapper has ended. The latter kind of prolongation is generally called resonant in the popular sense, and when reinforced, *echoing.*

3. *Continuity or interruption.*—It is seldom that a prolonged sound is uniform either in loudness or pitch; it almost always wanes or fades upon the ear, instead of stopping abruptly as a very short sound does; and, except it be a musical note, it does not preserve the same pitch. If instead of growing louder up to its maximum (*crescendo*), or gradually fading into silence (*diminuendo*—"the dying fall"), it rises and falls in loudness, the effect is a broken or "rumbling" or "clattering" sound; and when we can distinguish intervals so that the sound is made up of a series of separate sounds it is called *interrupted* in contrast to *continuous*, like the sound of horses' hoofs compared with that of the wind.

4. *Pitch.*—The pitch of a sound depends on the number of vibrations in a limited time of which the ear is aware. The waves of sound are said to be short and frequent or long and slow. Pitch therefore answers physically to colour, just as loudness does to light.

5. *Quality* or "timbre" is the character by which we distinguish between two voices when singing in unison. The sound may be equally loud, equally long, continuous and uniform, and equally "true," *i. e.* of the same pitch, and yet we distinguish between two voices or between two instruments played "in tune." The difference is believed to depend on a difference in the overtones or harmonics, but this doctrine of Helmholz is not now universally accepted. Timbre is of little practical importance in auscultation; but it should be remembered that it exists not only in the spoken voice, but in the vocal sounds we shall presently describe as bronchophony, pectoriloquy, and even ægophony. Moreover, by the timbre we can plainly recognise a familiar voice in the cough though not in a whispered speech, a proof that the quality like the pitch of the voice is of laryngeal origin.

6. *Tones and noises.*—Lastly comes a distinction which is clinically the most important of all the elementary qualities of sound. Some sounds are musical, *i. e.* their overtones (or secondary vibrations) have an exact numerical relation, as multiples of the fundamental tone, and are called harmonics. Other sounds, however, and these are the majority, have overtones which have no regular numerical relation to the fundamental note; they are not always harsh or unpleasing, and are therefore better called non-musical than unmusical. They are mere noises. The distinction admits of degrees, but it is a clearly marked one, and on recognition of the presence of "tone" in a sound depends much of the skill of the percussor and auscultator.

Resonance and dulness.—In different persons one cannot always elicit the same sound by percussion of the chest; nor ought the sound to be the same over different parts of the chest in the same person. But in health the range of percussion-sounds is limited, and most of them can be arranged in a series of which the limits are called "dulness"* and "resonance."† They may be thus defined:

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* Dulness or flatness on percussion: *Sonus obscurus; Fr.* Matité; *Germ.* Dämpfung.

† Resonance or clearness on percussion: *Sonus clarus; Fr.* Son Clair; *Germ.* Sonorer oder heller Schall. Skoda used the term *leerer Schall* for dull sound in distinction from *voller Schall* for a resonant one. It is important to bear in mind that "resonance on percussion" does not mean the same thing as what is called *resonance* in acoustics. This conventional way of using a scientific term is apt to lead to confusion, and would no doubt have been better avoided; but it is so universal that it can scarcely now be altered. We shall see that the same objection applies to "consonance," as used in auscultation, and also to "tone."

(1) Resonance is louder than dulness, *i.e.* the same force of a stroke will produce a louder and further-reaching sound on an empty barrel than a full one, on a chest full of air than on a chest full of water.

(2) A resonant sound lasts longer than a dull one, and it may be so prolonged as to be called amphoric or echoing resonance.

A percussion-sound may be sometimes broken or interrupted (see below under *bruit de pot fêlé*), but as a rule it is continuous and uniform, and in this particular there is no distinction between resonance and dulness.

(3) A dull sound is higher pitched than a resonant one.

(4) A resonant percussion-note has always more or less of tone, sometimes (as in amphoric resonance and the *bruit d'airain*) a pure musical note, more often an imperfect one; whereas a completely dull percussion-sound is a noise without any tone whatever.

The most absolutely dull note is heard on percussing the fleshy mass of the forearm or thigh. Next comes that obtained by percussion over liquid effusion in the pleura. Next the percussion-note of a solid lung, of the healthy heart just below the fourth left costal cartilage, or of the liver between the right seventh and eighth ribs.

The most resonant sound obtained by thoracic percussion in health is heard where the ribs cover a thick substance of lung, as in front below the clavicles, in the axillæ, or behind below the scapulæ.

Between the extremes of dulness and resonance there are all gradations, for which the expressions "partial dulness," "incomplete resonance," "muffled resonance," or "impaired resonance" are employed. These varieties of percussion-sounds are heard in health at the borders of a resonant or dull area, especially where a thin edge of lung overlaps a solid organ. At such points many different sounds are obtained, according to the amount of force used in striking. A gentle blow elicits a sound only from the parts immediately below the spot which is struck; a more forcible blow affects deeper parts as well. Thus it is usual to speak of "superficial" and of "deep percussion." But it is well for the auscultator to remember that deep percussion is only "hard hitting," and therefore to be sparingly used. We have no power of reaching the deeper organs except by adding to the force of the stroke. A resonant area interferes with the sound yielded by a dull area, although a dull area does not interfere with the sound yielded by a resonant one. Resonance spreads, but dulness does not.

When a solid organ is overlapped by lung, it is often necessary to employ deep percussion in order to detect the limit of the solid organ. But as a rule superficial percussion should be used when the object is to map out the relative positions of the viscera. When there is disease of the pulmonary tissue, one has to ascertain by repeated trials what amount of force is necessary to bring out an altered percussion-sound most distinctly.

As a rule, it is by comparing the two sides of the chest together in the person under examination, rather than by an absolute standard, that one judges of the results obtained by percussion. But if both sides are diseased this method may fail; and even when only one side is diseased it is necessary to have some kind of standard, or one might suppose that the healthy side was too resonant instead of the diseased being too dull, a mistake often made by beginners.

Since the infra-clavicular regions are frequently the seat of phthisis and the basal regions of pneumonia or pleural effusion, the axillæ are the safest regions for obtaining the normal resonance of the lungs in any individual case; and the right side is preferable, because a distended stomach sometimes gives a tympanitic quality to the axillary note on the left.

The differences in percussion-sound at different parts of the chest in health require careful study. In front the sound is modified by the position of the liver and of the heart; and immediately below the clavicles it is more resonant near the sternum than it is further outwards away from the adjacent trachea. Behind, in the supra-scapular regions, one sometimes has to use moderate force in order to elicit anything but a dull sound. Below the scapulæ the sound is generally almost as clear as under the clavicles; and the resonant area on the left side extends about a finger's breadth lower than on the right side where the liver encroaches on the thorax.

While the back is being percussed the patient should have his shoulders drawn forwards as much as possible, crossing his arms well over the chest. In this way the "interscapular region" is increased and the "scapular" regions are diminished.

The thinner a man is, the more resonant his chest is likely to be; in very muscular or very fat persons it is sometimes difficult to elicit a clear sound anywhere, particularly over the back of the lungs.

Tympanitic resonance.—We spoke above of the percussion note under the clavicles as the most resonant sound obtained from the thorax in health. If, however, we percuss the trachea or larynx we find the sound is louder, longer, and more "musical" still, and we find the same "over-resonance" on percussing the abdomen or the inflated cheek in health, and on percussing the chest in emphysema. This degree of *intestinal* resonance is called *tracheal* or *tubular* (C. J. B. Williams). A step further in the same direction brings us to *tympanitic* resonance (like that of a drum when beaten), which is often heard over the abdomen, particularly over the transverse colon, and almost always over the stomach, as well as over the pleura filled with air. In the last case the clear resonant note is usually called *amphoric*.

Originally a percussion-sound was called tympanitic (or tympanic, *i. e.* like the note of a drum) when it was such as would be yielded by an abdomen in which the intestines are distended with gas, a condition known as tympanites since the days of Hippocrates. The sound elicited by percussion over an emphysematous lung often approaches this tympanitic quality.

Other varieties of percussion sound.—When the sternum, the clavicle, or one of the ribs is struck, a high-pitched sound is heard of what is termed an *osteal* character. This has some degree of tone, and hence prevents our getting an easy and complete dulness where the heart is covered by the sternum, or the consolidated apex of the lungs by the clavicle, or the brain by the skull. Piorry, who drew attention to this osteal percussion-sound, believed that not only the lungs, heart, and bones had each its characteristic sound, but also the liver, spleen, and other organs. There is no reason for accepting this doctrine. All the solid viscera of the body give a dull note when struck, and the peculiarity of osteal resonance depends on the remarkable elasticity of bone.

Another peculiar percussion note is that heard over a tense stomach or coil of intestine, and called by the contradictory name of *tympanitic dulness*, or by the arbitrary and confusing term "boxy resonance." This is a short, rather faint, and rather high-pitched note, which has thus lost all the characters of resonance except tone. If a drum is braced tight, the note when it is struck is short, faint, and high-pitched, but is still musical. This variety of resonance is frequently heard on percussing a distended stomach or colon. A very similar percussion note is occasionally met with under the clavicle when there is effusion of fluid in the lower part of the chest, and even occasionally when the lower part of the lung is solid from hepatisation. Its presence was first recognised by Skoda, of Vienna, and it is often called Skodaic resonance.

The *cracked-pot sound** is exactly like the chinking of coins, or the sound produced by clasping the hands loosely together and striking them upon the knee. Probably it always depends upon the propulsion of air out of a space through a more or less narrow opening. It was originally noticed by Laennec, who gave it the name.

Lastly, it must be stated that, besides the sound yielded by percussion which is audible to bystanders, the physician is himself conscious of differences in the tactile sense of resistance offered to his fingers as he taps, and that this feeling is often of considerable assistance in enabling him to draw correct inferences from his observations.

Theory of these sounds.—With regard to the theoretical interpretation of percussion-sounds there have been great differences of opinion. Even now scarcely any two writers express the same views; and the variations in nomenclature are most confusing. It is most desirable that in percussion (and also auscultation) merely metaphorical, arbitrary and descriptive terms should be avoided, and the recognised terms strictly adhered to.

The seat of the vibrations which are elicited by percussion of the chest is believed by Dr Gee to be the middle-sized and largest bronchia; following Wintrich, he thinks that the pulmonary vesicles and the bronchioles are too small to yield it. But a great obstacle to acceptance of that view is that in bronchitis resonance is unimpaired, however completely the larger tubes become filled up with pus or mucus. The percussion-sound must be partly due to vibrations of the thoracic walls, seeing that it is damped by their thickness in muscle and by their being loaded with fat or with dropsical serum. Dr Bristowe maintained that a percussion-sound is "mainly due to the vibration of the thoracic walls alone." He assumed that "so much of each half of the thorax as bounds lung-tissue vibrates bell-like when any part of that half is struck; and that the impure musical sound which is elicited comprises a fundamental tone due to the vibration of aliquot parts of it." It is a strong argument in favour of this doctrine, that deformity of the chest, without any apparent thickening of the parietes, may give rise to dulness on percussion, notwithstanding that the lung beneath is quite healthy; when there is lateral curvature of the spine, for example, the rounded projection formed by the ribs on one side of the back generally yields a dull sound. That the percussion-note, however, is not solely due to vibration of the thoracic walls is shown by the fact that the lungs taken out of the chest are resonant on percussion, and also by the resonance of the stomach and intestines, where uncovered by the ribs, and the dulness of thoracic tumours and hydrothorax, under the ribs.

The Skodaic percussion-sound above mentioned was explained by Bristowe as due to the vibrating area being diminished, and consequently yielding a fundamental tone which is raised in pitch. Or, as Dr Hector Mackenzie puts it, if the upper part of the pleural cavity contains air and the lower serum (as in hydropneumothorax), the percussion-note over the air is of higher pitch than if there were no water, and is more decidedly musical. But the slack part of the lung above the fluid effusion in cases of ordinary hydrothorax allows vibrations almost as freely as if the air were free instead of contained in the spongy lung; hence the note is again high-pitched, though less pure and musical.

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* *Fr.* Bruit de pot fêlé; *Germ.* Zischender oder klirrender Schall (Geräusch des gesprungenen Topfes).

Clinical import of percussion-sounds.—The precise practical significance of the various modifications of sound elicited by percussion of the thorax must be considered in discussing each of the diseases of the lung and of the pleura. But, in general, it may be laid down that dulness on percussion means consolidation of the pulmonary tissue, either from infiltration of its cavities, or from its compression by fluid or otherwise. As a rule, the diagnosis between the various causes of a dull note is based upon considerations independent of percussion. But the dulness resulting from the presence of liquid in the pleura is more complete or absolute than that which arises in almost any other way; and the sense of resistance is also greater, especially when the quantity of liquid is large. We must remember, however, that dulness may be due, not to any change in the condition of the lung, but to obstruction to the conduction of vibrations to the ear, due to thick, and particularly to œdematous, pleuritic adhesions, or to obesity or anasarca of the chest wall.

A tympanitic sound is either due to extreme emphysema of the lung, or, if extreme, depends on the presence of air in the pleural cavity. There is, however, a very rare affection, diaphragmatic hernia, which may simulate left pneumothorax by escape of the stomach through the diaphragm into the chest. Moreover, a distended stomach may push up a shrunken lung until what appears to be thorax yields a tympanitic note.

The diagnostic significance of the amphoric echo and of the cracked-pot sound will be fully considered elsewhere.

The student should fix in his mind that physical signs can only give evidence of physical conditions. Resonance complete, excessive, or diminished, means, more or less fully inflated lung tissue. Dulness points to solidification of the lung with or without fluid in the pleura. But neither percussion nor auscultation can tell us the chemical or physical constitution of the solids and liquids in question, still less their pathological condition. Atmospheric air or oxygen in the pleura gives the signs of pneumothorax. Serum or pus or blood or beef-tea in the bronchi causes rattles. Hence all the terms we use should be of physical not pathological import.

Before leaving the subject of percussion it is well to repeat that "dull," "resonant," and "tympanitic" are conventional terms, best defined, practically, as those elicited by percussion over the forearm, the right axilla, and the stomach respectively; but that each of these sounds, and the many others to be heard by percussion of the healthy as well as of the diseased body, consist of a fundamental tone with endlessly varied harmonic or discordant overtones, and that each note may vary in loudness or amplitude of vibrations; in pitch or number of vibrations per second; in duration; in quality or *timbre.*

Myxœdema.—An effect of immediate percussion, not infrequently seen in emaciated persons, is the production of local contractions in the muscles which receive the tap. A rounded knot rises up at the spot where the pectoralis major is struck, and from this a smaller ridge spreads away, wave-like, along the fibres in both directions. This "idiomuscular contraction" has been supposed to be characteristic of phthisis; but it occurs in most wasting diseases, when the muscles are in a condition of physiological "exhaustion."

Auscultation.—We have seen that the discovery of Percussion fell on stony ground, but after fifty years the seed sown by Auenbrugger became fruitful. His treatise was translated into French by Corvisart in 1808,* and this physician devoted himself to investigating the extent of dulness and the cardiac impulse as signs of disease. Among those who followed Corvisart's practice at the Charité were two friends, Bayle and Laennec. In endeavouring to distinguish between "active" and "passive" enlargement of the heart (*i. e.* between hypertrophy and dilatation) when percussion showed its size to be increased, they carefully observed the character of its impulse, and were in the habit of applying the ear rather than the hand to the cardiac region for this purpose. One day (about 1815) Laennec was consulted by a young woman who had the general symptoms of disease of the heart, but in whom, as she was fat, he was unable to feel the impulse with his fingers. He was hesitating to put his head to her chest, when he remembered the fact that by applying the ear to one end of a plank one can hear the scratch of a pin at the other end. So he took a quire of paper, and rolled it up tight. Then, placing one end on the præcordial region, and applying his ear to the other, he found, to his pleasure and surprise, that he could actually hear the beating of the heart more plainly than when the ear was in immediate contact with the thoracic wall. He constructed a wooden cylinder for the conductor, and soon began to employ the new method of investigation, which he termed *l'auscultation médiate*, in the study of phthisis and other pulmonary diseases. He read the first memoir on the subject in 1818, and published his great work in the following year. In 1826 he died of phthisis, at the age of forty-five, having almost worked out the subject so far as concerns affections of the lung.

Laennec was well acquainted with the morbid anatomy of the diseases to be recognised, and this was the reason why his discovery was so fruitful. He showed how to distinguish by physical signs Pneumonia, Emphysema, Pleuritic effusion, and Pneumothorax; and he defined the most important variations of the respiratory murmur as well as the chief adventitious sounds, to which he gave the general term *râles*. He, like other pathologists at that time, was not aware of the importance of Bronchitis (*catarrhe pulmonaire*) as a separate disease, and the very term was still unused; but he described the signs of œdema of the lungs and of Phthisis almost as we should describe them now, and ascertained the existence and significance of the curious sounds called "metallic tinkling" and cracked-pot sound, as well as of the tactile vocal fremitus.

It is remarkable that of the very few references to sounds heard by listening over the chest which have been traced in medical writings of an earlier date, two are in the works of Hippocrates; he mentions the leather-like creaking of pleural friction and the splash obtained by succussion. According to Dr Gee, pneumonic crepitation is mentioned in van Swieten's 'Commentaries,' published in 1774. Why Harvey or Lower or Mayow or Auenbrugger himself never thought of putting his ear to the chest, and listening to the sounds of the beating heart and the expanding lungs, is hard to understand. We see now that no special instrument was needed; Laennec's cylinder was merely a convenience, not a necessity like the ophthalmoscope and laryngoscope, and anyone might have discovered immediate auscultation at any period in the history of medicine.

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* It had been translated into Dutch and published at Dordrecht in 1788.

The stethoscope.—In examining the back of the lungs we sometimes apply the ear directly to the surface of the chest, with only a towel or the patient's night-dress to separate one's ear from his skin. In this way one can rapidly judge of the state of a large part of each lung; and it is often more convenient than using an instrument. It is, as a rule, preferable with young children, who are apt to be alarmed by the sight of the stethoscope and hurt by its pressure.

In most cases, however, we employ mediate auscultation. The stethoscope, as originally designed by Laennec (*le cylindre*), has long ago been discarded; it was long and heavy, in shape like a general's leading staff or a thick and short ruler, and it was needlessly elaborate in construction.* This was gradually transformed into a light hollow stem of wood having a flat ear-piece at one end and spread out into a concavity at the other end, which is placed upon the chest. In applying it one must be careful not to hurt the patient by pressing too hard or by bearing unevenly on one side of the rim. The ear must be moved to the stethoscope, and not the stethoscope to the ear. There is no object in the perforation except for lightness; the sound is conducted by the solid substance, not by the column of air. A very light stethoscope and one made in two parts, wood and ivory or metal or cellulose, are each so far faulty; a somewhat massive form made in a single piece of cedar is acoustically better.

Many years ago a stethoscope was introduced in which the chest-piece and an ear-nozzle were connected by a flexible tube, and it is still used by some good auscultators. It is extremely portable, and as convenient as the binaural form for auscultation of the sides and back of the chest without raising the patient. It goes by the name of Dr Alison, of Edinburgh, or of Sir Andrew Clark.

For several years, however, it has been the custom to employ a stethoscope with a flexible stem and with two long metal and ivory ear-pieces, one of which is introduced into the meatus of each ear, and is held in position by an elastic band. To the use of this double or binaural stethoscope, as it is called, everyone should accustom himself, because of the facility with which the back and sides of the chest can be explored by it in persons who are too ill to sit up. Students like the instrument on account of the loudness with which sounds are transmitted through it; but it is not certain that what reaches the ear is always as clear and distinct as when a single stethoscope is employed, and the slightest movement of the flexible part of the binaural stethoscope produces noises which are apt to be perplexing. Other disadvantages of the instrument are that it is cumbersome to carry about, that the least contact with the patient's dress causes a loud noise, that it is almost useless unless applied directly to the skin, and that the natural sounds, as estimated by immediate auscultation, are exaggerated. Its advantages are its convenience of application to many parts of the chest, the absence of pressure on the patient's skin, the exclusion of other sounds by both ears being occupied, its allowing the auscultator to see while he is listening (not always an advantage), and its making respiratory and particularly cardiac murmurs easier to hear.

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* The specimen in the Royal College of Physicians in London is about ten inches long and rather more than an inch thick. It is perforated and divided into three parts, so as to make it half the length if desired, and to allow an "obturator" to be taken out or left in.

A kind of double stethoscope with flexible leaden arms was devised by the late Dr C. J. B. Williams, but abandoned as cumbrous and inconvenient. It consisted of two sections joined together so as to make it longer or shorter at pleasure, and the ear-piece was flat. He adds, "It has occurred to me that both ears might be advantageously employed in stethoscopic examinations," and describes an instrument like the modern double stethoscope. The modern form was, the writer believes, devised in Philadelphia, apparently by more than one physician.*

The double stethoscope is best used to determine the existence and character of slight or doubtful cardiac murmurs; it is useful for listening to the trachea and vessels of the neck, to the lungs above the scapulæ, and to the abdomen, and it or the single flexible stethoscope is almost indispensable for listening to the lungs of a recumbent patient.

For examining the heart and the front of the chest without removing a patient's underclothing, the ordinary wooden stethoscope is the most suitable instrument, and should always be used to confirm or modify the results obtained by the flexible stethoscope (whether single or double), particularly in cardiac cases and in auscultation of the voice.

For listening to the back of the lungs while the patient is standing or sitting up in bed, no form of mediate auscultation is so convenient as the application of the ear to the patient's chest covered by a thin towel.

Whatever variety of stethoscope may be employed, one must take care that the patient's clothes do not rest against it nor against the surface of the chest near the spot to which it is applied; and that he is not moving his hands on his chest. Another point to be kept in mind is, that if the instrument is placed on the hairy part of a man's chest a crackling sound is often produced, like what will be presently described as crepitation: this difficulty may be avoided by smearing a little oil over the surface.

AUSCULTATION OF THE BREATHING.—On listening over the lungs of a healthy person, one hears with each breath a soft, rustling, breezy sound, followed by a second one of similar quality but of shorter duration and fainter. It is a noise without tone, not loud as a rule, and sometimes faint, but in health, uniform and of low pitch. The only way of really learning to know it is to hear it again and again and in many different individuals. It is commonly called the *vesicular murmur*, having been so named when its seat was supposed to be in the air-vesicles of the lung; but this association with an unsound theory makes the term objectionable. Walshe proposed "pulmonary respiration sound" instead. "The normal *respiratory murmur*" is perhaps a still better term.† The former part of this sound accompanies the act of inspiration; expiration is usually attended by the shorter and fainter murmur, almost continuous with the inspiratory, but is often noiseless in perfectly healthy persons. The expiration sound is much shorter than that of inspiration.

In some cases the healthy respiratory murmur is much louder than in others. In children it is particularly loud, so that when under morbid conditions an equally loud murmur is heard in an adult, this is sometimes called "puerile breathing." In thin healthy adults it is often scarcely less loud. On the other hand, there are some people in whom the act of breathing is attended with scarcely any sound, even when one tries to make them breathe deeply, and these are often athletic men with what are called powerful lungs. One must therefore listen over different parts of a patient's chest before one draws conclusions from the degree of loudness of the murmur at a single spot. It is naturally louder where the thoracic walls are thin than where they are covered with thick muscular masses, and over the edges of the lung it is fainter than elsewhere; in fact, its intensity in health is generally proportionate to the degree of resonance on percussion at various parts of the chest. In the same way it is feeble when the walls of the chest are thickly covered with fat.

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* In a letter published in the 'Lancet,' Aug. 29th, 1829, Dr N. P. Comins, of Edinburgh, described a flexible stethoscope which he used in the Royal Infirmary of that city

† *Fr.* Souffle respiratoire, souffle vésiculaire; *Germ.* Normaler Lungengeräusch.

At the bases of the lungs, especially in persons who are confined to bed by whatever cause, the respiratory murmur is sometimes heard mixed with a crackling sound, which may be mistaken for crepitation, but which disappears when the breath is drawn deeply two or three times in succession; it is probably due to slight collapse from disuse, and has no clinical significance.

There are certain spots at which in many healthy persons the breathing is not accompanied by the normal respiratory murmur. They are (1) the space between the scapulæ over an area of variable extent, but somewhat lower in situation on the left side than on the right; (2) the region below each sterno-clavicular joint, especially on the right side and in females, and the part corresponding to the spines of the seventh cervical and first dorsal vertebræ. The sound heard over these parts is in many healthy persons *bronchial* in character, because it is transmitted direct from the main bronchi without being damped by conduction through spongy lung. It differs from the vesicular murmur in having a blowing character, in the expiratory part being as loud as the inspiratory, and in there being an interval between them. A similar sound is heard more loudly and constantly on auscultation over the trachea, and still more loudly over the larynx; here it is of a more "whiffing" quality and harsher, and is distinguished as "tracheal," "*tubular*," a term, however, which some writers use as synonymous with bronchial.

To be able, in practice, to distinguish a loud or harsh vesicular murmur from true bronchial or tubular breathing is the most important step in auscultation. For the latter sound is heard in several diseases at parts of the chest far removed from the main bronchi, and is one of their chief signs. In bronchitis bronchial breathing does not occur, but it may accompany any disease in which the lung is either compressed, or consolidated, or hollowed into cavities. In other words, its range is generally conterminous with that of percussion-dulness. Bronchial breathing is not by any means always louder than the "vesicular" murmur, but it has a blowing quality, it is higher pitched, and has more of tone; while it is less high-pitched and less musical than sibilant rhonchus.

The modifications of bronchial breathing concern its quality. A blowing character belongs to them all; and they all consist of an inspiratory part and of an expiratory part, separated by an interval. One distinguishing point is that the blowing sound is more or less hollow. In its most extreme form it has an echoing character, and resembles the sound produced by breathing into a large empty glass bottle; it is then named *amphoric*, or echoing tubular breathing.

A less marked degree of the same quality of sound is often called *cavernous*, because it is commonly heard over vomicæ which are sometimes spoken of as caverns in the lung; but the term is superfluous and misleading. A vomica may often yield consonating râles, and not amphoric breathing at all. Moreover, the names of physical signs should refer to their physical characters, and not to the anatomical conditions they denote, especially when their significance is not constant.

When bronchial breathing is "whiffing" but without a "hollow" quality, it is by most writers named *tubular*, although others, as above mentioned, employ "tubular breathing" and "bronchial breathing" indiscriminately.[*]

It must be understood that between these several modifications of bronchial breathing all gradations exist, so that it is often difficult to decide whether to call a sound bronchial or tubular, tubular or amphoric. Moreover there are undoubtedly gradations to be heard between the respiratory murmur of health and bronchial breathing, which have been called "vesiculo-bronchial" or "transitional" or "indeterminate."[†] But such doubtful sounds are rare, and soon develop into bronchial or subside into vesicular. Indeterminate terms and non-committing statements are dangerous resources; and it is therefore well to maintain as far as possible the distinction between tubular breathing and vesicular on the one hand, amphoric on the other.

It is a good rule to use the more striking epithets sparingly. The common errors of beginners are to mistake a loud or harsh respiratory murmur for bronchial breathing, to call bronchial breathing tubular, and tubular breathing amphoric.

Theory of breath-sounds.—As to the physical causes of the respiratory murmur, of bronchial breathing, and of its various modifications up to amphoric, there has been controversy. The most probable theory is that of the *veine fluide.*[‡] According to this a blowing sound is generated whenever a fluid (whether liquid or gas) passes suddenly and with sufficient momentum (*i. e.* for the same fluid, velocity) from a narrow space into a much wider one.

Dr Fagge with many others believed (chiefly from Chauveau's experiments) that while the sounds heard on listening over the trachea are produced at the glottis, those heard over the greater part of the lungs are produced where the bronchioles open into the lobules. This theory, however, has not been confirmed by other experiments, and there is always a doubt as to application of results from one animal to another.

Another theory, which appears to the present writer better supported by the facts of healthy and abnormal respiration in man, is that all the respiratory murmurs, whether "vesicular" or "bronchial," are produced chiefly, if not entirely, by a fluid vein formed at the larynx. The vibrations are transmitted by the walls of the trachea and bronchi to the lungs, and thence, if the lungs are solid, to the ear of the auscultator, unchanged in rhythm and but little in loudness, so as to produce the tubular ("bronchial," tracheal, or "laryngeal") breath-sounds, expiratory as well as inspiratory, which are characteristic of pneumonia. If, however, there is a fluid in the pleural cavity, the transmission of these sounds is as a rule stopped, hence the absence of any respiratory sound in most cases of hydrothorax or empyema. In health, precisely the same sounds are transmitted, though imperfectly, through the spongy lung, and reach the auscultator's ear greatly modified, as follows: the expiratory laryngeal murmur formed above the glottis is more or less completely lost; the inspiratory murmur is no longer blowing, it has lost its hollow, quasi-musical quality of tone, and it is fainter; it thus assumes the distinctive features of normal pulmonary or "vesicular" breathing.

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[*] *Fr.* Souffles bronchique, bronchiolaire, tubaire, glottique, soufflante, caverneuse, amphorique; *Germ.* Bronchialgeräusch, amphorisches Athemgeräusch.

[†] *Unbestimmtes Athemgeräusch* answers to the "vesiculo-tubular or subtubular" breathing of some English and the *respiration rude broncho-vésiculaire* of some French authors.

[‡] This is a physical, not a physiological term, and refers to the slender line of coloured water which is seen when solution of litmus is injected through a narrow orifice from a syringe into a large glass tube. The comparison is to a vein of metal.

An older theory than either of these is quite untenable—the theory, namely, that the respiratory murmur in health is produced by the entry of the inspired air into the air vesicles during inspiration (whence the inaccurate term vesicular murmur) and its exit during expiration. It is certain that no current takes place here; the residual air of the lungs is tranquil, and is renovated only by diffusion. According to the same theory, tubular or bronchial respiration is produced in the larger bronchi when the air vesicles are occluded; but surely we cannot suppose that there is any current in a tube which opens into solid lung, for there is no aspiration and nowhere for the inspired air to go.

Much the same arguments apply to fixing the seat of the inspiratory part of the pulmonary murmur at the opening of the bronchioles into the lobules, for there also it is difficult to believe that the velocity of aspiration is sufficient to cause audible vibrations—even if the other conditions of an audible *veine fluide* were present.

Stokes long ago observed that when the larynx is diseased it is often difficult or impossible to determine the condition of the lungs; and extensive ulceration of the vocal cords would prevent the formation of a *veine fluide* in the air which passes the glottis, and so render impossible the development of "bronchial" breathing in a solid lung below.

Of the modifications of bronchial breathing, from tubular to amphoric, all that need be said in this place is that the more marked the hollow echoing quality, the more one is justified in asserting that a large space filled with air is present either in the lung itself or in the pleural cavity.

There remain two terms employed as descriptive adjectives of the respiratory murmur which must be noticed. One is "vesiculo-tubular," or transitional, applied to a sound intermediate between normal breathing and bronchial (p. 1032). Such gradations exist, but they are not common; they are, as has been said, evanescent, and their use is apt to lead to inaccurate observation. The other is "harsh," applied to the respiratory murmur heard in cases of early phthisis, and of broncho-pneumonia in children. Probably it is either supplementary breathing in the unaffected lung, or it is a râle or bronchial breathing in the affected one. The same term harsh, as often applied, is identical with *puerile, i. e.* the loud breath-sound heard in a healthy child's chest. The pulmonary murmur is a noise, and all noises when loud are harsh or unpleasant to the ear.

Adventitious respiratory sounds.—The sounds derived from auscultation hitherto described have been identical with, or modified from, those that can be heard on listening over the healthy lungs or air-passages.

There are other sounds which are adventitious, and have no physiological representatives. Thus in pleurisy a friction-sound is heard, which will be discussed in a subsequent chapter.

In affections of the lungs and bronchi adventitious sounds are heard, of which the nomenclature is unfortunately much confused. Some writers, following Laennec, describe them all indifferently as "Râles,"[*] a term which was translated "rattles" by those who introduced his discovery into this country; or as "Rhonchi,"[†] a term used as synonymous with râles by many writers in Germany to denote any adventitious pulmonary sounds.

Continuous sounds.—In England the term *rhonchus* has been long used in a restricted sense, which seems to have been first given it by the late Dr Latham, of St Bartholomew's Hospital. In that sense it is limited to continuous, "dry" sounds, or to a single variety of dry sound. This is what is called sonorous rhonchus (Laennec's *râle sonore*), the other dry sound being aptly termed *sibilus* or sibilant rhonchus (*râle sibilant*). *Stridor* is a rhonchus so loud as to be audible at a distance.

The difference between the two is mainly one of pitch. Sibilant or whistling rhonchus (or sibilus) is high-pitched, and more or less musical; sonorous rhonchus (or rhonchus without qualification) is low-pitched, and not much more than a noise. The word means a snore, and rhonchi are often described as snoring, cooing, creaking, or stridulous. As a rule the higher-pitched, whistling sounds, are found in the smaller tubes, the deeper-toned rhonchi in the larger ones—in the primary bronchi, the trachea, the larynx, the fauces, or the naso-pharynx.

The mechanism of all these sibilant and other "rhonchi" is the same. A fluid vein is formed by swelling of the mucous membrane, or by accumulation of glutinous mucus so as to narrow the lumen of the air-passage, and so inspiratory and expiratory vibrations are produced.

The common and perfectly distinctive character of all these adventitious sounds is that they are *continuous*, like the pulmonary and tracheal murmurs, and like the murmurs of the heart, which also are the result of the formation of a *veine fluide* (p. 1032). They are often called "dry" sounds; but this only means that the mucus in the tubes is not abundant enough to occlude them and form bubbles as the air passes through it; and the term gives a false notion of their causation, for they are often heard when a patient is expectorating abundance of moist secretion. They may also be heard when narrowing of an air-passage is caused by a foreign body in a bronchus—by a tracheotomy tube, by a partially occluded glottis, by pressure of a lymph-gland or a tumour, or by cicatrices in any part of the upper respiratory tract.

Rhonchi are audible with inspiration or with expiration only, or with both. Those of expiration are often the louder.

Interrupted sounds.—From these continuous sounds must be broadly separated those which in England are called râles or "moist sounds" or "rattles." They are interrupted, not continuous, and are caused by a totally different mechanism from the *veine fluide* of rhonchi, namely, by air passing through mucus (or pus, or blood, or water), and making bubbles which burst, and thus cause a series of audible explosions. Whatever term is used, it is clear that the two kinds of adventitious sounds should be carefully distinguished. Bubbling or crackling or "moist" sounds would be correct enough, but each term has unluckily been confined to a particular kind of râle.

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[*] The French word *râle* or *rasle* corresponds to the German *Rasseln*, and our *rattle*.

[†] Rhonchus (ῥόγχος or ῥέγκος, snoring) is Laennec's *râle crépitant sonore*; Germ. Knarren, Schnurren.

Râles are heard with inspiration or expiration or with both, but expiratory râles are the exception.

When a patient is lying insensible with stertorous breathing, we often hear a grunting, sonorous rhonchus change into a large, bubbling râle as the mucus accumulates and completely, instead of partially, obstructs the passage of the breath.

Râles are somewhat arbitrarily distinguished according to the supposed size of the bubbles which produce them, which infers that of the spaces in which they are formed. We judge of this mainly by the number of crackles or explosions in a unit of time. Five or six during inspiration denote the trachea or a large bronchus as their seat; twenty or thirty point to a small bronchus. Moreover the "larger" râles are lower in pitch than the "smaller."

The gradation is sometimes made thus: *fine* (or small)—*submucous*—*mucous* râles, and then *gurgling;* but "mucous" is a bad epithet, for the fluid causing the râle may be pus or blood. "Small," "medium," and "large" râles are sufficiently descriptive terms.

In some cases râles, like rhonchi, are audible without the aid of the stethoscope. In fact, the "rattle in the throat" is only a large râle.*

Consonance.—There is another distinction between different kinds of râles which is of far greater importance than that of their apparent size, for it indicates whether the tubes in which they are found are surrounded by spongy or by solid lung. In the latter case they have a peculiar quality which in the former is wanting, and which the ear can be taught to recognise. We may call the râles heard when the lung is solid "bright," "clear," "musical," "ringing," or "metallic." Skoda called them "consonating" râles, but we must not assume that their peculiarities depend upon *consonance* in the strict acoustic sense of the term.† The character which we recognise by this word and the adjectives quoted above is that of possessing more or less tone or musical quality—like resonant percussion-sounds (p. 1024), or sibilant rhonchi (p. 1034).

The sounds which are termed crackling or crepitant râles (or crepitation) and subcrepitant râles are "smaller" (*i. e.* the interruptions are more frequent and shorter) than those of gurgling; and they differ from toneless or non-consonating râles of the same "size" or degree by their "metallic" or "musical" quality. One particular kind of râle, almost (if not quite) peculiar to an early stage of acute pneumonia, is termed *fine crepitation.* This sound is consonating and typically "bright" or musical, and it is never perfectly heard except when the lung is hepatised. It sounds less "moist" than any other râle. It will be more fully described and its origin discussed in the chapter on pneumonia.

Consonating râles are clinically found associated with dulness on percussion, with bronchial (tubular) breathing, and with the vocal sign called bronchophony.

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* Some of the râles heard in disease have been attributed to the sudden separation of surfaces that had been in contact. In 1871 Traube applied this view to explain the râles heard in the larger tubes by the momentary detachment of portions of viscid mucus from their sides, by the current of air in respiration. A point to which he drew attention is that a râle can be produced by pressing gently with the stethoscope upon the surface of a healthy lung of a recently killed animal; and Wintrich showed that a like result may be obtained by inflating the collapsed lungs after death ('Virchow's Hdbh.,' Bd., v, Abth. 1).

† We have seen that the same objection applies to our use of the terms "resonance" and "tone." But after all Laennec wrote before Helmholtz; and it seems impossible to invent suitable substitutes for these inaccurate terms, or if invented to get them adopted.

Non-consonating râles, chiefly heard in bronchitis, need no further designation than large, small, or medium, *i. e.* few or many, as above explained. They are clinically associated with resonance on percussion, with more or less harsh or coarse pulmonary murmur, and sometimes with rhonchi.

AUSCULTATION OF THE VOICE.—If one applies the stethoscope to the windpipe of a healthy subject, whether man, woman, or child, and bids them speak, one hears the words distinctly, with the pitch and timbre of the voice, but much more loudly and with a clang or resonance. If one listens to the chest while a healthy man speaks, we generally hear an indefinite humming or buzzing noise. In a woman the sounds come to the ear more clearly though less loudly. In neither case can the words that are uttered be distinguished. Only over the upper part of the interscapular region and in the first intercostal space on the right side is there in some persons a space where one can hear the voice clearly, though less loudly than when one places the stethoscope over the trachea.

When the lung is solidified it will convey the voice to any part of the chest distinctly, and more loudly than is ever the case in health. This is called "increased vocal resonance" or *Bronchophony*.

To appreciate vocal resonance, it is convenient for the patient to count, loudly, with a bass tone, and with a pause between each syllable—*staccato*. The pitch of the voice, the timbre, and the vowel sounds can all be recognised,—*i. e.* all the vibrations produced in the vocal cords are transmitted to the ear of the listener distinctly, and much louder than when heard without a stethoscope. The same loudness is heard (and felt) when the ear is placed immediately on the speaker's chest, and is also recognised in the inarticulate but vocal expiration of a crying child. The consonantal sounds, which are produced in the fauces and buccal cavity, are unheard or too imperfectly heard to inform the auscultator of the words pronounced.

Just as the normal vocal resonance may be increased until it becomes bronchophony, so it may be diminished until it can no longer be heard at all—or felt. This is due to impaired power of conducting the vocal vibrations, as bronchophony is due to a heightened power of conduction.

Laennec did not distinguish between bronchophony (a later devised term) and what he called *pectoriloquy*. This latter word he defined as hearing the patient's voice through the stethoscope as if he were speaking directly out of the chest into the ear of the auscultator,—*i. e.* the consonants as well as the vowels can be distinguished.

> "En me livrant à des recherches comparatives relativement à la résonnance de la voix chez plusieurs sujets sains et malades, je fus frappé par un phénomène tout à fait singulier. . . . . Lorsque, tenant le cylindre appliqué au dessous de la partie moyenne de la clavicule droite, je faisais parler le malade, sa voix semblait sortir directement de la poitrine et passer tout entière par le canal central du cylindre. Ce phénomène n'avait lieu que dans une étendue d'environ un pouce carré. Dans aucun autre point de la poitrine on ne trouvait rien de semblable" ('De l'auscultation médiate,' tom. i, § 22).

This curious sign will be better appreciated if we make the patient speak in a whisper, because then there are no loud vocal sounds to interfere with our hearing those produced in the mouth. Hence pectoriloquy is usually sought for as "whispering pectoriloquy;" but that is not the original meaning of the term.

Laennec laid more stress upon auscultation of the voice than of the breathing; the first section of his great work is headed "Exploration de la Voix." Pectoriloquy was the firstfruits of his great discovery, hence, as Dr Gee remarks, it is not surprising that he always clung to this sign with peculiar affection. It became his object to define it in such a way that it should become an unerring indication of a vomica. For this purpose he added to the characters of what he termed perfect pectoriloquy that of being limited to a very small part of the chest. But subsequent experience has shown that, like other "hollow" sounds, the most typical pectoriloquy may in exceptional instances be heard when there is no large cavity within the thorax. Thus pectoriloquy no longer carries the exaggerated importance which Laennec attached to it; indeed, some writers omit it altogether, and include all degrees of increased vocal resonance under the term bronchophony.

Bronchophony, as a rule, is associated with bronchial or tubular breathing or with consonating râles; pectoriloquy with tubular or amphoric breath-sounds, or with metallic gurgling. In other words, bronchophony attends those conditions in which the pulmonary tissue is solid; pectoriloquy is heard when, in addition to solid lung, there is a neighbouring empty cavity. Occasionally tubular breathing and bronchophony may be heard over a part of the lung consolidated by the pressure of fluid in the pleura. Diminished vocal resonance is due to a layer of liquid over the lung, or to very fat or œdematous parietes, which damp the vibrations of the voice.

Bronchophony and pectoriloquy depend on the vibrations in the glottis or mouth being partially damped by spongy lung-tissue, and transmitted almost unchanged by solid lung. The same explanation applies to bronchial breathing, consonating râles, and the loud transmission of the cardiac sounds to distant parts of the chest. The complete physical explanation has perhaps not yet been given,[*] but the differences in the sounds under discussion are readily appreciable by every one who takes the trouble needful to educate his ear; they mutually check and confirm one another; and long experience has given us confidence in assigning to them a diagnostic significance on which we can depend.

A curious modification of vocal resonance was named *ægophony* by Laennec, from its resemblance to the bleating of a goat.[†] It will be met with again as a sign of pleural effusion.

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[*] Laennec was content to assume that spongy lung was a bad conductor of sound. Skoda, as the result of experiments on the dead tissues, declared the conductivity of hepatised lung to be *less* than that of the healthy organ; but he could not reproduce the conditions under which auscultation is practised during life, and it is difficult to believe that a homogeneous material does not convey sound better than a series of spaces containing air. He maintained that *consonance* is the cause of bronchial breathing and bronchophony, and adduced the effect of the sounding-board of a violin, and the increased sound produced by a tuning-fork when it is placed upon a table; but these are examples of "resonance," not "consonance."

[†] "*De l'égophonie ou de la pectoriloquie chevrotante.*—La voix plus aiguë, plus aigre que celle du malade, et en quelque sorte *argentine*, produit seulement une illusion telle qu'il semble que quelqu'un parle dans la poitrine du malade. Elle a, d'ailleurs, un caractère constant d'où j'ai cru devoir tirer le nom du phénomène; elle est en quelque sorte, tremblotante et saccadée comme celle d'une chèvre, et son timbre se rapproche également de celui de la voix du même animal. Ce caractère ne présente que des variétés légères dont on peut se faire une idée exacte en se rappelant l'effet que produit un jeton placé entre les dents et les lèvres d'un homme qui parle, celui de la voix transmise à travers un roseau fêlé, ou le bredouillement nasal des bateleurs qui font parler le fameux personnage de tréteaux connu sous le nom de *polichinelle*" ('Ausc. méd.,' tom. i, § 154).

Its physical explanation is now generally referred to suppression of the fundamental note and lower harmonics of the voice.[*] The case was thus stated by the late Dr Stone, in his 'Croonian Lectures,' 1879.

If the patient was made to sing or to intone a good musical note, no ægophony was to be heard. Among spoken words a difference was found according to the vowel sounds they contain: the French A yielded hardly any ægophony; it was more marked with the E, still more so with the I, and most of all with U. Now Helmholtz showed that the different vowel sounds are formed by the addition of certain harmonic overtones in varying degrees of intensity to a fundamental tone which may be the same for all of them. According, then, to Stone the cause of ægophony is that the fundamental tone is intercepted in its passage through a layer of pleural exudation, while the overtones are allowed to pass and, being heard by themselves, give the peculiar character to the sound.

When the effusion in the pleura is large, vocal resonance is absent, and also ægophony; the harmonic overtones, as well as the fundamental tone, of the voice are both cut off.

Dr Frederick Taylor has since published a valuable paper on this difficult subject which will be found in the 'Medico-Chir. Trans.' for 1895, vol. lxxviii, p. 127. Together with Fagge, Bristowe, and Douglas Powell, he accepts Dr Stone's hypothesis, but maintains further that the peculiar "twang" of ægophony depends on the presence of discords or dissonance; and that this is due to beats occurring between the higher harmonics, and becoming audible from the reinforcement of the higher, and the suppression of the lower along with the fundamental note. These beats are further strengthened by resonance in the bronchial tubes, and the result is the peculiar, high-pitched, nasal, tremulous, discordant sound which is heard in ægophony—as in the bleat of a sheep or a goat, and in the feigned voice of "Punch," to both of which Laennec compared it. Dr Taylor, however, does not accept any constant relation of this physical sign to pleural effusion; and here he is supported by observations of his own and of other physicians, of ægophony being present in pneumonic consolidation with only dry pleurisy. He records a case of this kind in which no pleural effusion was found after death; and ascribes the ægophony to compression of the bronchia by a profuse pneumonic exudation, which, like pleural exudation, makes them resonate (in the strict sense of the term).

Ægophony is one of the most curious and striking of physical signs. It is recognised at once, and is almost always evidence of effusion in the pleura—almost, we must say, as of every "pathognomonic" sign.

PALPATION.—By applying the hand to the chest we are able to compare the extent of movements in respiration of the two sides and of the upper and lower parts of the same side. This use of the sense of touch corrects or confirms the observations of the eye.

Moreover, when the hand is placed upon the bare chest of a healthy man a tremulous sensation is felt when he speaks, especially if the voice is powerful and low in pitch. The sensation closely resembles that of the vibration felt by the hand when placed on the side of a purring cat, and was named *frémissement cataire* by Laennec. In fact the two sounds are produced in precisely the same way. In a woman or a child this tactile fremitus is often not to be perceived, and on the right side it is often more distinct than on the left.

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[*] After Laennec's and Skoda's attempts to explain the ægophony, M. Luton of Reims brought forward a theory of this kind in Jaccoud's 'Nouveau Dictionnaire,' published in 1866, quoted by Dr Taylor in the above-mentioned paper.

This normal sensation is known as tactile vibration of the voice, or *vocal fremitus*. It is increased when the lung is solidified, and is diminished or abolished when the lung is separated by liquid from the thoracic walls. In other words, it is increased under the same conditions as vocal resonance on auscultation, and diminished or absent when the vocal resonance and respiratory sounds are enfeebled or lost. In estimating tactile vocal fremitus the whole hand should be laid flat on the patient's chest, and he should be directed to speak loudly, slowly, and in as deep a tone as the compass of his voice will allow. As with other physical signs of the chest, comparison of the two sides is invaluable for bringing out the diminution or exaggeration of tactile vibration.

The practical importance of loss of vocal fremitus seems to have been first pointed out in the 'Journal Hebdomadaire' for 1829 by the French physician, Raynaud, who also discovered tactile friction in pleurisy.

Inspection.—It is well, when a complete examination is to be made, to begin by carefully inspecting the thorax as the patient stands or sits upright in as easy a posture as may be. The light should be made to fall first full, and then obliquely on the chest. The shoulders should be covered by a shawl while the front is examined. The number of respirations in a minute should be counted; their rhythm, particularly the length of expiration and the degree of pause between expansion and contraction should be noted; and the comparative movement of the chest and abdomen (denoting the predominance of thoracic or phrenic respiration) should be observed. Next the chest should be looked at in front, behind, laterally, and by looking down upon it from above the patient's head as he sits, for the eye to measure its relative dimensions, and to compare the movements of the two sides.

Bulging or flattening of one of the infra-clavicular regions is also easily detected by standing behind the patient while he is sitting, so as to look downwards over his shoulders. A general enlargement of one side is often easily appreciable by the eye; but one must make quite sure that the patient is sitting or standing perfectly upright, especially if the case is that of a child or of a young woman with a flexible spine. In infants one may grasp the chest with the two hands from behind, placing the thumbs tip to tip upon one of the vertebræ. In adults a measuring tape is often used. In measuring the chest the tape should be placed just below the angles of the shoulder-blades, and brought forward just below the nipples to meet over the sternum. The mean circumference obtained by measuring during tranquil, *i. e.* phrenic respiration, is the most useful; but full inspiration and expiration give another, the degree of expansion, and this is important in judging not so much of disease as of vigour and endurance.

As Dr Gee remarks, circumferential measurements are apt to be fallacious, because considerable increase in the sectional area of one side of the chest may leave the length of the periphery unaltered, by "the passage of the elliptical form into the circular." It is this which renders his *cyrtometer* so useful an instrument.* It is made of two long pieces of very narrow metal gas-tubing, of an eighth of an inch in diameter, which are fastened together by a short piece of caoutchouc tube slipped over their ends. The junction is placed over the spinous process of a vertebra, and the hollow metal rods are then carefully bent round the patient's body, so as to meet over the sternum. It is now easy to remove them without altering their shape; and by laying them upon a sheet of paper one can obtain an accurate tracing, which shows exactly the configuration of the two sides of the chest, and enables them to be compared. One must not forget that the half circumference of the chest on the right side is in many healthy persons greater than that on the left side, the difference being sometimes as much as an inch; this depends on active use of the right arm.

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* Somewhat similar cyrtometers were invented by Woillez and by Björnström, and are much used in France and Germany.

Various instruments, called *stethometers*, for the measurement of the movements of the chest have been devised by Gibson (1848), Quain (1858), and other physicians. One of the best is Dr Arthur Ransome's, of which a description may be found in the 'Medico-Chirurgical Transactions' for 1873.* But although they have yielded information as to the exact degree of impairment of mobility of different parts of the chest wall in various diseases, it seems doubtful whether any one of them has been employed in actual practice by other observers than its inventor, the reason being that they are troublesome to use, and that they bring to light few facts that may not be ascertained without them.

A similar remark applies to the exact mensuration of the several diameters of the chest carried out by means of calipers, with a graduated quadrant, devised by Woillez, Wintrich, and other physicians.

Nor does it appear that in clinical practice any valuable results can be attained by the use of an instrument invented by Dr John Hutchinson many years ago for the purpose of measuring the amount of air that can be expelled from the chest by the fullest possible expiration. This instrument, called the *spirometer*, was described in the 'Med.-Chir. Trans.' for 1846. There are great practical difficulties in obtaining correct results: for few persons succeed in "blowing" their full amount of air into the instrument until they have had some practice, so that it seems to have completely disappeared from insurance offices.

Common symptoms.—There are certain symptoms which belong to most diseases of the Chest, and which are most conveniently considered here. They are Dyspnœa, Cough, and Pain, and to these we will add the characters of the Sputa.

DYSPNŒA.—The use of this term is commonly limited to cases in which a sensation of "shortness of breath" is experienced, together with more or less discomfort or distress. When the respiratory movements are deeper than natural, or more frequent, or both deeper and more frequent, without the patient being conscious of the disturbance, it is better to describe the condition as frequent or exaggerated respiration.

It is a remarkable fact that persons affected with phthisis and other chronic extensive diseases of the lungs, may continue to breathe as slowly as in health, and with no more effort, so long as they are at rest. The amount of oxygen supplied to the blood is no doubt much reduced; but it suffices for the wants of the system. The body adjusts its requirements to its necessities, and the reason why persons affected with emphysema or asthma almost always grow thin is that they instinctively learn to take very little food. A still more important method of adjustment is the avoidance of all bodily effort. So soon as such a patient begins to walk, especially on rising ground, dyspnœa sets in; for muscular exertion at once demands more oxygen than is contained in his arterial blood. So, again, the supervention of pyrexia in a case of this kind leads at once to a disproportionate increase in the rapidity and in the depth of the breathing; while on the contrary, as Cohnheim remarked, we see the subsidence of dyspnœa after the crisis in acute pneumonia before the affected lung has begun to clear.

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* See a series of tracings obtained by another instrument, the stethograph, in 'Allbutt's System [illegible], pp. 828-9, 832-4.

Another form of dyspnœa results when enough blood cannot reach the lungs, as in disease of the heart; and a third, when the blood is deficient in hæmoglobin, so that there is no means of conveying the oxygen to the tissues, as in anæmia.

Sometimes very marked dyspnœa arises without evidence that the oxidation of the blood is defective. This is the case, for example, in diabetic coma, with its air hunger; and a distressing shortness of breath may be the earliest symptom of chronic Bright's disease. Probably in diabetic and uræmic dyspnœa the bulb is irritated by poisoned blood. Another kind of dyspnœa is often ascribed to hysteria; but, like other hysterical symptoms, it is not always accompanied by other signs of that disease. This nervous dyspnœa is really only rapid and shallow breathing, without cyanosis.

Of renal dyspnœa a striking instance once came under Dr Fagge's notice. He was seeing his out-patients, when the attendant nurse asked him to listen to her chest, because her breathing had become difficult. After careful investigation, nothing amiss with either the lungs or the heart could be detected. Then, as she said she was thirsty, her urine was examined for sugar, and the result being still negative, it was tested with nitric acid, which brought down a large quantity of albumen. A few months later dropsy set in, and her case soon ended fatally.

Of acute respiratory affections, mechanical obstruction of the air-passages by foreign bodies, by laryngeal disease, and by diphtheria, are attended by the most marked and urgent dyspnœa; of chronic affections it is most marked in cases of emphysema, asthma, and syphilitic stenosis of the larynx or trachea. The difficulty is chiefly inspiratory in laryngeal obstruction, chiefly expiratory in asthma, and both in emphysema.

Phrenic dyspnœa.—There is a peculiar form of dyspnœa which depends upon paralysis of the diaphragm. Its characters were first recognised by Duchenne. So long as the patient is at rest his breathing is perfectly easy, but the slightest effort at once distresses him and quickens his respirations; when he walks he experiences a sense of suffocation as soon as he has made a few steps; in mounting a staircase, and even in speaking, he is obliged to stop every instant to take breath. When he sighs he feels as though the abdominal organs were being drawn up into his chest, and the act of defæcation is much embarrassed. His voice is weak, and there is difficulty in coughing and sneezing, because he cannot take the preliminary deep inspiration. Hence, even a slight attack of bronchitis is attended with danger. If one looks at the surface of his body while he breathes, the characteristic indication of paralysis of the diaphragm is generally at once apparent. During inspiration, when the ribs rise and the chest expands, the epigastrium and the hypochondriac regions are drawn in; during expiration they are pushed forwards. In other words, their relation to the thoracic movements is exactly the reverse of what it is in health. Sometimes it is not so easy to see the alteration as to feel it, with the two hands placed just below the cartilages of the ribs. If only one side of the diaphragm is paralysed, as is sometimes the case, one hypochondrium is drawn in, while the other one protrudes naturally.

Among the examples of this affection recorded by Duchenne, there are some in which it appeared at an advanced stage of progressive muscular atrophy, and others in which it was associated with paralysis of many other muscles as the result of lead-poisoning. Walshe saw it well marked as a sequel of diphtheria. Erb cites Oppolzer as having observed it at the age of puberty without any cause being discoverable. Another cause, quoted by Duchenne from Aran, is extension of inflammation from the peritoneum or from the pleura; and he gives a case of empyema in which the muscular tissue of the corresponding side of the diaphragm was of an orange-yellow colour, and the fibres had undergone fatty degeneration.

When the diaphragm is paralysed, it appears still to retain its power of responding to faradic stimulation of the phrenic nerves.

The best method of stimulating these nerves is, according to Duchenne, as follows:—By two fingers, placed just outside the edge of one sterno-mastoid muscle, the skin is at first drawn slightly inwards; they are then separated, leaving between them an interval, upon which a small conical metal rheophore is pressed, so as to be just over the spot where the phrenic nerve lies upon the scalenus anticus. The rheophore is now given to an assistant to hold, and the same procedure is repeated on the opposite side of the neck. When both rheophores are fixed the operator takes one in each hand. He passes through them a current, which should instantly give rise to a contraction of the diaphragm, shown by the abdominal walls being pushed forwards, while the lower ribs are separated. Sometimes, however, the platysma interferes with this result, contracting with such force as to jerk the rheophores out of position; and sometimes it is necessary to shift them a little before one can succeed in acting on the phrenic nerves.

Erb recommends a different method; he places one pole upon the neck, and the other over the attachment of the diaphragm to the costal cartilages. Whatever position may be adopted for the rheophores, the stimulus should be so used that the resulting contraction of the diaphragm may fall in with the natural respiratory movements. The current should be stopped as soon as the muscle has acted, and a few seconds after it should be reapplied.

This procedure seems to be of therapeutical value. By its means Duchenne succeeded in restoring the functions of the diaphragm in a man who had advanced progressive atrophy of other muscles, but in whom the phrenic paralysis was as yet recent.

Orthopnœa.—There is a kind of dyspnœa in which there is no distress and little acceleration of the breath so long as the patient is either standing or sitting up in bed; but so soon as he lies down difficulty in breathing is apparent.* That the diaphragm descends more freely when the weight of the liver and other viscera draws away from it, and that both sides of the chest act better when the pressure of the body does not hamper them, either on one side or behind, is readily understood. It is as natural as the increase of dyspnœa which follows movement, or ascent of rising ground. But it is not easy to explain why the advantage of an upright position is so much more marked in cases of cardiac dyspnœa than in those of bronchitis, or phthisis, or anæmia. As a clinical fact, however, the marked form of orthopnœa in which a patient sitting up in bed is able to converse easily, but is quite unable to lie down, and can only sleep propped up with pillows or in an arm-chair, is rarely seen except in cases of organic disease of the heart. It may sometimes be observed in emphysematous patients, and as a complication of dyspnœa is frequent in spasmodic asthma, in ascites and renal dropsy, in grave anæmia, in advanced phthisis, and in cases of pleural effusion or pneumothorax.

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* "Ægri recto corpore residere cupiunt, qui habitus est ad eam rem aptissimus." —Aretæi, 'De causis et signis acutorum morborum,' lib. ii, cap. 1.

Cheyne-Stokes respiration.—This is the title given by German writers to a curious variety of dyspnœa first noticed by Dr John Cheyne, of Dublin; and afterwards described by Professor Stokes, who regarded it as a symptom of fatty degeneration of the heart. It has also been called tidal, cyclical, or periodic dyspnœa. An attack usually, but not always, begins with dyspnœa; this is followed by a gradual slackening of the respiratory movements until they fail altogether, and a complete pause ensues for half a minute or even longer. This condition of apnœa is succeeded by slow and shallow breathing, which gradually quickens and deepens until it resumes its former character. The whole process is of variable length, and is usually repeated several times within a few hours; often it recurs during several days or even for weeks and months.* The total interruption of the breath, gradually led up to and in like manner gradually recovered from, is the essence of the phenomenon.

The physiology of Cheyne-Stokes breathing has been discussed by Traube, Schiff, Filehne, Luciani, Wertheimer, and many other writers. It may be observed in rabbits and other animals after considerable hæmorrhage; and is seen clinically in cases of apoplexy, meningitis, and cerebral tumours, in the uræmic state, and in the *coma vigil* of extreme anæmia and of exhaustion from fevers. It may occur during chloroform narcosis, or as a result of the morphia habit. It often comes on while the patient is asleep or comatose, but sometimes when he is in possession of his faculties, walking about, and fully aware of the peculiarity of his breathing. In other cases he will be sitting reading in a chair, and go through the curious process with apparently no sense of discomfort. The writer has seen both conditions, one in a man of about thirty-six in the hospital for Bright's disease, the other in an old gentleman of eighty, who afterwards developed signs of chronic renal degeneration, but at the time was apparently only anæmic and feeble from age. In this case the Cheyne-Stokes breathing lasted, with occasional intermissions, from August, 1890, to his death in May, 1891.

The pupils usually become contracted during the height of the fit, as in other conditions of apnœa, but this is not constant; the pulse is quickened n the ingravescent stage as the number of respirations is increased, and subsides with them, until the pause follows, when it becomes slow and ncompressible.

There is little doubt that the immediate cause of this curious phenomenon is some change in the respiration-centre of the bulb. It is seldom or never observed in uncomplicated diseases of heart or lungs, and rarely ccompanies marked orthopnœa or severe dyspnœa. It is a symptom of ervous origin, and probably connected with deficiency in oxyhæmoglobin f the blood supplying the bulb. Its relation to fatty degeneration of the eart is perhaps rather that both are results of anæmia than that a feeble rculation causes it, for it is very rare in cases of cardiac failure from alvular disease. Its rhythmic character may be compared with the ythmic rise and fall in blood-pressure, described by Traube and Hering.

As a prognostic symptom Cheyne-Stokes breathing is very grave. Unoubted cases of recovery after it has appeared have been recorded, but as rule it only comes on in the latter stages of cerebral disease, of anæmia, uræmia, or cholæmia, and points to exhaustion of the nervous centres and to approaching death.

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* Dr Samuel West published a remarkable case of Cheyne-Stokes breathing in the ancet' for 1890, p. 545; and Dr J. D. Mann reported one prolonged for more than a r ('Brain,' 1890, p. 178).

Cough, as is well known, always begins with a deep inspiration; the glottis is then closed, and, a sudden expiratory effort being made, the glottis is allowed to open, causing a loud sound, and allowing a blast of air to pass out, carrying with it mucus, blood, or any foreign body present in the air-passages. Laryngeal disease may modify the characters of cough, giving it a hoarse, or rough, or metallic quality, or rendering it almost noiseless; in fact, a well-sounded cough may almost be taken as an indication that the larynx is not seriously diseased.

The nervous mechanism by which cough is effected is reflex. As a rule, the irritation which gives rise to it starts from the respiratory mucous membrane, and leads to expectoration. But sometimes the most violent and repeated efforts of coughing bring away nothing. The cough is then said to be dry (*βήξ κενή—tussis sicca*).

The air-passages may contain mucus too viscid and too firmly adherent to be expectorated; or some part of the respiratory tract is slightly inflamed, so that the surface is sensitive to the passage of air over it, or to the disturbance produced by speaking. The most common and, so to speak, physiological cough, is that which results as a reflex action from a foreign body in the air-passages or from any irritation felt as "tickling" about the glottis or fauces. Sometimes, however, the starting-point of cough is outside the air-passages, and this fact is of practical importance.

The following appear to be the chief kinds of cough which are not due to affections of the respiratory tract.

1. *Throat cough.*—In animals as well as in man, irritation of the pharynx produces cough in many individuals, but not in all. There is therefore no theoretical difficulty in admitting that catarrh of the fauces may be attended with cough, without there being a corresponding affection of the larynx; but the parts being continuous it must always be difficult, if not impossible, to say that this is actually the case, especially as the glosso- and arytæno-epiglottic folds and the lateral edges of the epiglottis are among the most sensitive structures. It is a different question whether an œdematous and elongated uvula gives rise to cough by coming into contact with the parts behind the base of the tongue. It is generally believed, and many uvulæ have been removed to cure a cough for which no other cause could be discovered. But while this treatment sometimes succeeds, its failures are also frequent.

2. *Ear cough.*—That cough can be excited by irritation of the external auditory meatus has been known long. Dr Cornelius Fox ('Lancet' for 1867) related the case of a patient who experienced a feeling of irritation of the larynx and had a violent suffocating cough whenever he introduced a toothpick into the left ear; while long-continued singing would cause him pain in the ear. Again, a healthy-looking woman, aged fifty, had for eighteen months a most distressing cough; as she was deaf in the right ear the meatus was examined, and was found to contain a hard plug of cerumen, and to have a small ulcer in its floor. Almost immediate relief to the cough followed extraction of the wax and the application of nitrate of silver to heal the ulcer. In a patient of Toynbee's a cough was cured by the removal of a piece of necrosed bone from the external meatus; and it is probable that a foreign body, such as a bead in the ear,

may set up a cough. The afferent nerve in all such cases appears to be the auriculo-temporal branch of the fifth.

3. *Tooth cough.*—Dentists tell us that the stump of a tooth may be the starting-point of a cough, and a cough in infants during the first dentition has been known to cease after the gums have been lanced. In investigating an obscure case, therefore, the teeth should not be forgotten.

4. *Stomach cough.*—In the last century it was believed that dry cough is often produced by disorder of the digestive organs. As often happens in like cases, what has long since ceased to be taught by the faculty has become an article of faith with the public. Thus coughs in children, which are really due to catarrh of the upper air-passages, are still commonly referred to the stomach. Or patients set down to the same cause the dry cough of early phthisis, attended (as it often is) with nausea and loss of appetite and pain in the side. Kohts failed altogether to excite cough in animals by irritating the stomach. He cites from 'Brücke's Physiology' a case in which a boy coughed day and night until he vomited, whereupon the cough at once ceased; but he adds that Brücke himself believed the starting-point of the affection to have been, after all, something in the air-passages, which became dislodged when the stomach expelled its contents.

A patient of Professor Leyden had repeated attacks of biliary colic, and every time became affected with dry cough and with pain in the right hypochondrium twenty-four hours before the jaundice set in. Walshe says that he has known the trifling irritation due to the presence of an *Ascaris lumbricoides* keep up reflex cough for several weeks.

5. *Pleuritic cough.*—We are probably right in regarding the dry, short, frequent cough of pleurisy and pleuro-pneumonia and phthisis as reflex. It accompanies the pain and the friction-sounds of pleurisy, and disappears with effusion or when bronchitis supervenes.

6. *Nervous cough.*—Kohts found that he could sometimes excite cough in animals by mechanical or electrical stimulation of the floor of the fourth ventricle, and he thinks that the centre for this reflex act is situated somewhat higher than that for respiration. In hysteria, as is well known, a hard, dry, barking cough is common, and this may be supposed to be centric in its origin.

A remarkable instance of this was recorded by Dr John Harley in the 'Med. Times and Gazette' for 1863. The patient, a girl aged fourteen, uttered a short bark seventy times a minute without intermission, so that, according to calculation, she must have coughed 40,000 or 50,000 times in the course of the day. She had had the cough a fortnight when she came under observation. She was treated with valerianate of zinc and with a cold douche and friction to the spine, and in three days the cough ceased.

A very similar case, in a child aged eight, was described by Dr Whytt more than a century ago under the name of nervous cough. A remarkable feature in each of these cases was that the cough ceased as soon as the patient lay down.

Another kind of nervous cough is the short, frequent cough of embarrassment, which may be compared with the fidgeting, twirling the thumbs, playing with a button or a chain, and tapping with the foot or finger under the influence of self-conscious emotion.

Sputa.—The old distinction between dry and moist coughs is a valid one. When there is no secretion, the cough is either "reflex" or "nervous," or it is due to local inflammation in the early stage. The natural course of catarrhal inflammation, as seen in the eye and the mucous membrane of the mouth, is for a dry condition to be succeeded by increased secretion of mucus, at first thin, then more tenacious, and finally, when "concocted," as the old physicians called it, muco-purulent. The expectoration is an important symptom (or physical sign) of the kind of secretion going on in the respiratory tract.

The sputum consists of mucin in alkaline saline solution, combined with leucocytes. When the latter predominate it is purulent. Air is more or less abundantly mixed with it, and frothy blood may be present. Saliva frequently furnishes the greater part of the material in the spitting-cup. Fibrin, forming casts from the larynx, trachea, bronchi, or minute bronchioles, is sometimes obviously present, or may be detected by the microscope. Epithelial cells, usually buccal, fragments of elastic tissue, and great variety of microbes—saprophytic or pathogenic—may be detected with more or less preparation. Elastic fibres show that destructive processes are at work—Dittrich's plugs, Charcot's crystals, and Curshmann's spirals will be referred to hereafter (p. 1072).

Clear mucous expectoration points to an early stage of bronchitis and tracheitis; muco-purulent to a later stage of the same, or to the bronchitis of phthisis; abundant frothy untenacious sputum to bronchorrhœa; pure pus to rupture of an empyema, or if foul and expectorated at intervals, to bronchiectasis. Expectoration of pure blood, if disease of the heart and aorta and of the blood itself is excluded, is an almost certain sign of phthisis; tenacious sputum with blood tingeing it "rusty," golden-yellow, red, or green, is the characteristic sign of lobar pneumonia; while mere streaks of blood usually come from the larynx or fauces. Nummular sputa are frequently seen in phthisis, but are far from characteristic of it.

It must be remembered that children under ten very rarely expectorate, and up to thirteen or fourteen, as well as in old age, the same inability is often met with. In such cases, when the contents of the stomach are vomited, the presence of characteristic sputum may sometimes be recognised.

Pain is a symptom of various thoracic diseases, but it may also be referred to the chest without other indication of disease.

Walshe speaks of "pains deeply felt within the chest, and shooting in the direction of the pulmonary branches of the vagi and sympathetic," as existing "independently of any other deviation from health, not only local but general." He also refers to "various anomalous and more or less painful sensations, felt deeply within the chest by phthisical patients." These may be called pulmonary neuralgia.

But in the large majority of cases thoracic pain is referred to the walls of the chest, and especially to the infra-mammary or infra-axillary region. Various names are given to pain in these situations, according to the views held with regard to its nature. This "pain in the side" may be called "pleurodynia," "thoracic myalgia," or "intercostal neuralgia."

It is sometimes difficult to determine whether a pain in the side is due to pleurisy, anæmia, mitral disease, or gastric disturbance. The stomach seems to be sometimes its starting-point when it is on the left side, which is most often the case. The same is true of the pleurodynia of cardiac disease and of chlorosis. Another frequent cause is ovarian irritation, especially in hysterical women. Caries of the spine, too, must be thought of, and we must remember that a pain in the side, if recent, may depend on an attack of shingles. Nor must we forget the possible presence of disease or injury of a rib.

In July, 1877, I was consulted by a lady, the wife of an old schoolfellow, who told me that, having had a cough all the previous winter, she had one night felt something crack in her left side while she was coughing. Ever since then she had suffered from a continuous gnawing pain there. On examining the side there was considerable enlargement of one of the lower ribs, which seemed to be clearly the callus of a fracture. She sometimes felt pain in the side towards night when fatigued, and changes of weather seemed to increase it.

In a lecture reported in the 'Lancet' for 1882, the late Mr Marshall related the case of a woman aged thirty-five, who in the severe weather of the spring of 1881 caught cold, shivered, and was attacked first with pain in the left side and then, a month later, no less severely in the right. Mr Marshall found two firm oblong swellings, one along the lower border of the right fifth rib, and the other at a corresponding spot on the left eighth rib. When they were pressed upon she experienced acute pains shooting through to her back. They gradually softened into abscesses and were opened, when parts of each rib were found to be eroded and softened. Ultimately some pieces of dead bone came away, and she recovered well.—C. H. F.

Tuberculous and syphilitic periostitis must also be borne in mind, although both are less common in the case of the ribs than of the sternum.

In describing the diseases of the Chest, the order followed will be—Bronchitis, with pulmonary collapse, Emphysema and Bronchiectasis; Pleurisy, Empyema, and Pneumothorax; Pneumonia, Broncho-pneumonia, and Cirrhosis of the lung, with pulmonary Syphilis and Gangrene; pulmonary Tuberculosis or Phthisis, and, lastly, the functional disorders of respiration known as Spasmodic Asthma and Hay-fever.

BRONCHITIS

"Forte si tussire occeperit, ne sic tussiat,
Ut cuiquam linguam in tussiendo proferat."
PLAUTUS, 'Asinaria,' iv, 1.

History—General symptoms: cough, pain, expectoration, dyspnœa—Physical signs: rhonchus, sibilus, râles—Diagnosis—Morbid anatomy in recent and prolonged cases.
Acute Bronchitis—Capillary Bronchitis—Its symptoms and prognosis—Pulmonary collapse—Its production and relation to broncho-pneumonia.
Chronic Bronchitis—Symptoms—Varieties—Sequelæ.
Emphysema—Its anatomy and origin, expiratory and inspiratory—Atrophic emphysema—Symptoms and signs of emphysema.
Bronchiectasis, fusiform and saccular—Fœtid bronchitis.
Ætiology, prognosis, and treatment of bronchitis generally.
Plastic bronchitis—Its rarity—Anatomy, course, and symptoms—Treatment.

Definition.—An inflammation of the mucous membrane of the air-passages, particularly of the trachea, bronchi, bronchia, and bronchioles.

The disease now called bronchitis* is very common in our climate, and the name familiar, but it does not appear in medical literature before its introduction in 1812 by Peter Frank in Germany, and in 1814 by Badham in England. Previously the disease was known as "a cough," "a catarrh," or "a defluxion on the breast." Sydenham separated the more severe forms from pneumonia as "peripneumonia notha."

The upper limit of bronchitis may be taken as the glottis, for in the most frequent form of catarrhal bronchitis the larynx and trachea are involved, and the Greek term βρόγχος denoted the larynx as well as the trachea. Laryngitis, however, is a necessary term for several forms of local disease, even though, as in diphtheria, the process seldom stops in the larynx, but descends to the trachea and bronchi. The lower limit is probably best fixed at the entrance of the bronchial tubes into the lobules, excluding the intralobular air-passages, and is marked by the columnar ciliated epithelium and muscular fibres. Most cases of bronchitis are tracheitis, with secondary inflammation of the larger bronchial tubes.

When the disease is severe and extensive, it is often called "capillary bronchitis," but there are no bronchioles so minute as the largest capillaries, and it is probable that most of the cases so designated are really cases of bronchitis complicated by lobular (catarrhal) pneumonia.

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* The word is formed, like so many others, on the analogy of pleuritis. Derived from βρόγχος, the windpipe, bronchitis properly signifies any disease of that organ. But by usage the termination carries with it the notion of inflammation, as in peritonitis, orchitis, and enteritis.

The term Bronchitis, without qualification, is usually applied to *catarrhal* inflammation, and does not imply either plastic exudation or ulceration. There are many forms of bronchitis; and these differ, both in symptoms and in course. We will, therefore, first enumerate the symptoms and physical signs which belong to all alike, and afterwards give separate accounts of the more important varieties.

Common symptoms.—Foremost among the general symptoms is *cough.* This is never absent, and it is often severe, with a loud, barking, or ringing character. It may consist of isolated explosions, succeeding one another more or less regularly, and sometimes with extreme frequency. Or it may occur in paroxysms, which often end in retching or actual vomiting. It becomes more troublesome when the patient lies down, and often comes on when he first gets up in the morning, owing to accumulation of secretion in the air-passages during the night.

Many patients complain of a raw, rough, or sore feeling behind the sternum; and the cough may give rise to more or less myalgia in the thoracic walls. Not infrequently this muscular pain is felt at the epigastrium; but another cause of epigastric oppression is fulness of the right side of the heart or sometimes, perhaps, of the liver, from obstruction to the venous circulation. The irritation which sets the cough up is often definitely referred to some one spot along the course of the trachea; sometimes there is only a vague tickling sensation, which cannot be localised, and often the cough causes a sharp cutting pain in the fauces for the moment. But the chief distress of bronchitis is not pain, comparable in severity to that of pleurisy, colic, or neuralgia, but inability to sleep or even to lie still, and difficulty in relieving the irritation by free expectoration.

The *sputum* in Bronchitis—if we except the remarkable variety known as plastic or fibrinous—is tolerably uniform in character. At first it is absent, then thin mucus collects, with occasional pellets of viscid consistence; gradually this contains, instead of a few mucus-corpuscles (small uninuclear leucocytes), numerous large granular cells with tripartite nucleus; and as these increase the secretion becomes looser, more abundant, and more yellow, and is called muco-purulent. As the catarrh subsides, the secretion diminishes again in amount, loses its yellow colour, and acquires the character of large pellets of mucus of iron-grey colour from the particles of carbon inhaled wherever the smoke of chimneys defiles the air.

Another symptom in all severe cases of bronchitis is *dyspnœa.* It depends on the mechanical obstruction in the small tubes to the entrance of air into the pulmonary vesicles.

Riegel seems to have first drawn attention to the fact that in all affections of the bronchioles the dyspnœa is expiratory rather than inspiratory. Sometimes the act of inspiration is quite short and easy, while that of expiration is difficult and much prolonged; sometimes they are both embarrassed; but inspiration appears never to be alone impeded in bronchitis, as it often is in affections of the larynx or trachea. This special tendency to expiratory dyspnœa, when the smaller air-tubes are inflamed, can scarcely depend on paresis of the muscular fibres of the bronchia, since their contraction is but slow.

Another peculiarity of the breathing, which may often be noticed in children, is that each expiration is instantly followed by an inspiration, the pause in the act of breathing taking place at the end of each inspiration and not at the end of expiration, as it does normally.

In some severe cases there is *orthopnœa*, so that the patient must be propped up with pillows, but this is usually an indication that there is emphysema beside bronchitis.

When dyspnœa is considerable there is more or less lividity or *cyanosis*. This shows itself in the face and hands. In extreme cases the face becomes turgid, flushed, and bloated; the veins of the neck are dilated and throb with each pulsation of the right ventricle; and the superficial veins generally are fuller than natural. This condition of venous congestion contrasts with the pallor which also accompanies the dyspnœa of phthisis, of diphtheria, of cardiac and of renal disease.

Physical signs.—The signs of bronchitis are less numerous than those of almost any other disease of the chest. They are mainly auscultatory, for the percussion-sound is quite unaltered unless the case is complicated with pleural effusion or with a secondary complication, as emphysema, or collapse, or broncho-pneumonia.

The pulmonary murmur is but slightly altered in character, if at all; but it is often replaced by adventitious sounds. In some cases, and those not the least grave, the respiratory sounds become faint and indistinct; or occasionally they may be absent over a part of the lung, from the corresponding bronchial tube being plugged by mucus. In this case one can usually bring back the breath-sounds at that point by making the patient cough.

Sometimes in bronchitis the inspiratory murmur becomes louder and perhaps rougher in quality, and expiration may be accompanied by a similar sound. This is sometimes erroneously called bronchial breathing. The latter has a blowing character, possesses tone, and is strictly limited to certain parts of the chest; moreover bronchial breathing, except over the sternum and between the scapulæ, is accompanied by more or less dulness on percussion. It cannot be too strongly impressed on those who are learning the use of the stethoscope that neither bronchial breathing nor any of its modifications occur in uncomplicated bronchitis. Nor is the vocal resonance in any way altered.

The adventitious sounds which occur in bronchitis are those that have been already discussed, under the names of "rhonchus" and "sibilus" (p. 1034), and "râles" (p. 1035). *Rhonchus* (or "sonorous rhonchus") is a loud, continuous snoring, grunting, or cooing noise, often audible by the patient himself and by those about him, and due to vibrations that can often be felt by the hand placed upon the surface of his chest. It is formed in the trachea and larger bronchi, and its cause is the presence of a mass of viscid mucus partly obstructing the entrance of air, and producing a *seiser fluide*. The proof of this is that it can very generally be made to disappear, at least for a time, by the patient's coughing. Indeed, it comes and goes of its own accord, being heard first in one part of the chest-and then in another, as mucus happens to accumulate in different branches of the bronchial tree.

Sibilus (or "sibilant rhonchus," as some prefer to term it) is a high-pitched, continuous, whistling sound. It is formed in the smaller bronchi and is of graver import than rhonchus, since inflammation of the smaller air-passages is more dangerous than that of the larger. It seems to be due to narrowing of the calibre of the affected tubes, either from partial obstruction by mucus, or from swelling of their lining membrane. In either case, it cannot be got rid of by coughing, and usually remains in the same spot for hours or days altogether.

Many terms have been employed to denote the particular quality of these continuous "dry sounds."* Sometimes they resemble "snoring," as the term *rhonchus* would imply, sometimes the hoarse "cooing" of a wood-pigeon; often they are high-pitched and musical like a box of pipes, sometimes deep in tone, like the bass notes of the organ, and sometimes "wheezing," "squeaking," or "whistling."

These *râles*, or interrupted additional-sounds, which accompany bronchitis, are decidedly less frequent and characteristic than the "dry" sounds, and may be of every variety, from gurgling or "bubbling" *râles à grosses bulles*, to small submucous rattles. But they are never "consonating," inasmuch as the lung-tissue round the tubes in which they are formed still remains spongy. If râles are not universally distributed through the lungs, they are, as a rule, most marked over the lower lobes, and behind rather than in front. Signs that might suggest the presence of bronchitis in the upper lobes only—especially if limited to the upper lobe on one side—should always arouse a strong suspicion of phthisis.

It is worthy of mention that sometimes no râles can be detected in cases in which the profuse expectoration would certainly have led one to expect them—only dry sounds are audible.

Diagnosis.—The symptoms and physical signs above enumerated are not sufficient to justify a final diagnosis of bronchitis. We must add certain negative points by which the presence of other affections of the air-passages or lungs is excluded. Particularly in cases of which the clinical history makes it not impossible that the lung may contain scattered tubercles, great caution must be exercised in forming an opinion. Sometimes, but very rarely, the occurrence of secondary nodules of a malignant new growth in the lungs offers another chance of error. The absence of more than slight pyrexia, the absence of blood-discs from the sputa, and the absence of consonating râles, are some of the negative facts which point to bronchitis.

The difficulty is not so much in saying that bronchitis is present as in determining whether it is the primary affection, or only a complication. For it is exceedingly apt to arise in the course of a great variety of diseases—as measles, enteric fever, rickets, phthisis, organic lesions of the heart, and Bright's disease.

Morbid anatomy.—In mucous membranes, as in the skin, the morbid appearances produced by inflammation are far less conspicuous after death than during life; and a reason why this is eminently the case with the bronchial mucous membrane is afforded by the abundance of elastic fibres in its structure. In many acute cases there is redness and swelling of the mucous membrane, with a velvety appearance; in many every part of the air-passages, up to the trachea, is filled with a yellow or brownish opaque secretion. But it is sometimes far from easy to determine the presence of bronchitis at an autopsy. A good method of detecting muco-pus in the smaller tubes is to slice off the edge of the lung, and press the tissue towards the cut surface, when a yellow bead appears at each little orifice.

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* As above explained (p. 1045), the terms "dry" and "moist," as applied to sounds, are misleading, and the metonymy is almost absurd, but usage is very difficult to alter.

The histological changes in bronchitis have been carefully studied, but they are not always marked. Socoleff ('Virchow's Archiv,' vol. lxix) examined artificial bronchitis in dogs and in rabbits produced by insufflation of bichromate of potass or of a weak solution of chromic acid. Hamilton ('Practitioner,' 1879) studied cases in the *post-mortem* room of the Edinburgh Royal Infirmary. Both observers are agreed that a very early change is the detachment of the ciliated epithelium, which seems to be thrown off in flakes, and which remains absent during the whole course of the disease, to be regenerated when recovery takes place. In a young man who died of opium-poisoning, in from ten to sixteen hours, the ciliated cells were already to a great extent shed. Many undergo fatty degeneration; others, no doubt, are expectorated; others are inhaled into the smaller air-tubes, where they may be seen lying in large detached masses among the other catarrhal products. There is an obvious analogy between this exfoliation of the columnar layer of the bronchial epithelium and desquamation of the cuticle from inflammation of the skin.

During the further progress of the attack the basement membrane is covered only by a layer of flat cells, from which there project here and there pyriform or oval corpuscles, of transitional character, which are covered by a more or less abundant mass of leucocytes, embedded in a mucoid fluid. A point on which Hamilton lays stress is that the basement membrane itself becomes thickened and swollen, apparently from œdema.

In all but very early and very slight cases of bronchitis the mucous or submucous tissues are, in their whole substance, more or less filled with leucocytes, which collect in lines along the lymph-spaces between the fibrous bundles and around the vessels. Both Hamilton and Socoleff are convinced that the leucocytes which appear in such large numbers upon the free surface of the mucous membrane are not derived by emigration from the blood-vessels, but are formed by germination from the flat cells that lie immediately in contact with the basement membrane. Socoleff's chief reason for maintaining this opinion was that in animals killed twenty-four hours after the commencement of the morbid process, he found leucocytes on the free surface of the mucous membrane, although its substance was at that time entirely free from them. Hamilton insists on the difficulty which leucocytes derived from the blood would have in traversing the thickened basement membrane, and on the fact that in his preparations he could discover no indication that this was taking place. But it is perhaps worthy of notice that Socoleff himself figures ciliated epithelial cells having in their interior red blood-discs, which must have made their way through. And one naturally hesitates to accept observations made upon deep-seated tissues, as overthrowing the results of investigations made upon the cornea and other superficial structures (cf. vol. i, p. 45).

An important change occurs in the mucous glands. They become swollen so as to be sometimes as large as hemp-seeds, according to Riegel. Their epithelium undergoes very active proliferation, and the newly-formed cells become distended with mucin, and appear to be the source of the mucus that forms a large part of bronchitic expectoration. This mucus, however, becomes mixed with leucocytes and epithelial cells of "transitional" form, as well as with saliva and air-bubbles.

When bronchitis has existed for a great length of time before death, the changes found after death are somewhat different. The mucous membrane is often pale and grey, with but few vessels visible. In many cases it presents a number of delicate longitudinal ridges, which Rindfleisch has shown to consist of an overgrowth of connective tissue, containing numerous cells, and bundles of elastic fibres running in various directions. According to Hamilton the muscular coat is sometimes found to be hypertrophied, sometimes atrophied. The cartilages shrink and disappear, undergoing the same change which occurs in articular cartilage, namely, the absorption of the matrix from the periphery inwards, with the formation of "medullary spaces" filled with leucocytes. In many cases the mucous glands also are destroyed. At an earlier period their orifices are widely dilated, giving the mucous membrane a minutely pitted appearance when looked at with a good light; and sometimes they become inflamed, forming minute funnel-shaped ulcers.

Acute Bronchitis.—The ordinary "idiopathic" inflammation of the trachea and larger bronchial tubes is clinically part of the catarrh or "cold" affecting the respiratory organs, which is one of the commonest accidents of the colder and damper climates.

The throat (*i. e.* the fauces) is commonly the first to feel the effects of a "chill;" then comes coryza and inflammation of the nasal passages, often spreading upwards into the frontal sinuses and conjunctiva. Before this "weeping" stage is over, the larynx is affected, as is shown by hoarseness or aphonia, and then the catarrh spreads downwards.

As in the other regions, the mucous membrane is probably at first dry and "irritable," next swollen and hyperæmic, and then abundant mucus is poured out with relief to the "raw feeling" and painful dry cough. Gradually more pus cells appear in the expectoration, the cough becomes looser, and the patient easier.

In a sharp attack the cough is often very distressing, and especially violent when the patient attempts to lie down; he complains of a raw sensation behind the sternum; and pressure upon the trachea may not only be painful, but may at once excite cough.

The violence of the cough often causes severe headache, increased with each paroxysm, and the patient's sleep is seriously impaired. This form of the disease, however, is not dangerous if it is confined, as in the great majority of cases it is, to the trachea and larger bronchi. In most cases the expectoration gradually diminishes and the patient is well in a week or ten days.

In others, however, cough still recurs every morning when the sputum accumulated during the night is brought up; and this lasting until a fresh "cold" brings on a fresh accession of catarrh, the acute gradually becomes a chronic complaint.

Capillary form.—Very different is the course of acute bronchitis when it attacks the bronchioles throughout the lungs, for this may be one of the most rapidly fatal of all diseases. The word is a misnomer. and the old term "Suffocative Catarrh" would be better.

It usually is the result of previous bronchitis affecting the larger tubes, but occasionally is primary and sets in with a sensation of chilliness, or less frequently with a rigor, which may be repeated. The degree of pyrexia varies; the temperature may range up to 104°, especially in children; more often it is at a lower level; and it does not run any typical course. The head and the upper part of the body become covered with sweat. The hands and the surface generally feel hot, and the face is more or less flushed.

The pulse is frequent, sometimes so frequent that it cannot be counted. It is often tense and full, perhaps from contraction of the systemic arterioles.

But the most prominent symptom of this form of bronchitis is the dyspnœa. The patient sits up, with chest heaving and with nostrils quivering, unable to utter more than two or three words at a time, using his shoulders and arms in violent efforts to breathe. On carefully inspecting the thoracic movements, one finds that there is a great obstacle to the entrance of air into the lungs. The epigastric and hypochondriac regions of the abdomen recede at every inspiration; in children all the lower ribs and lower part of the sternum may be forcibly sucked in. The supra-clavicular and the supra-sternal spaces also recede, but, on the other hand, as Seitz pointed out, the upper ribs often remain almost motionless in a position which is that of a forced inspiration, giving to the corresponding part of the chest a vaulted shape.

The cough of suffocative bronchitis is often exceedingly harassing. At first it is usually dry, there being nothing in the air-passages to be expectorated. Afterwards it is accompanied by more or less abundant sputum. The secretion of the inflamed bronchial mucous membrane goes through stages very similar to those that may be observed during the progress of a cold in the head, or the ordinary bronchitis above described. The dry stage sometimes lasts several days, or even throughout the whole duration of the disease. But it must not be forgotten that infants and children as old as ten or twelve years swallow whatever they cough. In older patients, when sputum first appears, it is as a rule scanty, and dislodged with great difficulty, the patient perhaps coughing many times in rapid succession, until he is purple in the face, before he can get relief by bringing up a translucent pellet of mucus. But in other cases the spitting-jar becomes filled in a few hours with a considerable quantity of a greyish-white glairy liquid, which has numerous air-bubbles entangled in it. Under the microscope this kind of sputum is found to contain remarkably few leucocytes or epithelial cells.

As already remarked, in bronchitis the tubes cease for the time to be lined with columnar epithelium. It is therefore probable that when a few cells of that type are seen in the matters expectorated (except at the very commencement of the disease) they have been derived from healthy and not from inflamed parts of the air-passages, just as flat epithelial cells are often seen which come from the throat or the mouth.

Event.—In many cases the inflammatory exudation accumulates so as to threaten death by suffocation. Râles become audible all over the chest, and are so loud that no trace of the vesicular murmur can be anywhere detected. Indeed, they are often heard at a distance from the patient. Still more important as a warning of danger is the supervention of cyanosis: the cheeks, the lips, and the hands assume first a faint lilac, and finally a leaden colour; the blood is no longer duly aërated, and a condition of asphyxia has begun. At the same time the alæ nasi open with each inspiration, like the nostrils of a blown horse. The respirations also rise in number, till they become three or four times more frequent than in health.

A still graver symptom is the failure of effort on the part of the respiratory muscles; the breathing gradually becomes more and more shallow, until at last it may be represented only by a slight flickering movement of a few of the ribs, or by a faint jerking contraction of the diaphragm. With this, too, the patient ceases to be conscious of the necessity for active breathing. Instead of remaining upright he sinks down in bed, with his head in any chance position. His mind may wander for a time, and then he becomes unconscious. Sometimes death is preceded by respiratory convulsions.

Prognosis.—As a rule, if acute bronchitis ends fatally, it does so by spreading to the smaller tubes or to the air-vesicles themselves. In some rare cases the patient succumbs within twenty-four or forty-eight hours; but it not infrequently happens that when the disease has apparently been subsiding favourably, a relapse occurs which puts an end to all hope of recovery.

In very old persons the prognosis is always grave; it is so likewise in those who are very fat, or who are already weakened by previous illness, or who have progressive muscular atrophy affecting the shoulder or trunk muscles, or any considerable deformity of the spine or of the chest. Primary bronchitis also is less dangerous than when it complicates pneumonia, disease of the heart or kidneys, or one of the exanthems—particularly small-pox.

In infants, the chance of recovery is better in proportion to the age; while the gravity of the disease is greatly increased by the presence of rickets. We must not, however, give a wholly unfavourable prognosis even for the most severe cases in children. It is surprising how rapid may be both the pulse and the breathing, for two or three days together, in those who ultimately recover. This is one of the diseases in which, as an old practitioner once told the writer, one must never give up a child until it is in its coffin.

Complications.—In acute bronchitis the digestive organs are often disturbed more than is accounted for by the very moderate pyrexia. The tongue is coated with a thick whitish-yellow fur; there may be nausea and vomiting, and the bowels may be obstinately constipated. In children it is sometimes difficult to say whether the abdominal or the bronchial symptoms are primary. Pleurisy with effusion is not very uncommon along with bronchitis, even when there is no pneumonia, or tubercle, or Bright's disease to cause it.

But the most important complications of acute bronchitis, when it attacks the smaller tubes, are *collapse* of the pulmonary tissue and *broncho-pneumonia.* The latter is distinct in its origin, pathology, and histology from true or acute pneumonia; but since, unfortunately, that term is applied to at least three separate diseases, it will be more convenient to consider them together in the next chapter.

Pulmonary Collapse is anatomically identical with a state of lung which is seen in infants as the result of imperfect respiration, and which is nothing else than a persistence of the fœtal condition of the tissue. That, however, is properly termed Apneumatosis or Atelectasis (ἀτελής = imperfect, ἔκτασις = expansion). It affects the whole of both lungs if the child has never breathed at all, or parts of the lung (especially the anterior edges) if it has breathed incompletely, from having been prematurely born, or being weakly, or having its air-passages obstructed by mucus.

A German writer, Jörg, is generally credited with having been the first to point out, in the year 1832, the real nature of the atelectasis, which before was supposed to be congenital pneumonia; and it has been stated that the patches of collapsed lung also were up to that time confounded with red hepatisation. But Bright in 1828, writing of the morbid appearance found in the lungs of two children who had died of whooping-cough, showed that he clearly recognised the difference between these two conditions: indeed, it is unmistakable. A collapsed part of the lung is reddened, and the colour of its cut surface is reddish brown, or, when covered with pleura, reddish purple or violet; but a section of it looks perfectly dry, smooth, and homogeneous; it has not the dull, lustreless, and granular appearance of hepatisation. Moreover, its surface lies below the level of the adjacent air-containing parts of the lung, whether seen in sections or on the pleural surface; if it reaches the free edge of the organ it forms a notch there. Lastly, inflation from the bronchus will usually restore it to its normal appearance. Sometimes, however, collapsed pulmonary tissue is at the same time œdematous, and then its characters are less marked, its cut surface being moist and emitting serum when gently squeezed.

Lichtheim found in his experiments that acute œdema of the collapsed lung sometimes occurred, so that, although airless, it was bulky, soft, and moist. The retention of secretion in obstructed tubes is suggested by Jürgensen as a probable cause of inflammatory irritation; and Lichtheim, in some instances in which an animal survived for a considerable time after obstruction of the bronchus, describes the lung as looking almost like a sacculated kidney, full of dilated channels distended with pus.

It is, however, certain that, as a rule, pulmonary tissue, when collapsed as the result of disease in the human subject, remains uninflamed. If there are patches of broncho-pneumonia in the same lung, their presence is merely a coincidence.

Collapse after bronchitis must be distinguished from the airless condition called *carnification*, which is caused by compression from pleural effusion; here the tissue is bloodless as well as airless, and the colour is slaty or mouse-coloured instead of being reddish brown.

The way in which collapse arises was well illustrated in a case which occurred at Guy's Hospital in 1874.

A child, aged two years and two months, died four days after the performance of tracheotomy for a chronic laryngeal affection. On the under surface of the left lung there was a narrow red line of collapsed lung-tissue. This had running through it a tube which (like all other tubes in the same part of the organ) happened to have become dilated, as the result of the chronic obstruction to the child's breathing. That tube was plugged at its upper part by a small fragment of sponge, about a quarter of an inch long, which had evidently fallen into the trachea at the time of the operation. The limitation of the collapse to the part of the lung served by the obstructed tube was perfect.

Collapse is in most cases secondary to closure of the corresponding tube by viscid mucus. The precise way in which this brings about collapse is now ascertained. Gairdner, in 1850, suggested that the plug acted like a ball-valve, allowing air to escape during expiration, and preventing its entrance during inspiration. But this explanation would not account for the complete disappearance of the air, inasmuch as the elastic force of the pulmonary tissue and that of the confined air must soon become inadequate to raise the valve. Lichtheim, of Berne, in an important series of experiments recorded in the 'Arch. f. exp. Path.' for 1879, showed that in rabbits collapse follows within twenty-four hours after a bronchus has been plugged by a piece of laminaria, which rapidly swells and prevents passage of air in either direction. It is therefore evident that the affection must depend mainly, if not entirely, upon absorption of the air by the blood which circulates in the walls of the alveoli; and Lichtheim gave reasons for believing that the several gaseous constituents of the atmosphere are taken up successively, the carbonic acid and oxygen first, and afterwards the nitrogen. A further result of his investigations is the proof that the elasticity of the pulmonary tissue is not exhausted until it has become completely devoid of air. For without the aid of this elasticity, absorption by the blood would cease before collapse could become complete.

The state in which a lung is found when there has been slight narrowing of the space in which it lies (whether from pleural effusion or enlargement of the heart or pushing up of the diaphragm) is inexplicable, unless it be admitted that whenever even a small part of the organ fails to be acted on by the forces which are concerned in inspiration, its elasticity brings about a total collapse of its substance, notwithstanding that the tubes which serve it may be patent. The truth of this doctrine we should not have expected; but there seems to be no doubt of it. Now, in bronchitis, at least in children, it often happens that large portions of the lower lobes of the lungs are unacted upon by inspiratory forces, for the lower ribs and even the lower part of the sternum are often drawn inwards, instead of rising, during the act of breathing. The same thing occurs in croup and in other diseases attended with laryngeal obstruction. It therefore appears probable that collapse of the lower and anterior edges of the lungs is generally, if not always, due directly to the cessation of inspiratory traction upon those parts of the organs. Bartels, as far back as 1860, showed by dissection that it is often impossible to demonstrate any plugging of the tubes passing to collapsed areas of pulmonary tissue. So, again, we may refer the collapse of the bases of the lungs, which is so frequently observed after death from enteric fever, to the shallowness and imperfection of the respiratory movements. Even when collapse depends upon obstruction of tubes, an important factor in its production is a deficiency of power in the muscles of the thorax. For, with strong muscles, there is not only the chance that a fit of coughing may expel a plug of mucus, but also that a vigorous inspiratory effort may succeed in drawing air into the tissue in spite of it. Accordingly in adults collapse scarcely ever occurs as the effect of primary bronchitis. Even in children its development is greatly favoured by a rachitic state of the ribs, and perhaps also by the muscular weakness resulting from measles or any other acute disease; and the younger the child the more likely is it to show collapse of the lungs under a bronchial attack. The lung gradually recovers as the child gains its health and grows in strength,* but the effects on the thorax often remain as permanent deformities.

It is possible that a similar explanation may be found to apply to the curious girdle of small varicose arborescent veins which are often seen more or less completely bordering the abdominal aspect of the ribs. They are not uncommon in apparently healthy adults, and are rare in children.

The view that inspiratory falling in of the lower part of the chest is he chief cause of collapse of the lungs, does not exclude the opinion that xtensive collapse due to obstruction of the corresponding bronchial tubes nay sometimes in its turn lead to a falling in of the thoracic walls. Dr ee, for example, describes unilateral shrinking of the chest as resulting in ome cases from collapse of the whole lung in consequence of plugging of its main bronchus. Generally speaking, however, the space in the pleural cavity vacated by a collapsed portion of pulmonary tissue is filled up by over-distension of other parts of the organ; or, if an entire lung should become emptied of air, the opposite lung undergoes enlargement and displaces the mediastinum.

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* So far as appears, collapse, whether arising from bronchitis or from laryngeal obstruc- on, is always a temporary condition; for the affected parts of the lung again receive air recovery takes place from the primary disease. I have never seen in the *post-mortem* om any unmistakable results of collapse at a former period.—C. H. F.

With the exception of the collapse which occurs at the extreme bases of the lungs as the result of inspiratory retraction of the chest walls, this condition, when secondary to bronchitis, seldom affects more than small portions of each organ, lobules or groups of lobules scattered here and there, some on the surface, others in the interior. Hence it does not often give rise to very definite physical signs; though, if many patches should exist in close proximity to one another, it is possible that there would be more or less dulness on percussion, and deficiency of vesicular murmur. As regards prognosis, it can only be said that collapse aggravates the dyspnœa already produced by the bronchitis.

Chronic Bronchitis.—This, like the acute form, varies widely in its degree of importance and of severity in different cases. Some of its mildest forms are seen in children and in young adults, who from time to time have attacks of what is termed bronchial catarrh, until, as they get older, they perhaps ultimately "grow out" of them. Other cases, chiefly in persons advancing in age, take the form of a winter cough.

Year after year, during the cold season, they become troubled with a cough, which leaves them entirely in the summer. Sometimes it occurs only in the morning when they rise from bed; sometimes it goes on at intervals throughout the day; sometimes it is very bad at night, so as to disturb their rest. For a long time there is no dyspnœa; but gradually they find that in muscular exertion, as in walking uphill, or in mounting stairs quickly, the breath becomes a little short and hurried. Even then it is surprising how little heed is paid to these symptoms, which, among the poorer classes, seem to be taken almost as a matter of course. In making autopsies we repeatedly find the tubes in the lower parts of the lungs filled with pus, and even dilated, and the lungs themselves markedly emphysematous, in the bodies of patients who had perhaps died in the surgical wards of the hospital, and who had never made any complaint of cough or dyspnœa.

After a few years the cough continues during the summer; and there is constant dyspnœa, the breathing being hurried and wheezing, especially during exertion. For a long time there is no loss of flesh; but at length wasting occurs, and it may reach an extreme degree. There is probably always some emphysema when the cough and dyspnœa are continuous throughout the year.

It is only during the earlier stages of chronic bronchitis that it is possible for the patient to recover, so as to remain henceforth free from its return when exposed to cold or damp. But even in advanced cases the disorder may often be kept at bay if the patient is able to avoid changes of temperature, and to spend winter and spring in a warm climate, or remain indoors throughout that season of the year. In this way life may not infrequently be preserved to its natural term.

When death occurs, it is sometimes as the result of an intercurrent acute attack; for those who suffer from chronic bronchitis are exceedingly liable to exacerbations.

In other cases chronic bronchitis ends fatally by the supervention of dropsy, like that which attends primary cardiac disease. The right chambers of the heart are found after death dilated and full of dark blood,[*] and the trunk and branches of the pulmonary artery are thickened, just as in cases of mitral stenosis. In one instance Dr Fagge found on measurement that the wall of the pulmonary artery was actually thicker than that of the aorta, and it was also atheromatous. Such changes probably never take place until emphysema of the lungs has developed itself; and the obstruction to the pulmonary circulation is sufficiently explained by the diminution of the capillary area in the pulmonary system of vessels. Traube laid stress upon another cause of increased pressure in the pulmonary artery, namely, the defective movements of expansion and retraction of the lungs in breathing, which, in a healthy chest, further the flow of blood through the pulmonary capillaries.

Ultimately the spleen and kidneys become indurated, the stomach congested, and occasionally the liver may become "myristicated." It is, however, remarkable how rarely these results of long and extreme venous congestion are seen in cases of chronic bronchitis with emphysema, compared with their constancy in those of mitral disease. One element in bringing about these more remote changes is the occurrence of granular degeneration in the muscular substance of the right side of the heart. Another is a like degeneration of the diaphragm, as pointed out by the late Mr Callender in the 'Lancet' for 1857, and by Zahn in vol. lxxiii of 'Virchow's Archiv.'

Varieties and complications.—Laennec described a chronic form of bronchitis as *catarrhe sec*. Such cases are recognised by Riegel as characterised by almost complete absence of expectoration. They may last several months, and often end fatally, at least in children. The most prominent symptom is a paroxysmal cough, which is so violent that the face becomes purple and the veins of the neck swell out. The only sputum seen in an adult is a little tough mucus. Pyrexia is absent or very slight.

Another variety of chronic bronchitis is attended with a remarkable flow of thin watery albuminous liquid from the mucous membrane, so that the name of *bronchorrhœa serosa* has been given to it. Laennec relates the case of an old man of seventy, who for ten or twelve years spat up about four pints of this watery secretion every day, and yet remained not ill-nourished. But in other instances, as Andral afterwards remarked in his 'Clinique Médicale,' extreme emaciation occurs, with weakness and pallor.

Chronic bronchitis, if it lasts long, gives rise to certain secondary affections of the pulmonary vesicles and of the air-passages which add greatly to its gravity. They are known as "pulmonary emphysema," and "bronchiectasis" or "dilatation of the bronchi."

EMPHYSEMA[†]—that is pulmonary or vesicular emphysema—must not be confounded with that condition of the subcutaneous and other connective tissues which was originally so named and still bears the title of surgical or traumatic emphysema. The latter depends upon infiltration with gas as the result of injury to some air-containing structure. The same inflation of the areola or lymph-spaces of the connective tissue has been sometimes practised by fraudulent horse and cattle dealers, so as to make an animal appear plump, or to obliterate the hollow over the orbits which denotes age or poor condition in a horse. In the lung itself infiltration of air into the subpleural connective tissue does sometimes, though rarely, occur, and may go on until, passing along the root of the lung, the air diffuses itself through the mediastinum, and reaches even the superficial fascia of the neck and of the chest; this last affection is technically known as "interlobular" or "interstitial" and "subpleural emphysema."

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[*] "Cordis ventriculi præsertim dexter, sanguine distenduntur."—Richards Brown (1795).

[†] The word is classical: *ἐμφύσημα*, inflatio, from *ἐμφυσάω*; but it is applied by Hippocrates to what we should now call flatulence.

The term is also applied to inflation of the connective tissue with gas from decomposition—a condition not infrequent after death, and sometimes seen in cases of gangrene during life.

An admirable description of vesicular emphysema as regards both its anatomical character and its symptoms was given by Laennec; before then it had almost escaped notice. Rokitansky first showed that it consists in part of an over-distension of the pulmonary alveoli, but also in part of an atrophy of their walls, causing their cavities to run together into irregular spaces, sometimes of very large size.

Over-distension of the alveolar tissue is sometimes seen as an acute condition. In children who have died after a few days' illness of laryngeal diphtheria, or acute bronchitis, it is common to find the lungs very bulky and looking far more open-textured than usual. One may speak of this condition as emphysema; but it is clear that there has been no time in such cases for the occurrence of atrophy of the alveolar walls; and in all probability, if the patients had recovered, the lungs would quickly have returned to their normal state. Even when some amount of emphysema seems to be really present, it may happen that immediately after an attack of dyspnœa a great apparent increase of the condition can be made out by physical signs, which yet subsides again within twenty-four hours. Hertz, in 'Ziemssen's Handbuch,' mentions the case of an asthmatic patient, aged thirty, in whom he observed such a transitory over-distension of the lungs on several distinct occasions.

Anatomy.—The presence of emphysema of the lungs in the dead body is recognised partly by touch and partly by sight. The substance has a peculiar soft and silky feel; it scarcely crepitates, if at all, when squeezed between the finger and thumb; and pressure upon its surface readily causes a deep pit, which remains after the pressure is removed, proving the loss of its normal elasticity. Another effect of the same cause is that the lungs do not collapse when the chest is opened. They often remain distended, and the left lung covers the heart, so that scarcely anything is to be seen of that organ. In a case which was observed at Guy's Hospital in 1868, one lung overlapped the other behind the sternum by an inch and a quarter. A similar condition, which must have been pathological, was found in a body which Dr Fagge, when demonstrator of anatomy, had frozen for the purpose of making a transverse section; a wax model of that preparation, which is now in the museum, shows one lung covering the other for some little distance; there was advanced phthisis, and it is very likely that emphysema also existed, although this was not proved to be the case. In extreme instances the lungs bulge in all directions, both during life and after death, displacing the structures around them. Their apices protrude far above the clavicles; and their bases bulge so that the diaphragm instead of being arched upwards is flattened. In two cases the pericardial sac was pushed inwards at its lower part, so that Moxon described the heart as resting upon and as being separated from the surface of the diaphragm by cushions of lung.

Sometimes there are large bullæ or blebs containing air, of all sizes up to that of a walnut or a pigeon's egg. These are seen chiefly along the anterior borders of the lungs, but sometimes also along their inferior borders or near their roots; and particularly in the ear-shaped process of the left lung. Sometimes, even when the lungs are highly emphysematous, no large cavities are to be seen.

The tissue, if closely inspected, is found to be full of spaces of the size of small shot or of millet-seeds. One noticeable appearance is a rounding off of the free edges of the lungs, and their outer surfaces often show marks of the ribs, the intercostal spaces having yielded so as to allow the lungs to bulge outwards. Emphysematous lungs are of a grey colour, mottled with spots and lines of pigment; they are soft and inelastic, but not friable, and their cut surface is dry and bloodless.

The earliest change in a lung that is becoming emphysematous is, according to Rindfleisch, a dilatation of the infundibula into which the alveoli open; according to Hertz, a nearly uniform dilatation of infundibula and alveoli. Gradually the alveolar walls waste, until nothing is left of them but low ridges projecting a little way into an oval or rounded space, which represents each infundibular cavity. After a time the septa between these spaces in their turn thin away and become perforated. Thus the result is a progressive increase in the size of the cavities with a diminution of their number. An aggregation of fatty granules round the remains of the nuclei of the alveolar epithelium is commonly present; and in the 'Med.-Chir. Transactions' for 1848 Rainey maintained that emphysema is primarily due to a fatty degeneration. A more important histological change is a remarkable diminution of the natural, yellow, elastic fibres which surround the alveoli. There is, of course, more or less destruction of capillary blood-vessels. Rindfleisch speaks of the vessels as collapsing until "only a narrow ribbon-like band is left, which may be recognised as an obliterated vessel by its greater transparency amid a dark, often pigmented, parenchyma, and by its uniting with other bands like itself to form the usual anastomotic network." He goes on to say that relatively wide communications are opened up between the pulmonary artery and the pulmonary and bronchial veins. These anastomoses appear in well-injected lungs as peculiar elongated unbranched channels of the same diameter throughout, strikingly contrasting with the far more numerous, extremely tortuous, and dilated arteries, for the contents of which no such supplemental mode of escape exists.

Pathogeny.—With regard to the mode of origin of emphysema of the lungs there have been many different opinions.

Laennec's hypothesis was that the tubes in cases of bronchial catarrh being obstructed by swelling, or by an accumulation of mucus, the air which found its way into the alveoli during inspiration became unable to escape during expiration, inasmuch as the expiratory force was less than the inspiratory. In other words, he thought that emphysema was the result of a process the exact converse of the ball-valve action which, as we have seen, was supposed by Gairdner to be the cause of collapse. Louis objected that the ordinary seat of catarrh is the base and lower part of the lung, whereas the parts most apt to be affected by emphysema are the apex and the anterior margin.

In 1851 Gairdner advanced the theory that emphysema arises solely during inspiration. His view was that collapse or reduction in bulk in one part of a lung is a necessary antecedent to the development of emphysema in another part. During inspiration, when the chest becomes enlarged, if each and all of the lobules cannot expand to fill it, some of them must be stretched unduly; and this produces emphysema.

For a few years Gairdner's view was widely accepted, and emphysema was held to be essentially "complementary" or "compensatory" either to collapse, or to retrogressive tuberculous disease, or to some other contracting lesion of the lung. But in 1856 Jenner published in the 'Transactions' of the Royal Medical and Chirurgical Society a paper to prove that the development of emphysema occurred during expiration. The same doctrine had already been taught in Germany, as far back as 1845, by Mendelssohn, in a work entitled 'Der Mechanismus der Respiration und Circulation.' Gairdner urged that it is impossible for emphysema to be produced by the act of expiration, even with a closed glottis, because the force by which the air becomes compressed within the lung opposes exactly as much resistance without as it creates pressure within. Jenner now pointed out (as Mendelssohn had done before) that certain parts of the thoracic walls are yielding, and consequently incapable of maintaining this resistance. Both these observers indicated the apices of the lungs as being devoid of adequate protection against an expanding force from within; and we have seen that Louis had long before shown that the apices were especially apt to become emphysematous. Jenner remarked that during a fit of coughing the supra-clavicular regions may be seen to bulge, and that by placing one's hand upon them one can feel that they are distended by a considerable force. If the apices are the seat of emphysema this bulging under violent expiration is extreme, and percussion over the bulging parts may elicit an almost tympanitic sound. He further showed that the upper costal cartilages are to some extent yielding, and that therefore the alveoli of the anterior margin of each lung become affected with emphysema as well as those of the apex. Other parts which he also named as apt to become emphysematous were—the margin of the base of the lung, the part of the lung near its root below the entrance of the bronchus, and the little ridge of lung which, on the right side, projects behind the trachea. The base of the left lung generally he showed to be less firmly supported than that of the right, the liver being more unyielding than the stomach; and he cites Louis as having found the left lower lobe emphysematous twice as often as the right one.

The correctness of Jenner's theory has since been supported by observations made in certain cases of congenital malformation of the chest walls. Thus in the case of Groux, who had a fissure of the sternum, the anterior part of the lung protruded through the fissure in the act of coughing. Ziemssen met with an example of absence of the *pectoralis minor*, and of the entire sterno-costal part of the *pectoralis major*, so that the intercostal muscles of the four upper spaces were covered only by fascia and by integument. During forced expiration these spaces bulged from 1 to 1½ mm. above the level of the ribs; when the muscles of one space were faradised that space for the time remained flat, the others bulging as before. Further evidence is afforded by cases in which, after the cicatrisation of penetrating wounds of the chest, the affected parts have ultimately become the seat of hernia of the lung, as the result of weakening of the thoracic parietes. Many such instances may be found collected in a little work published by Desfosses in 1875. It may be noted, too, that horses are liable to an affection of the lungs identical with emphysema, as the result of the straining efforts which they are called on to make, during which they keep the glottis closed. As Sir William Jenner says, in vol. iv of 'Reynolds' System,' "no one who watches a horse draw a heavy load up a short steep incline on a damp cold day can doubt this. While making the effort the horse holds its breath, having previously inflated the lungs. No sooner, however, does this animal cease its effort than the glottis is opened, and the air suddenly expressed from the lungs. The degree to which the air was compressed may be judged by the distance to which, and the sudden violence with which, the cloud of breath-vapours is seen to be driven forth." In his paper in the 'Med.-Chir. Transactions,' Jenner had shown that the parts of the lung that are emphysematous in a "broken-winded" horse are those which are so placed as to be least able to resist pressure. It is curious that a capital description of this affection of horses was given before the end of the seventeenth century by Sir John Floyer, in a treatise on asthma; the passage is cited in full in 'Watson's Lectures.'

Emphysema then, in its most common form as the result of bronchitis, is the result of expiratory pressure with a closed, or partially closed, glottis. The expiratory muscles forcibly compress the air within the chest, and if all parts of the thoracic parietes were equally unyielding no harm would result. But as certain parts can and do yield, some of the compressed air is driven into the corresponding alveoli of the lungs, and gradually breaks down their structure in the manner already described. No doubt the resistance of the chest walls in different regions fails progressively, more and more, as the affection advances. Thus the sternum and the upper cartilages become arched forwards, a change which probably is due to the frequently repeated application of an expansile force from within the thorax. It is only as the result of long-continued pressure that the diaphragm can become flattened, and that the lung can protrude inwards beneath the heart, as described above.

Even when a part of the lung (generally the apex) is shrunken by retrogressive tuberculous disease, many pathologists agree with Jenner in thinking that the development of emphysema in the tissue around, particularly along the anterior edges, is due to the pressure of air driven into the alveoli by coughing. Others, however, still hold that to such cases Gairdner's theory remains applicable, and that the emphysema is "complementary" in the strict sense of the term.

Although a frequently repeated cough is one of the chief causes of emphysema, yet in man, as in horses, other actions beside coughing may increase the expiratory pressure so as to produce the same result. This is the case, for example, with all violent efforts in which the glottis is kept closed to fix the chest. Dragging or lifting heavy weights, straining at stool, even the act of parturition, may be mentioned as possible causes of emphysema. Thus Waldenburg is cited by Hertz as having seen the affection develop itself in a medical student who, having come from a country place where he had no occasion to ascend the stairs, occupied in Berlin an apartment on the fourth floor, up to which he ran without stopping several times daily. Hertz himself met with a similar case in a young shopman, whose lungs became emphysematous in about a year, without any cough or bronchial catarrh, as the result of his having to carry heavy goods up a high staircase in haste a great many times every day. For persons who have suffered from bronchitis, and in whom the distending process has already begun, it is most important to avoid all occupations or amusements that involve repeated expiratory efforts. Playing a wind instrument may sometimes be exceedingly injurious; for, although the glottis is not closed, the air within the chest is kept under great pressure, while it is being slowly allowed to escape.

Atrophic emphysema.—Of late, German observers have been disposed to attribute emphysema in part to primary changes in the pulmonary tissue independent of the mechanical conditions just described. A *senile atrophy of the lungs*, bearing a close resemblance in its characters to emphysema, is generally admitted by pathologists; it was originally described by Dechambre in 1835, from observations made at the Salpêtrière. Sir William Jenner speaks of it as "*small-lunged emphysema*," in contrast with the ordinary form of the disease, which he calls "large-lunged emphysema." As he says, the small size of such lungs, their lightness, and the very small space into which they may be compressed, are often most remarkable. When the thorax is opened after death they "fall in like an inflated bag of wet paper." The subjects of senile atrophy of the lungs are commonly thin, shrivelled, and withered-looking. Their chests are very small and narrow, the lower ribs being so obliquely placed that they almost reach the crest of the ilium, and so closely packed as nearly to come in contact with one another. The lungs are so reduced in size that the extent of præcordial dulness may be increased, notwithstanding that the heart partakes of the general wasting. Yet there is commonly little distress of breathing, because the volume of the blood is at a minimum, and because the deficiency of muscular power forbids active exercise.*

Predisposition.—That in younger persons some cause for emphysema must exist, beyond expiratory pressure upon the alveoli, is argued by Hertz from the fact that in certain families several members are found to suffer in succession, as the result of comparatively trifling affections of the air-passages. Schnitzler, for example, saw three brothers, whose parents were still alive and well, but who all became the subjects of emphysema at the age of thirty, without definite cause. Dr Jackson, of Boston, cited by Walshe, found that "of twenty-eight emphysematous persons, eighteen had either a father or a mother, or both, similarly affected; whereas of fifty non-emphysematous people, three only sprang from emphysematous parents." To meet such cases a relation between emphysema and the supposed "gouty diathesis" has been surmised.

So far as concerns the mere occurrence of this affection in different members of the same family, it is important not to overlook the fact that they may all have been alike exposed to the causes of bronchia catarrh, and perhaps all unduly susceptible of taking cold. But Cohnhei and other German writers have looked for an explanation of emphysem apart from mechanical causes, in a varying degree of elasticity of the pu monary tissue. In this connection certain observations of Perls are interest, recorded in vol. vi of the 'Deutsches Archiv.' By means of a pressure gauge he determined in a large number of cases the degree of force with which the lungs retracted when the pleural cavities were opened in the dead body; and he found that after death from enteric fever, or (in one case) from phosphorus-poisoning, their elasticity was reduced almost to nothing. Cohnheim believed that in a large proportion of cases emphysema depends upon a congenital defect of development in the elastic tissue of the lungs. But a paper which he quotes by Eppinger in the 'Prag. Vierteljahreschrift' for 1876 does not bear out this assertion; for although Eppinger found that in emphysematous lungs there is a reduction of the network of elastic fibres in the alveoli, he yet appears to have recorded this as a change due to the development of the disease, and not as an antecedent malformation.

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* It has always, however, seemed to me that the supposed likeness of this seni atrophy of the lungs to emphysema is really due to the fact that a slight degree of emph sema resulting from the bronchial catarrh; to which aged persons are so liable, is common mixed up with it. Hertz, in his chapter on atrophy of the lungs in 'Ziemssen's Handbuc speaks of bronchitis as a frequent "complication;" and he also mentions that the bro chioles are very thin and "generally uniformly dilated, seldom irregularly sacculated." B bronchiectasis can hardly be otherwise than mechanical in its origin; and it seems reaso able to take the same view of the pulmonary rarefaction, which Hertz describes as bei most marked at the apices and along the anterior edges.—C. H. F.

In this connection a case recorded by Hertz is of great interest. It is that of a regimental cornet-player, aged thirty, who had always been able to use his instrument without any difficulty, even on the march. He was attacked with double pneumonia, which subsided in a week; he felt quite well, had no cough, and began to practise with his cornet. But in the course of the next seven months he discovered that he was no longer able to take sufficient air into his lungs to maintain a long note for the proper period, that he was short-breathed on exertion, and could no longer play while marching. Hertz found on examination that the lungs were markedly emphysematous, which had not formerly been the case; and his supposition is that the pneumonia had damaged their texture, so that they were not able to resist expiratory pressure as before.

Clinical symptoms.—The recognition of emphysema is based mainly upon physical signs. The chief subjective symptom of the disease is *dyspnœa.* The patient first notices that he is short of breath when he exerts himself, as in running upstairs; but after a time difficulty of respiration becomes a permanent condition from which he is never entirely free. He cannot get the air out of his chest as he used to, and he has a continued sense of inflation or distension. It is true that many emphysematous persons affirm that their dyspnœa is only occasional; but as Walshe in such cases remarked, the patient is often deceived, a moderate amount of dyspnœa having become to him a second nature—a thing unperceived, and giving rise to no discomfort.

It does not always happen that the affection advances; it may continue stationary, and life may be maintained until extreme old age, provided that intercurrent attacks of bronchitis can be prevented.

In most cases, however, emphysema becomes more and more marked every year. At last the noisy hurried breathing may become so short that he patient cannot utter a sentence without stopping in the middle. At night he has to be propped up by pillows. His distress is aggravated from ime to time by intercurrent bronchial catarrh, or by the supervention of sthma, or by pushing upwards of the diaphragm, as the result of distension f the abdomen with flatus, or with undigested food. The face, the hands, nd at last the whole body become livid, as if from capillary bronchitis. In act, chronic bronchitis is so often complicated with more or less of emphysema, that it is impossible to separate the effects of each. Cough may be ntirely absent in cases of emphysema unless there is bronchial catarrh; ut many persons whose main disease is emphysema have a constant cough, nd expectorate a frothy liquid, or pearly-grey pellets of mucus.

Hæmoptysis is not generally admitted among the symptoms of emphysema. Sir Dyce Duckworth, however, in the eleventh volume of the 'S Bartholomew's Hospital Reports,' declares that it is not infrequent; an the following case confirms the fact that it may occur.

> In 1869 a woman, aged forty-nine, was brought dead into Guy's Hospital after ack of severe hæmoptysis, and at the autopsy the only disease that could be discover s an extreme degree of emphysema of the upper lobes of the lungs, with some exces ous tissue forming the interlobular septa. The air-tubes were full of clots. She w d to have suffered for three months from wheezing and short breath. On the mornin her death she woke up at 4.25 a.m. with "coughing and vomiting of blood through th se and the mouth, and was suffocated in ten minutes."

Ultimately emphysema gives rise to great wasting, and to extreme e eeblement of the muscular strength. A noteworthy circumstance, me oned by Walshe, is that the over-distension of the chest renders the bod unnaturally buoyant in water, so that the patient is astonished to fin mself able to swim more easily than before, at a time when he is growin more and more incapable of other kinds of exertion.

Physical signs.—Of these the most important are derived from *pe ussion*. Even slight degrees of emphysema may be detected by carefull apping out the areas of the heart and of the liver. Instead of beginnin t the upper border of the fourth left costal cartilage, the cardiac dulnes egins only over the fifth or even the sixth cartilage. Instead of beginnin t the upper border of the sixth right rib, the hepatic dulness begins only a he level of the seventh or of the eighth. If in a case of bronchitis one find hat percussion over these two organs yields normal results, one is generally afe in declaring that no appreciable amount of emphysema has ye eveloped itself.

As the disease advances, the heart becomes so completely covered by he lungs that no cardiac dulness at all can be detected, the pulmonary resonance above meeting the tympanitic note of the stomach below. At the same time the apex-beat ceases to be felt in the normal position, in consequence of the downward displacement of the diaphragm carrying the heart with it; and cardiac pulsations can often be felt in the epigastrium. On the right side pulmonary resonance now extends down to the margin o the thorax. The edge of the liver may sometimes be felt in the hypochondrium; but very often this is not the case, and there may be so marked a reduction in the area of the hepatic dulness, that one may be tempted to suspect cirrhosis. This seems to be due to the great increase in the antero-posterior diameter of the thorax preventing the liver from coming in contact with the parietes of the chest and abdomen to the normal extent. Another noticeable feature of well-marked cases of emphysema is the clear character of the percussion-sound over the sternum as high as its upper border; and over the bases of the lungs behind a clear percussion-sound is elicited to a much lower level than normal.

Moreover the percussion-sound, over parts of the chest where it ought naturally to be resonant, is over-resonant, and according to Walshe and Gee there is often a fall in its pitch. The change in the percussion-sound is most obvious over the back of the lungs, a sonorous drum-like note being readily produced where normally there is often difficulty in eliciting a clear resonant sound.

Even more valuable indications of emphysema are afforded by *inspection*. As already explained, the sternum and the upper ribs arch forwards as the result of their yielding to the frequently repeated respiratory pressure which is the cause of the affection. The sternum not infrequently becomes convex in a vertical plane, with an angle, known as the *angulus Ludovici*, at the junction of the manubrium with the gladiolus. The clavicles also are more bent than under normal circumstances. The curve of the dorsal vertebræ becomes greatly increased, so that the back is rounded. The effect of these alterations in the chest walls is to give it the inspiratory form. It is aptly said to be "barrel-shaped." By the cyrtometer its horizontal circumference is shown to be almost circular. Sometimes the rounding of the ribs and of their cartilages continues to the very bottom of the thorax, and the hypochondriac regions are permanently expanded to the fullest possible extent. One result of this is, as Hertz has pointed out, a transverse groove, which crosses the abdomen horizontally from one twelfth rib to the other; it is due to the stretching of the upper part of the transversalis abdominis muscle, which is fixed to the rib cartilages, as compared with the relaxed condition of the lower part, which has no such attachment. This groove may form a conspicuous, and at first sight puzzling feature of a case, when ascites is also present. But in other instances the lower ribs and their cartilages are flattened, or even hollowed inwards; this occurs especially when the pulmonary affection began in bronchitis or whooping-cough at an early period of life, so that the bases of the lungs became collapsed.

The "subcostal" angle at the ensiform cartilage is far more open than usual, or obtuse, as in deep inspiration.

The upper intercostal spaces in emphysema are either unaffected, or narrowed, while the lower ribs are widely separated. Stokes declared that he had never seen the spaces otherwise than depressed; but Walshe and others have observed them to be prominent or even bulging when the lungs beneath are highly emphysematous.

The high shoulders, increased depth of chest, short and full neck, raised ribs, closer together above and wide apart below, with the obtuse substernal angle, are the physiological characters of the chest during deep inspiration, and can be imitated by filling the lungs and holding the breath. The same fulness of the chest may be the result of distension of the pleura by fluid on one side or the other, but when bilateral it is characteristic of emphysema alone. It is the exact opposite of the "expiratory" form of chest seen in advanced phthisis. The one is the result of hypertrophy, the other of atrophy of the lungs.

During inspiration the chest in well-marked cases of emphysema is seen to be almost motionless. It is, indeed, impossible for the upper ribs to rise and expand, as they should do, because they have permanently assumed the position of inspiration in health. There is, however, a jerking movement upwards of the thorax as a whole, produced mainly by contractions of the sterno-mastoid and scalene muscles, which start into unnatural prominence. If the lower ribs are thrown outwards as much as the upper ones, the chief agent in inspiration must be the diaphragm. But in many cases the condition is reversed. The lower ribs still retain a certain degree of mobility, but the diaphragm is pushed downwards, so that it can do little towards enlarging the thoracic cavity. It may then be observed that the epigastrium becomes hollowed during the act of inspiration.

Expiration is prolonged and laboured, with fulness of the parts above the clavicles and of the great veins at the root of the neck. Violent cough causes still greater bulging of the supra-clavicular spaces; and one must not confound this condition with distension of the veins, or even with the sudden protrusion of an aneurysmal sac during the act of coughing. Another point to which Jenner has drawn attention is, that if one is feeling the pulse of an emphysematous patient while he coughs violently, one perceives the artery to become suddenly full and tense, and it ceases for a brief space afterwards to beat.

Auscultation gives comparatively little information in cases of emphysema, apart from signs of the bronchitis that is so commonly associated with it. One sign of emphysema given by the stethoscope is enfeeblement or nearly complete absence of the vesicular murmur. The bronchial breathing which may normally be heard over the roots of the lungs behind is often wanting in emphysematous patients; but this is not infrequently the case in those who are healthy. The most constant auscultatory sign is that expiration is exceedingly prolonged owing to the loss of elasticity in the lungs. Walshe says that, instead of being only one third the length of the inspiration, it may be four times as long; in other words, its relative duration may be increased twelvefold. The growling, squeaking, or wheezing expiratory sound of emphysema is commonly spoken of; but this sign belongs to concomitant bronchitis and not to the dilatation of the vesicles. In one case, at Guy's Hospital, as the tubes became free, the expiratory rhonchi entirely disappeared, although the extremely feeble state of the inspiratory murmur and the over-resonance of the percussion-sound clearly showed that emphysema still existed.

In cases of emphysema the *vocal resonance* is much diminished, and tactile vocal *fremitus* is often entirely lost.

In the latter stages of the disease a tricuspid regurgitant murmur may often be detected; and even at an earlier period increased pressure in the pulmonary artery may be indicated by an accentuated second sound. Epigastric pulsation and pulsation of the veins of the neck with each beat of the heart may also be observed. These symptoms are all the result of obstruction in the pulmonary capillaries having led to dilatation of the right side of the heart and incompetence of the tricuspid valve.

When air has escaped into the subpleural connective tissue, producing what is known as "interlobular" or "interstitial emphysema" (p. 1060), there is sometimes heard a friction-sound very like that which occurs in pleurisy. This sign was originally noticed by Laennec. Most writers since then have been disposed to doubt the correctness of the observation; but it has been confirmed by Gairdner; and the late Dr Hudson, of Dublin, in his edition of Stokes's works, says that he also met with a case in point.

The *treatment* of emphysema will be considered with that of bronchitis at the end of the present chapter.

Hypertrophy of the lungs.—The barrel-chest above described, *i.e.* the thorax when filled by the ordinary or "large-lunged" emphysema, is closely simulated by a condition which is physiological rather than diseased, and may be described as uniform overgrowth of the lungs. It occurs to some extent in all healthy persons who are engaged in hard manual labour or in athletic sports. The chest of the youth who improves his wind by rowing, or of the workman who is accustomed to carry heavy weights, becomes expanded, and the lungs increase in volume and capacity. When the efforts made with the glottis closed are too great, rupture of the air-vesicles is apt to occur, and thus true emphysema may be mingled with physiological hypertrophy; but this is far from a necessary complication.

Ascending ladders and climbing hills, especially when a weight is carried on the shoulders, has a similar effect. Greater efforts of inspiration are made, the lungs are expanded more perfectly, and the breathing capacity of the chest is increased.

Habitually breathing rarefied air produces similar but exaggerated effects. In persons who live in mountainous regions both causes of hypertrophy of the lungs combine; they are frequently ascending heights, often with burdens on their shoulders, and they are constantly breathing air at a low pressure, so that each cubic inch of lung gives them less oxygen to aërate their blood. The result is that the thorax becomes enlarged in all its dimensions. Instead of a yard's girth for a man of good stature, and a metre's for a tall man, the chests of men of five feet six inches or even less measure more than forty inches in circumference.

This condition may be observed in Swiss guides; but it has hitherto been found most constantly and highly developed among the natives of the lofty table-lands of the Andes, in Bolivia.

DILATATION OF THE BRONCHI; BRONCHIECTASIS.—This condition was first described by Laennec. It is seldom a primary disease, and is often found associated with fibroid induration of the tissue between the enlarged tubes; such cases will best be discussed in the account of Cirrhosis of the lung. But more often we meet with bronchiectasis as the result, not of interstitial pneumonia, but of bronchitis.

Anatomy.—All writers divide bronchiectasis into cylindrical and sacculated, and the definition is of pathological and clinical importance.

In the *cylindrical* form the tubes sometimes run through the substance of the lung, with but little diminution of calibre, until they end abruptly beneath the pleural surface; their appearance is often compared to that of the fingers of a glove, but it rarely happens that the dilatation is so uniform throughout their whole length. In one instance, which occurred at Guy's Hospital in 1873, the bronchial tubes were so enlarged as to be conspicuous at the root of the lung, "pushing the lobes apart from one another," as it is expressed in the report of the autopsy. More often the medium-sized and smaller tubes are alone affected, when the bronchial dilatation may be obvious on the cut surface of the lung. Too many large orifices are visible, from which pus wells up in abundance; or, if the tubes are empty, we open them up with scissors and find them dilated. According to Dr Fagge's observations, slight forms of bronchiectasis are much more often met with in the extreme bases than in any other parts of the lungs. Not infrequently he recorded enlargement of the principal tube passing into the ear-shaped process of the left lung when no such change could be made out elsewhere. This was when that process was emphysematous; and, indeed, these less-marked examples of bronchiectasis are very often found in association with emphysema. On the other hand, when the bronchial affection reaches an extreme degree, there is seldom a corresponding amount of rarefaction of the pulmonary tissue. If the tubes in any one part of the lung are universally dilated, running to the surface side by side, and perhaps as large as quill pens, or even larger, the parenchyma between them is perforce reduced to a small space. In these cases it is sometimes difficult, at the first glance, to say whether the affection is or is not secondary to a fibroid change in the lung tissue. But in the most marked and typical cases of cylindrical bronchiectasis, such as occur in children after whooping-cough or measles, the tissue between the dilated tubes is often quite free from induration or other morbid changes.

It must be understood that cylindrical bronchiectasis is by no means absolutely uniform in diameter at different points. Sometimes the tubes gradually widen as they approach the surface. Sometimes they have fibrous bands or trabeculæ projecting from their walls here and there, and rendering their calibre very irregular.

The *sacculated* dilatation of the bronchial tubes, also, varies widely in character in different cases. The most typical form is one which presents appearances described as follows by Dr Fagge:—"When a section is made of the lung the cut surface appears to be covered with an immense number of shallow, smooth-walled depressions, like so many minute saucers. Each of these has in its floor a very small rounded orifice, and it is obvious that they are all sections of small bronchiectases, which probably were spherical before they were cut across, but have become flattened by their own elasticity, and by that of the adjacent pulmonary tissue. In all likelihood, if the lung in such a case could be inflated and dried, and the parenchyma then cleared away so as to expose the tubes in their continuity, each would be found changed into a regular series of globular dilatations, so as fairly to deserve the epithet moniliform. In other instances sacculated bronchiectases are more unequal in size and irregular in form; but it is probable that most of such cavities are really not dilated tubes, but smooth-walled vomicæ which have been formed by ulceration." It is to these latter sacs that in all probability the statement of Biermer applies (in vol. v of 'Virchow's Handbuch'), that the majority of the sacculi of bronchiectasis have openings only towards the trachea, and are closed on the distal side. Still less are true bronchial sacs ever converted into shut cysts.

Dilated bronchial tubes often have exceedingly delicate walls, being apparently thinner than they were before they began to increase in size. But sometimes the tissues are thickened, the lining membrane being velvety and showing the changes above described as occurring in bronchitis.

Pathology.—As to the way in which bronchiectasis arises, there can be little doubt that it is the mechanical result of expiratory pressure, being in fact produced by exactly the same cause as ordinary emphysema. To explain satisfactorily why in one case the alveoli should yield, and in another case the tubes, is perhaps impossible. But we may fairly suppose that it depends upon the degree to which the walls of the tubes have been softened by inflammation, and also, it may be, upon the comparative amount of elastic resistance originally possessed by the lungs and bronchi in each individual. (See Grainger Stewart's paper, 'Edin. Med. Journ.,' July, 1867.)

A more frequent cause of dilated bronchi is chronic interstitial pneumonia, but with this form we are not at present concerned.

Independently of previous organic changes in the lung, bronchiectasis is not frequent either in children or adults. It sometimes complicates emphysema, but more frequently takes its place. In children it is most often a sequel of whooping-cough, and sometimes comes on very early, and so severely that the patient grows up in a state of permanent cyanosis, with cold extremities and short breath. In adults bronchiectasis occurs in middle rather than advanced life, and is almost always preceded by bronchitis, seldom by tuberculous disease, and never by acute pneumonia.

Diagnosis.—It is only when bronchiectasis has reached an extreme point that it is characterised by definite *physical signs*. As a rule, the chief indication of this affection is the presence of râles which appear to be too "large" and loud to be formed in the undilated tubes of the part of the lung in which they are heard, as, for instance, at the extreme base, or along the anterior edge. If, however, several tubes cylindrically dilated are arranged side by side, while the lung tissue between contains but little air, there may be more or less marked bronchial breathing, with bronchophony. Such a case could scarcely be clinically distinguished from primary chronic pneumonia, with bronchiectasis as a result. Again, the question of the diagnosis of a saccular dilatation of a bronchial tube from a phthisical vomica, to which stethoscopists formerly gave much attention, applies to the dilatation which attends cirrhosis of the lung, not to cases arising out of bronchitis. One circumstance which is strongly indicative of bronchiectasis is for the physical signs over a certain part of the lung to undergo more or less regular variations from time to time, being now well marked, and now again indistinct or absent.

As for its *symptoms*, dilatation of the tubes is of course in part concerned in causing cough, dyspnœa, and lividity; but at the bedside one can never separate its share in producing these effects from that due to the bronchitis associated with it, and perhaps also to concomitant emphysema. The symptom that enables one to diagnose bronchiectasis with confidence is a peculiar way of expectorating which may in some cases be observed. For some hours, perhaps, there is no cough at all; but during this time secretion is accumulating in the dilated parts of the air-passages, the sensitiveness of which appears to be blunted, so that they do not resent its presence. At last perhaps some runs over into a tube which is still healthy, and the result is a more or less violent fit of coughing. The liquid that has collected is suddenly expelled, pouring out of the patient's mouth, and even through his nose, so as to half fill his spittoon. Sometimes this process is set in action by percussion of the chest. Sometimes it occurs, especially in the morning, when the patient rises from the recumbent posture. When this accumulation has been got rid of he usually feels much more comfortable than before, the breathing is easier, and the chest less oppressed. Such intermittent expectoration (or in children vomiting) of pus is a sure sign of dilated bronchi.

A few cases have been recorded (vol. xv, 'Deutsches Archiv,' Gerhardt) in which painful swelling of some of the joints occurred as a sequel of bronchiectasis. This is probably analogous to gonorrhœal synovitis, or to that which sometimes follows dysentery or leucorrhœa or parturition. The occasional supervention of abscess of the brain as a complication of suppuration in the air-passages or in the lungs has been already referred to.

Fœtid or putrid bronchitis.—When the bronchial tubes are dilated, bronchitis is often accompanied with the expectoration of foul-smelling sputa, and sometimes with horrible fœtor of breath. Occasionally putrid bronchitis has been recorded without bronchiectasis; but as a rule it is only when liquids have been long stagnant in some part of the air-passages, or in a space communicating with them, that putrefactive changes occur.

The characteristic symptom of this rare disease, as was first pointed out by Traube, is the presence in the matters expectorated of certain soft, friable, smooth masses, of a dirty greyish-yellow colour and very fœtid odour, varying in size from a millet-seed to a bean. Such bodies had been originally noticed by Dittrich in 1850 as plugging the affected tubes in fatal cases; and in Germany they are commonly called "Dittrich's" o "Traube's plugs" (*Bronchial pröpfe*). Microscopically they are made u of pus-cells and granules mixed with oil-globules. At a later stage they also contain certain long, narrow, acicular crystals, which Virchow describe long ago in the first volume of his 'Archiv,' as consisting of a fatty acid These crystals are colourless, often sharply bent or twisted, sometimes collected together in sheaves or in thick bundles. According to Guttman they contain a combination of palmitic and stearic acids. The curiou spirals named after Curschmann, who described them in 1887, may als occur, microscopical fragments of clear mucus, wrapped round by a slende thread-like coil, which is also amorphous.

In 1867 Leyden and Jaffé discovered that the granular matter consist of round or rod-shaped bacilli or beaded chains or filaments (*Leptothri pulmonalis*). The presence of leptothrix appears to be the reason wh iodine often gives a violet or blue tint to the mass, as Virchow and Gamge independently observed. Chemical analysis of the sputum of putrid bronchitis, in different cases, has shown that it may contain volatile fatty acid (valerianic and butyric), leucin and tyrosin, ammonia, and sulphurette hydrogen.* Leyden and Jaffé succeeded in inducing in ordinary muco-purulent sputum a putrefactive process like that in the expectoration o putrid bronchitis.

The sputum is generally abundant, and often separates in the spittoo into three layers: of these the uppermost is muco-purulent, opaque, greenish yellow, and frothy; the middle is a transparent albuminous liquid, lik serum; the lowest is granular, and of a dirty yellow appearance, consistin of pus and Dittrich's plugs. Sometimes the sputum is of a uniform chocolate colour from mixture with blood.

The odour of the patient's breath and sputum in cases of putrid bronchitis is often identical with that of gangrene of the lung. But in other cases it is altogether different. There is, of course, great difficulty in defining the distinction verbally; Guttmann compares it with the smell that pervades a soap factory. Dr Laycock's statement is that in one of his patients the odour was like "that of the may-flower or of apple-blossom, with a kind of *arrière-goût* of fæces." Dr Fagge, who rivalled the younger Babington in acuteness of smell, often observed this kind of smell, "especially when the dilated bronchial tubes were emptied with a gush of enormous quantities of fluid." Probably in such cases there is no active process going on in the walls of the affected tubes themselves. On the other hand, in many of those cases in which the odour is like that of gangrene of the lung, the development of fœtor in the sputa indicates the abrupt commencement of a destructive change, both in the air-passages and in the pulmonary parenchyma, which rapidly brings about a fatal issue.

The credit of having first pointed out the clinical features of cases of this kind belongs to Dittrich. His description is that it "commonly arises in persons of the middle period of life, who have suffered for years from bronchial catarrh, with abundant muco-purulent expectoration, and who may either have already begun to waste, or may still remain well nourished.

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* See a paper on the chemical characters of fœtid expectoration by Gamgee ('Edin Med. Journ.,' March, 1865), and one by Bamberger in the 'Würzburger med. Zeitschrift, 1864.

Suddenly, and without apparent cause, the sputum becomes offensive, of a dirty grey colour; the breath also stinks, poisoning the air around. Thereupon follow severe dyspnœa, fever of typhoid character, rapid collapse, an earthy, dirty yellow complexion, and ultimately cessation of expectoration, coma, and death."

After death the walls of some of the bronchial tubes are found sloughing, with more or less extensive areas of pneumonic consolidation, passing here and there into gangrene. Other parts of the lung tissue are œdematous, exuding a fœtid liquid. The bronchial glands are swollen, soft, and of a dirty grey colour.

The occurrence of hepatisation and gangrene of the lungs is by no means limited to the seats of bronchiectasis. Pneumonic patches may be scattered throughout every part of the lungs, and probably owe their origin to the inhalation into healthy tubes of putrid particles derived from others already diseased.

But the issue of putrid bronchitis is not always fatal. Slight cases sometimes end in recovery because either there was no ulceration of the walls of the tubes, or because the necrotic process was limited, and healing took place after detachment of the sloughs. Other cases, again, run on for months with but little change in the symptoms, and without marked impairment of the general health.

Ætiology of bronchitis generally.—The chief cause of primary or acute bronchitis is exposure to cold. No doubt cold air entering the air-passages through the mouth sometimes acts upon them as a direct irritant. The nasal mucous membrane warms the inspired air; but when persons go out of doors in cold and foggy weather, there is often catarrh of the nasal mucous membrane which compels them to breathe through the mouth.

Probably bronchitis may also be set up by the action of cold upon the surface of the body. A patient may "take a chill" by getting wet through, by sitting in a draught, by lying on damp grass, or by merely sitting out of doors when sweating after exertion. It is when the body has been heated and is cooling that there is danger of catching cold, because whenever the body produces more heat than is required to maintain its temperature, the thermotaxic centres act on the vaso-motor mechanism, and the cutaneous capillaries become dilated. Hence, after a Turkish bath, one plunges into cold water, which causes contraction of the blood-vessels, before one ventures to sit in a room at an ordinary temperature. Rosenthal, and afterwards Riegel, have shown by direct experiment that if animals after exposure to great heat are removed and placed in air which is not warmed, they go on cooling until their temperature falls below normal point.

In most cases the first effect of external cold is to set up sore throat and coryza, and then the inflammation spreads to the trachea and bronchi.

Some persons are far more sensitive to the action of cold than others. The risk of catching cold may, however, be often obviated to a great extent by "hardening" the skin, that is, by exposing it regularly to sudden changes of temperature, so as to accustom its vessels to contract promptly and vigorously. The most effectual way of doing this is to sponge the surface with cold water, or to use a cold douche every morning after a tepid bath. The cold bath by itself is all that is needed by robust persons with an active circulation, in whom it is followed by a good reaction. Even with young children a warm bath is best followed by rapid sponging with cold water.

Many secondary or predisposing causes of bronchitis are really conditions which favour the injurious action of cold. Thus the disease is very apt to attack young children, on account of their feeble powers of resistance. A curious point that came out in some inquiries made by Geigel into the infant death-rate in Würzburg was that bronchitis was less fatal to illegitimate than to legitimate children, the reason perhaps being that the latter are more coddled and kept warm, and so are more sensitive to cold.

The prevalence of bronchitis in Europe is, as might be expected, least in the hot season of the year, from June to September, and its proportionate frequency in different months is not the same as that of acute pneumonia. This is true also of the geographical distribution of the two affections. Bronchitis increases in frequency from the equator towards the poles, but the increase is not uniform in all longitudes, for it varies with the climatic conditions of each particular country. What favours it most is not a low mean temperature, but the occurrence of sudden and violent changes. In some parts of the tropics bronchitis is by no means uncommon at the end of the hot season. The countries in which it is of most rare occurrence appear to be Egypt, the western prairies of North America, the plains of India, part of the West Indies, and California.

The entrance of irritant substances into the air-passages during breathing is another and perhaps the most important cause of bronchitis. In discussing the ætiology of phthisis we shall have to consider the influence of various occupations, in which the inhalation of dust is almost inevitable. Such occupations frequently cause bronchitis, which may either be followed by the development of phthisis, or may run a chronic course without complication until it ends by the supervention of an acute attack, or by dilatation of the heart and dropsy. Pulverulent substances which happen to be coloured, such as carbon or oxide of iron, often tinge the sputum deeply. Yet a miner, whose lungs are black with coal-dust, may spit up a yellow muco-purulent fluid, containing no carbon whatever, when he is away from the mine. For the bronchial mucous membrane never itself becomes the seat of anthracosis; even the peribronchial tracts of fibrous tissue derive their black deposit from the surrounding pulmonary alveoli, and not from the bronchial epithelium.

The inhalation of gases, especially nitrous or sulphurous acid, is exceedingly irritating to the air-passages, and not infrequently sets up acute bronchitis in workmen whose occupations expose them to it. But, according to Hirt, the chronic form of the disease is comparatively seldom traceable to this cause; for after one or two acute attacks a tolerance seems to be established, and no further ill effects are observed. On the other hand the emanations from certain oil-works, from tar factories, and from the pans in which brine is evaporated to make salt, are believed to have a beneficial influence on the bronchial mucous membrane.

Quite apart from any irritation, whether thermal, mechanical, or chemical, we must recognise various contagia as causes of bronchitis, particularly those of measles, enteric fever, and septicæmia.

The *diagnosis* of Bronchitis rests on the presence of the symptoms and physical signs mentioned above. The only difficulty is to determine whether it is a primary affection, or secondary to fevers, pneumonia, tuberculosis, or diseases of the heart or kidneys. It is interesting to notice that Richards Brown, writing in 1795, gives the diagnosis of Bronchitis from Peripneumonia vera, Catarrhus pestiferus (*i. e.* Influenza), Pleurodynia, Angina pectoris, and Hepatitis.

The *prognosis* of Bronchitis rests on the following conditions:

When secondary to other diseases, as exanthems and other fevers, diphtheria, pneumonia, cardiac or renal disease, it is graver than when primary.

When it is confined to the larynx, trachea, and larger bronchi, it is seldom dangerous; when it invades the smaller tubes, and particularly when complicated by collapse, catarrhal pneumonia, emphysema, or bronchiectasis, it is much more grave.

The danger of uncomplicated bronchitis is limited to childhood and old age. At the two extremities of life it is, in this country, the most common cause of death during the winter months.

Occasionally, however, even in young adults, acute uncomplicated bronchitis is fatal. The writer has only twice seen this, once in a young man and once in a young woman, both between twenty-one and twenty-eight. In both cases he expected to find acute miliary tuberculosis, but in both cases an autopsy showed that this and every other complication was absent. Such cases are very exceptional. Indeed it may be almost absolutely stated that a reported death of an adult under sixty years of age from bronchitis was really due to phthisis.

Special treatment by drugs.—In the milder forms of acute *bronchial catarrh*—or tracheo-bronchitis—little is necessary beyond placing the patient in an equal temperature, of about 63°. Small doses of ipecacuanha, with neutral salts like nitre, are probably serviceable by favouring exudation from the inflamed mucous surface. The application of mustard plasters or of hot poultices to the throat and to the upper part of the chest often gives great relief to the sense of soreness along the trachea and behind the sternum. In slight cases inhaling steam is an efficient and agreeable remedy.

Very different measures are necessary in cases of *capillary bronchitis.* Sometimes, if suffocation appears to be rapidly impending and the right side of the heart to be overloaded, it is advisable to bleed from the arm. Antimony is often valuable for a day or two, and also the ethereal tincture of lobelia in half-drachm doses at frequent intervals. In one of Dr Fagge's worst cases life appeared to be saved by alternate doses of turpentine and of champagne. Ammonia in frequent doses is another most valuable drug.

It is important to maintain a moist state of the air round the patient. A kettle on the fire, with a long tube, throwing steam out near the patient's bed, fulfils this indication better than anything else. But when it is used, one must never forget that the ceiling and the curtains necessarily become saturated with damp, and that if the temperature of the room should be allowed to fall a few degrees during the night, or in the early morning, a chill will result, which may possibly be fatal to the patient. In some cases direct inhalation of steam gives much relief, or a medicated spray may be employed, containing conium juice, or morphia, or salines such as chlorate of potass or ammonium chloride.

After a few days it is generally necessary to substitute for nauseant drugs, like ipecacuanha or antimony, such remedies as carbonate of ammonia, squill, and senega; and to add brandy in accordance with the state of the pulse.

In *chronic bronchitis* a great variety of medicines are useful. If the cough is dry and hard, ipecacuanha is most serviceable, and to this squills and nitre are often usefully added. If it is distressing by its frequency and apparent aimlessness, bromide of ammonium or of potassium often gives relief, and, if the kidneys are healthy, morphia or opium may be taken with great advantage. The compound tincture of camphor is an excellent form to prescribe; and after a few hours' sleep the patient may wake greatly refreshed, and in all respects better. It is, however, always necessary to consider, before prescribing opiates in a case of bronchitis, whether one is likely to do harm by checking cough, and so preventing the tubes being emptied of their contents. If there is lividity, stupor, or even drowsiness, such medicines must be carefully avoided. In many cases, especially if the expectoration is viscid and abundant, sal ammoniac is useful; it may be given in doses of gr. xv to gr. xx, with a little syrup of lemon or extract of liquorice to conceal its disagreeable taste. Iodide of potassium is another salt which often does good service in chronic bronchitis.

In cases in which there is excessive exudation and secretion from the surface of the mucous membrane balsamic remedies are applicable—tolu, benzoin, Peruvian balsam, benzoic acid; other internal remedies are copaiba, turpentine, ammoniacum, or one of the fœtid gum-resins, such as assafœtida.

The treatment of *Bronchiectasis*, and particularly of fœtid Bronchorrhœa, by internal remedies is seldom satisfactory. Antiseptics, such as creasote, carbonate of guaiacol, terebene, thymol, may be given as frequently as they can be taken in pills or capsules, and sometimes succeed in removing fœtor and checking secretion; but more often the patient or his stomach rebels, and the drugs have to be laid aside.

Dr Poore in these cases, and also in chronic phthisis with profuse secretion from vomicæ, has advocated the patient eating garlic until its odour is perceptible in the breath and the skin. He finds it very useful in replacing the foul odour of the breath and sputa by a pungent smell like that of syringa; and Dr Ewart testifies to the value of the treatment.

Putting the patient in a chamber filled with the vapour of heated creasote for ten or thirty minutes, or even longer as he becomes used to it, has been tried by Dr Chaplin, Dr Ewart, and some other physicians with success. The eyes must be protected from the fumes. The smell is penetrating and diffusive, and the cough excited severe; but the result seems to be effectual emptying and purification of the dilated tubes.

A more convenient and easier—but it must be confessed, less effectual—method is that of using medicated insufflations, or sprays or inhalation of vapours impregnated with turpentine, thymol, terebene, compound tincture of benzoin, iodine, or creasote.

Several years ago Grainger Stewart proposed and carried out the direct injection of drugs dissolved in olive oil. He used 10 parts of menthol and 2 of guaiacol in 100 of oil, injected twice a day into the trachea through the larynx; and succeeded in curing a case of fœtid bronchiectasis which had resisted other methods of treatment.

Incision and drainage of a large bronchial cavity has been performed, but with less favourable results than similar treatment of a large single adherent vomica in a case of phthisis.

Sulphurous waters are believed by those who prescribe them to be of service in fœtid bronchiectasis, and patients are sent to Harrogate and to the Pyrenees to drink them.

General treatment of bronchitis by air and waters.—Of late years several physicians, first in Germany and since in this country, have made use of both compressed and rarefied air in the treatment of various bronchial and pulmonary affections. In the earlier attempts recourse was had to pneumatic chambers, made somewhat after the fashion of the diving-bell, in which the patients sat for an hour at a time, under a pressure of $1\frac{3}{7}$ to $1\frac{3}{5}$ atmospheres. It is obviously in very exceptional circumstances only that such elaborate constructions can be available in practice, and therefore attention has been more recently devoted chiefly to the invention of portable forms of apparatus, by which the patient is made to inhale air of varying degrees of pressure without being himself immersed in it. Most of these instruments are upon the principle of the ordinary gasometer used to receive coal gas; an air-containing cylinder, open below, is suspended in another cylinder, open above, so that the one can move freely up and down within the other. By pouring more or less of water into the outer cylinder, and then either pressing down the inner cylinder with weights or lifting it up to varying heights, the air inside it may be compressed or rarefied to any desired extent. The object is to make the patient *inspire* compressed air, or *expire* into rarefied air. A tube from the inner cylinder is connected with a mask, which can be fitted air-tight over the nose and the mouth. There is a stopcock, which is turned by the patient each time he breathes, so that the mask communicates with the cylinder either during inspiration or during expiration (as may be intended), whereas on reversing the movement it communicates with the external air. Waldenburg, who invented this machine, advised that compressed air should be inspired for five, ten, or fifteen minutes, and then, after a pause, that expiration into rarefied air should be practised for a similar period. In most cases two or three sittings a day are sufficient.

So far as emphysema is concerned, the greatest benefit is to be derived from expiration into rarefied air. The inspiration of compressed air might be expected to tend towards increasing the distension of the alveoli. But, on the other hand, expiration into rarefied air might also do harm by augmenting the flow of blood to the bronchial mucous membrane, whereas inspiration of compressed air would increase the blood-pressure in the systemic vessels, and unload the right side of the heart. It is therefore best to alternate the two methods, as Waldenburg and others advise. There is strong testimony to the beneficial action of this mode of treatment, in augmenting (at least for a time) the activity of emphysematous lungs, and in relieving the symptoms of bronchial catarrh. (See Dr Theodore Williams' three lectures in the 'British Medical Journal,' April 18th, 1885; Dr Gamgee's paper, *ibid.*, December 18th, 1886; and the fourth chapter in Dr Hale White's book on non-medicinal therapeutics, 1889.)

Inhalation of oxygen gas is often of signal, though only temporary, benefit.

In some cases of chronic bronchitis it is the habit to send the patients to certain Continental spas. According to Braun those waters are the best which contain a considerable amount of chloride of sodium, as well as of carbonate of soda, such as Ems in Germany and Mont Dore in France.

Probably the same benefit is derived from drinking the waters of Bath or Matlock.

The diet of patients suffering from chronic bronchitis requires regulation. Rather free diluents are in most cases desirable, and probably the mineral waters just mentioned act chiefly in this way, promoting free and loose secretion from the bronchial mucous membrane. By common experience beer does harm, and even London labourers with a cough avoid porter, and drink gin instead. Cheese is also believed to be bad for a cough; and Pliny's dictum—*nuces tussientibus inimicæ*—still holds good, probably from the mechanical irritation of the fauces which they produce.

When it is thought desirable for a bronchitic patient to spend the winter and spring away from home, the choice lies usually between climates which are soft and "sedative" (such as Torquay, Falmouth, Penzance, and the Channel Isles, Montpellier, Madeira, the Canaries, and Egypt), those which are stimulant without too much risk of exposure to cold winds (as Mentone and San Remo, or Ajaccio in winter, and Margate, Folkestone, and Lowestoft in summer and autumn), and those which are surrounded by pine woods (as Arcachon and Bournemouth).

PLASTIC BRONCHITIS.*—This singular disease has characters so peculiar that it cannot be grouped with the ordinary catarrhal forms of bronchitis. It consists in the exudation of a plastic material from the walls of the air-passages which coagulates and forms fibrinous "casts" of their channels. A like exudation may occur by extension of a morbid process downwards from the larynx in diphtheria, and much less frequently by extension upwards from the pulmonary alveoli in pneumonia; but both of these are totally different in pathology and symptoms. The former has been described with membranous laryngitis in the chapter on Diphtheria, and depends on infection by Löffler's bacillus. The latter depends on infection by the diplococcus of Pneumonia.

Plastic bronchitis is one of the rarest diseases—"an affection of great rarity" (Walshe); "höchstselten" (Riegel); "äusserstselten" (Biermer). The experience of Sir Thomas Watson was remarkable, in having had under his own observation five well-marked examples.

These "bronchial polypi"† were known very early. Dr Nicholas Tulp (the lecturer in Rembrandt's famous painting, "The Lesson of Anatomy") records and figures two specimens brought up by a Dutch sea-captain suffering from hæmoptysis: "*Effudit duos insignes venarum ramos adæquantes singulos expansæ manûs magnitudinem*" ('Obs. Med.,' Amst. Elz., 1652, cap. xiii, p. 122, tab. iii, iv). Afterwards cases were recorded by the younger Bartholin, Cheselden, de Haen, Morgagni, Hunter,‡ Cheyne of Dublin, and Stokes. Most of the early cases (1690—1730) were published in the 'Philosophical Transactions.' An excellent account by Peacock, based on thirty-four cases, appeared in the 'Path. Trans.' for 1853 (vol. v, p. 43). Biermer collected fifty-eight cases ('Virchow's Hdbh.,' 1867), and Dr Samuel West fifty-two ('Practitioner,' vol. xliii, 1886).

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* *Synonyms.*—Fibrinous bronchitis—Croupous bronchitis or Bronchial croup of some German writers—Bronchial polypi of older authors—Angina polyposa.

† The word "polypus" in the medical literature of the seventeenth and eighteenth centuries was not used in its present application to pedunculated tumours of the nares, or uterus, or rectum, but in its original signification, which survives in zoology—of a body with many feet (an octopus or a fresh-water polyp),—and was applied to the fibrinous coagula found after death in the trunk and branches of the pulmonary artery.

‡ Hunter's case occurs as a short appendix to his 'Treatise on the Blood, Inflammation, and Gunshot Wounds.' The patient was a man of twenty-two, who spat mucus often mixed with blood. He recovered. The figure agrees with those illustrating the cases of Fuller, Peacock, Salter, Tuckwell, and Fagge, in the 'Pathological Transactions' (vols. v, ix, xvi, and xxi).

Anatomy.—In almost all cases of plastic bronchitis the patient expectorates masses of the peculiar exudation. It usually appears rolled up into a sort of ball, and covered with mucus and blood. This is easily removed by floating it out in water, and one then sees that there is a complete cast of some part of the bronchial tree, extending perhaps down to its finest subdivisions, so that, according to Biermer, the minute terminal filaments may actually show bulbous ends moulded in the infundibula themselves. The colour of the cast is whitish yellow or grey; its consistence is tough and elastic; it is almost always made up of many concentric laminæ, separated here and there by narrow spaces and with more or less definite central cavity, containing mucus or bubbles of air. The more delicate filaments are said to be generally solid.

The laminated structure affords a distinction from the branching clots which are sometimes formed in the air-passages as the result of hæmorrhage, and which are quite homogeneous. Biermer doubted whether blood ever coagulates so as to form casts of the bronchial tree: but Walshe spoke of it in positive terms, and there could hardly be a better authority.

The casts in cases of fibrinous bronchitis, when examined microscopically, are seen to consist of a hyaline or slightly fibrillated base, in which are embedded large numbers of leucocytes. They seldom contain red blood-discs in any quantity. Several observers have noticed Charcot's crystals in them, and also Curschmann's spirals. In one case Waldenburg found that the thicker parts contained only a few formed elements, but abundant fat-globules.

The material is spoken of as coagulated fibrin, like that of inflammatory exudations on serous membranes. But in some cases it appears to be soluble in lime water or solutions of potass or soda: and this character brings it nearer to mucin.

The length of a bronchial cast is commonly from one and a half to two and a half inches, but sometimes it may be four or five inches, or even (as in a case of Riegel's) over six inches. The diameter of its thickest part is seldom greater than that of a goose-quill—much less than that of the space in which it was formed. Biermer has pointed out that from its appearance one can sometimes draw an inference as to the part of the lung from which it came, whether from the short rapidly branching tubes of the upper lobe, or from the comparatively longer tubes of the lower lobe. The masses expectorated at different times by the same patient often resemble one another so exactly in size and in the arrangement of their subdivisions as clearly to show that they have all in succession been derived from the same tract of mucous membrane. For example, in a case recorded by Kretschy seven casts appeared one after the other, all of which came from the middle and lower lobes of the right lung. In fatal cases it is not usually found that the tubes which have poured out the fibrinous exudation show any marked morbid changes. The mucous membrane is sometimes reddened, sometimes pale, but always healthy-looking. In two instances Biermer found the epithelium still remaining beneath loose casts; but he was himself disposed to think that these cases might be exceptional; and Kretschy has since stated that in his case there was no trace of epithelium in that part of the air-passages which contained the plastic material. The submucous tissue may be swollen and infiltrated with serum. The pulmonary alveoli are usually unaffected, but they have been found sometimes collapsed, sometimes over-distended.

Symptoms.—The expectoration of casts of the lower air-passages is generally attended with severe cough and dyspnœa; and this may be the first indication that the patient is ill. But in many cases there is an antecedent stage of ordinary bronchial catarrh; and this may last for a long time. Sometimes the disease sets in with rigors, loss of appetite, thirst, oppression of the chest, and pyrexia, so that an attack of pneumonia seems impending. Presently a dry, hard, and painful cough appears, and the breathing becomes rapid, up to 40 or more in the minute, attended with the anguish of impending suffocation, with lividity, and with a small tense pulse. There may be some pain in the side, and a feeling of soreness within the chest, but on the whole the attack is more distressing than acutely painful. At first nothing is expectorated, or only a little mucus. The cough may even, it is said, go on for days before any fibrinous masses appear. More often a cast is detached and got rid of after a few hours, and by this the cough and dyspnœa are generally relieved, at least for a time. Hæmoptysis often occurs at intervals during the paroxysm, not only at the time when the cast is being expectorated, but also previously. The quantity of blood is not large; perhaps it amounts to a tablespoonful at a time. The case of the late Prof. Daniell, recorded by Watson, is exceptional in the fact that from two to eight ounces were spat on each occasion; and it appears that in more than half the cases there is little or no hæmorrhage.

Physical signs.—Examination of the chest throws little light upon cases of plastic bronchitis. If a large tube is blocked, absence of the pulmonary murmur may be made out over some part of one of the lungs. The fact that the violent cough fails to clear away the obstacle might perhaps suggest the presence of something more than a plug of mucus, and the diagnosis as to the cause of the obstruction would then lie between fibrinous casts, a foreign body, and stenosis of the trachea. But, as a rule, no suspicion of the real nature of the case arises until a cast has been expectorated. There is not usually any change in the percussion-sound, but Walshe had repeated occasion to observe dulness, as complete as that of pneumonic consolidation, and dependent upon collapse of the lung-substance; and he adds that local pneumonia now and then occurs, attended with crepitation, bronchial breathing, and rusty sputa. When many tubes are blocked, the movements of the corresponding side of the chest may be impeded, and the lower ribs may be drawn in during inspiration. Râles are sometimes audible over the affected part of the lung, especially when the cast is becoming loose; they are "large" and non-consonating, without any distinctive character.

Course.—The expectoration of a single cast very rarely brings an attack of plastic bronchitis to an end. As a rule the relief is only temporary. After some hours the cough and the dyspnœa return, and are followed by the appearance of another cast. This process is usually repeated about once in twenty-four or in forty-eight hours for several days, and then the affection slowly subsides. Smaller pieces may be spat up at very frequent intervals; but being embedded in mucus, they sometimes remain unnoticed unless specially looked for.

Prognosis.—It may well be supposed that the expulsion of such large masses as sometimes come from the air-passages in this disease is not unattended with danger. In 1865 Dr Fagge showed to the Pathological Society a cast which was taken from the body of a girl aged seven, having been found lying across the bifurcation of the trachea, with its branches extending into the ramification of the right bronchus, but with its broad end occluding the left bronchus. She had been expectorating similar masses for three days; and on the very day on which she died she had already, at 3 a.m., brought up a cast of about the same size as that which killed her at 3 p.m. in a violent fit of cough and dyspnœa. It is therefore clear that Walshe and others went too far in giving a favourable prognosis in cases of plastic bronchitis without reservation. Lebert, in a paper in the 'Deutsches Archiv' for 1869, divided 44 cases (collected from various sources) into acute and chronic: of seventeen of the former, four ended fatally, of twenty-seven of the latter, only three; but one of the four fatal acute cases, that recorded by Nonat, was probably a case of diphtheria. As a rule, when death occurs, it is caused by extension of the disease into so large a part of the bronchial tree that cyanosis follows, with stupor and somnolence. Riegel, however, relates a case in which, although the patient died in an attack of asphyxia, after spitting up large casts nearly every day for three weeks, the air-passages were all found empty after death.

In a certain number of cases plastic bronchitis has been observed to end in phthisis.

Lebert placed in a separate category cases in which plastic bronchitis has run on to a fatal termination without expectoration of casts. They occurred chiefly in children, and generally in association with bronchopneumonia after measles.

Recurrence.—When an attack of plastic bronchitis has passed off, leaving the patient apparently well, it by no means follows that the disease is at an end. One of the most curious points about it is its liability to return again and again at irregular intervals, sometimes during a very long period. Walshe met with an instance in which the expectoration of casts continued, with occasional brief intermissions, from the spring of 1843 to June, 1857, when he lost sight of the patient. Other physicians have recorded cases which were almost as protracted; and often the general health seemed to be scarcely affected.

Ætiology.—With regard to the causes of plastic bronchitis, scarcely anything can as yet be said. It is much more common in males than in females. The period of life at which it is most frequent is between ten and thirty. One case, recorded at the advanced age of seventy-two, had lasted seven or eight years.

Adding to the 31 cases tabulated by the late Dr Peacock in the fifth volume of the 'Pathological Transactions' 24 additional ones collected from various sources, we find that of these 55 cases, 42 occurred in men and 13 in women. (Biermer's figures are 39 male to 19 female patients.) Of 37 patients whose ages are given, 5 were between five and ten, 12 between eleven and twenty, 10 between twenty-one and thirty, 8 between thirty-one and fifty, and 2 between fifty-one and sixty.

The writer has had only two typical cases under his care, one a boy of twelve, about 1870, who spat up repeated casts with some amount of blood, and recovered; the other a man of forty-three, who died of acute pneumonia (May, 1890) in the course of one of repeated attacks of bronchitis with expectoration of large fibrinous casts. A third case (published in the

'Pathological Transactions' for 1880, p. 30), in a woman of thirty-two, was fatal after tracheotomy, but here the trachea and primary bronchi were affected, so that a continuous fibrinous cast was brought up; and clinically as well as pathologically the case was peculiar.

A remarkable circumstance, all the more striking because of the extreme rarity of plastic bronchitis, is its occurrence in different members of the same family. Fuller met with it in two sisters; and Watson relates the cases of two brothers, both of whom were affected within a twelvemonth.

The disease is supposed to be rarer in southern countries than in the north of Europe. Riegel says that, like acute pneumonia, it is most apt to occur towards the end of spring, when there are great daily variations of temperature. In one instance the recurrence of the attacks appeared to be connected with the catamenial periods. Eisenlohr met with a case in which fibrinous casts were expectorated during the second week of enteric fever.

Treatment seems generally to be altogether ineffectual. Waldenburg, however, saw a case in a girl, aged eight and a half, who for more than four years had been coughing up fibrinous masses at intervals of a few days, and in whom a whey cure and the daily inhalation of lime-water succeeded in arresting the disease in six or seven weeks. Indeed, a spray of lime-water, or a solution of an alkaline carbonate, should always be employed in plastic bronchitis; the only doubt is whether they reach the lower air-passages in sufficient quantity. Emetics appear to be sometimes useful: probably it is best to use apomorphia hypodermically. Biermer recommends an active mercurial treatment; and others have prescribed iodide of potassium with apparent advantage. Dr Ewart speaks favourably of injections of olive oil into the trachea, so as to facilitate the expulsion of the branching coagula.

Walshe held that neither inhalation of iodine, nor exhibition of alkaline medicines, nor the best of general health, nor the most favoured climates, have the least effect in preventing or curing the attacks of this paradoxical disorder.

DISEASES OF THE PLEURA

"Side-stitches that shall pen thy breath up."
The Tempest.

PLEURISY—*Its anatomy—Its physical signs—Pleuritic effusion—Compressed and carnified lung—Dulness—Ægophony—Symptoms—Course and events—Empyema—Diagnosis—of dry pleurisy—of serous effusion—of empyema—Hydrothorax—Hæmothorax—Pleuritic effusion in children—Complications of pleurisy—Its ætiology and relation to tubercle—Prognosis—Treatment of pleurisy and of empyema—Paracentesis and its results.*

PNEUMOTHORAX—*Origin and pathology—Post-mortem characters—Physical signs—Diagnosis and symptoms—Prognosis—Treatment.*

PLEURISY or pleuritis* is mentioned by Hippocrates, and by other Greek writers, who meant by it pain in the side. Afterwards, the term, by a natural metonymy, was transferred from the symptom to its more usual cause, inflammation of the pleura. This, however, only meant at first what we now call the parietal pleura. The true anatomy of pleurisy and its recognition by percussion and auscultation were only discovered in the nineteenth century.

Anatomically, pleurisy closely resembles inflammations of the other serous membranes. The earliest morbid appearance is said to be injection of the smaller blood-vessels; but few can have seen this. As a matter of fact, one often finds patches of recent lymph on the surface of a lung without any reddening, when pleurisy has set in shortly before death from pneumonia or rheumatism or injury. Hyperæmia may accompany inflammation, but does not occur separately from it.

Exudation of coagulable lymph on the opposed surfaces of the serous membrane occurs at the first onset of an attack of pleurisy, of which it forms the "plastic stage." If the morbid process stops here, the case is said to be one of dry pleurisy. When the inflammation afterwards subsides, the fibrin sometimes undergoes complete absorption, leaving the pleura in its natural condition, or only a little dull and opaque. But more often the two surfaces have meantime cohered, and remain henceforth connected by separate bands or by a uniform layer formed of connective tissue, well supplied with blood-vessels. If the plastic stage, as is more often the case, is succeeded by exudation of serum, the case becomes one of pleurisy with effusion, and this may be serous or purulent.

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* Πλευρῖτις, sc. *νόσος*, *i. e.* the "side-complaint," the stitch in the side. The adjectival termination, *ιτις*, has been taken from this word and from *φρενῖτις*, and applied to denote the most common of complaints, inflammation, as in the modern terms Peritonitis, Nephritis, Orchitis, and many less legitimate forms; but there is no such connotation in the Greek.

Pleurisy is the most frequent and typical example of what was described in an introductory chapter as serous inflammation (pp. 52, 55). The pleura is only a part of the membrane which once formed a continuous mesoblastic lining of the other subdivisions of the original pleuro-peritoneal space or cœlom. Hence it is not surprising that the nearest pathological allies of pleurisy are pericarditis, peritonitis, and hydrocele. In a more distant degree it is related to endocarditis, synovitis, and lepto-meningitis.

The three principal kinds of pleurisy we shall find reproduced in the other serous membranes: watery, lymphatic, or *serous* effusion, with few leucocytes and little fibrinous coagulation; *plastic*, with formation of fibrin from the "coagulable lymph;" and *purulent*, with abundant exudation of leucocytes. The two former are "irritative," due either to injury, to exposure, or to such primary conditions as rheumatism, Bright's disease, or pneumonia, or to the local presence of tubercle or cancer. The latter is septic, and always associated with the presence of pyogenic microbes. We frequently find double pleurisy complicated with pericarditis, and less frequently with peritonitis also. Such cases are most often met with in the course of tuberculosis, particularly in children, or secondary to Bright's disease, or to cancer, or less frequently due to repeated rheumatic inflammation. But occasionally they are seen without any such explanation.

The writer once had a striking case of this condition, in a lad who, after several years' illness, died. At the autopsy the pleura, pericardium, and peritoneum were all much thickened, and there was double hydrothorax, hydropericardium, and great ascites, which communicated with the tunica vaginalis on the right side by an unclosed isthmus. When the serous membrane was stript off the viscera, the lungs and heart, the liver, kidneys, testes, and other organs were found to be perfectly healthy. No tubercle could be discovered anywhere.

Dr Frederick Taylor has described such cases, and an Italian translator of his 'Manual of Medicine' has proposed for them the term *Polyorromentitis* ('Brit. Med. Journ.,' Dec. 15th, 1900).

Signs of dry pleurisy.—Auscultation furnishes a sign which is of itself almost conclusive as to the presence of lymph on the pleura. This is the "friction-sound" or "pleuritic rub," due to the movement upon one another of the two roughened serous surfaces. Hippocrates described the pleura as "creaking like leather." Yet Laennec, though he must have often heard this sound, failed to understand its meaning, and supposed that it indicated emphysema, especially what he described as interlobular emphysema.* Its right interpretation was reserved for Raynaud.

The most characteristic pleuritic rub consists of an irregular succession of short, harsh sounds, which give one exactly the impression of something catching or dragging against an obstruction and then slipping, but only to catch or drag once more. It is more or less interrupted, toneless, and loud or "near." It has been compared to rubbing cloth together or to tearing a piece of thick paper. The patient himself is often conscious of a rough, grating sensation each time he breathes: and sometimes one can *feel* the rub by placing one's hand over the affected part of the chest, *i. e.* the vibrations which affect the ear also affect the hand, just as a sonorous rhonchus or a loud cardiac bruit may be appreciated as a tactile fremitus or a thrill. Sometimes a rub accompanies both inspiration and expiration, and sometimes it is to be detected only at the end of a deep inspiration, when the lungs are most expanded. It is probably never expiratory alone.

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* There are still some who think that a friction-sound may be due to emphysema (p. 1068), or to miliary tuberculosis of the pleura. Walshe believed that a friction-sound may be heard without any lymph, from "simple vascularity" of the pleura.

Another kind of pleuritic rub is equally diagnostic. Instead of resembling the tearing of paper, it is, as Hippocrates heard it, just like the creaking of the sole of a boot. The "grating" rub is more like a râle; the creaking rub more like a rhonchus.

A rub may be heard within twelve hours from the beginning of the disease, and in cases of dry pleurisy it may persist for days or possibly even weeks with but little alteration. But as a rule it disappears after a short time, because serum is effused and keeps the two surfaces apart. If the inflammation is spreading, it may, after it has ceased to be audible at one spot, be discovered at another, but a rub is not often present over a large area at once. The part of the chest at which one is most likely to hear it is in the axilla, outside the nipple, or in the back outside the angle of the scapula. The reason is not only that pleurisy more frequently affects the surface of the lower lobe than that of the upper, but also that the descent of the diaphragm causes a movement of the pulmonary upon the costal pleura, which is wanting elsewhere. Occasionally, however, a rub can be heard over the front of the chest as high as the clavicle.

A well-marked rub with respiratory rhythm may be due not to pleurisy, but to peritonitis, particularly in the right hypochondrium. In his well-known paper on the "Difficulties and Fallacies attending Physical Diagnosis," Addison cited a case in which lymph on the adjacent surfaces of the liver and of the parietal peritoneum caused a "crepitus, which closely resembled a mucous rattle" ('Collected Works,' p. 87).

A pleuritic rub may have a cardiac rhythm if it be due to lymph effused on the anterior margin of the left lung. Again, a pleuritic friction-sound may be so "ill-marked" as we say, *i. e.* may vary so far from the characteristic grating or creaking, that it is impossible for the ear of the most practised auscultator to distinguish it from a mucous râle or a squeaking rhonchus.

The locality, with the fact that coughing does not alter the sound, helps us to a correct diagnosis. A rub is very rarely heard near the apex; it is undisturbed by expectoration, and it is most often heard at the end of inspiration, instantly checked by the pain which is felt, never during expiration alone.

The only other physical signs of the plastic stage of pleurisy are a certain degree of impairment of movement of the affected side of the chest and a corresponding enfeeblement of the respiratory murmur.

Pleuritic effusion.—In most cases of pleurisy liquor sanguinis (plasma or coagulable lymph) is effused into the serous cavity, often in large quantity and with great rapidity. Four or even six pints are not uncommonly found, and Watson cites a case in which Crampton, of Dublin, drew off from the left pleura as much as fourteen pints. The plasma or "lymph" is usually translucent and of a yellow colour, perhaps containing a few shreds of fibrin. It has an alkaline reaction; according to Fräntzel its specific gravity may vary within wide limits, from 1005 to 1030; but it is most of between 1015 and 1025.

In other cases it is more or less opaque, and on standing throws dow layer of greenish-yellow pus. If the effusion is decidedly purulent it called an *empyema*.*

There can be no doubt that the formation of pus in the pleural cavit often a gradual process, the liquid being at first serous or sero-purulent, a becoming more and more opaque as the leucocytes in it increase in numbe in such cases both the parietal and the pulmonary surfaces are coated w layers of fibrin. But there is also no doubt that primary suppuration m occur, particularly in cases of septic abscesses in other parts of the bo and the pleura may remain smooth and shining.

A pleuritic effusion may be tapped and pure serum escapes, a seco time it is perhaps somewhat turbid, and on a third tapping purulent. T essential condition for suppuration in the pleura, as elsewhere, is the p sence of pyogenic organisms, streptococci, staphylococci, or occasional pure pneumococci. Of these, the first are the most common, and the la the next in frequency; or there may be a mixed infection, as perha always in cases of tuberculous empyema.

In many, perhaps in most, cases of empyema, serum, coagulable lymp and pus are effused together; and, whenever this is the case, not only there more or less soft, shaggy fibrin on the visceral and parietal pleur but the two layers form adhesions, and thus the exudation is circumscribe One compartment may contain pus, and another more or less purule serum. Such a circumscribed or loculated empyema is often found in th lower part of the chest, sometimes between the lung and the diaphragm sometimes between two lobes of a lung, and, very rarely, at the apex.

Purulent, as compared with serous pleuritic effusion, is far mo common in children than in adults. It is the rule after admissio unsterilised air to the pleura and in septic or traumatic cases, and when secondary to variola, but it is rare when secondary to rheu fever, to Bright's disease, or to cancer, and not common when of tuberc origin.

When the effusion is not inflammatory at all, but passive, as in ca of general dropsy from disease of the heart, it is without pus, or blo or fibrin, when drawn off, and if it forms a coagulum on standing this voluminous, light, and flocculent. This condition is distinguished as drop of the pleura or hydrothorax.

Sometimes a pleuritic effusion is of a deep purple colour, or brigh clear red, from admixture of blood. This may be due to the fact that t patient is suffering from scurvy or purpura; or it may depend upon t presence of tubercles, or more frequently of cancerous nodules, scattered the pleural membrane.

Occasionally when the pleura is tapped, the fluid is found to be opa and milky. This is due usually to the presence of minute oil-drops wh form an emulsion with the alkaline serum, or to particles of a fibri nature, which cause a granular opalescence. Such chylous hydrothorax is comparable to chylous ascites and hydrocele, with which it may co-exist, and depends no doubt in most cases on rupture of lymphatics, or of the thoracic duct itself into a serous cavity; but occasionally no evidence of such a rupture has been discovered. (See 'Lancet,' 1879, i, p. 834, and 1890, ii, p. 875.) The writer has seen only one case of this curious condition besides the following.

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* Empyema in Hippocrates applies to any collection of pus, external as well as intern In England the meaning attached to the word *empyema* is, that of a collection of pu the pleural cavity. But on the Continent, in spite of etymology, collections of serous fl and even of blood, are included under the same name (see Littré and Robin's 'Dictionary It has also been used as a synonym for thoracocentesis, so that when the pus escap through an intercostal space, and has to be let out with the knife, an *empyema necess* is said to arise. These misapplications of a recognised term are surely inexcusable.

A man of fifty-seven was admitted into No. 22, Philip Ward, in April, 1897, with ascites and effusion in the left pleura. He had also symptoms that pointed to either cancer or cirrhosis of the liver. In May the ascites had increased and he was tapped. Sixteen pints of chylous fluid were drawn off on the 8th, and twenty-two pints, of the same milky aspect, on the 22nd. He died on June 4th, much emaciated and slightly jaundiced. There was found after death some more of the chylous effusion in the peritoneum, which showed no signs of inflammation, and the fluid found in the pleura was of exactly the same pale, opaque, milky character. There was cancer of the pancreas, not involving the head of the gland, with secondary deposits in the mesenteric and lumbar lymph-glands, but not in the lungs. The effused liquid was alkaline, and beside albumen contained a considerable amount of glycogen. The minute particles which gave it its opacity were chiefly proteid, not fatty in reactions.

Liquid pleuritic effusion usually gravitates into the most dependent part of the serous cavity. At the commencement of the attack, lymph may have covered the front and side of the lung; but when serum or seropurulent fluid is poured out, it settles in the back and lower part of the chest if the patient is in bed, or on the sofa. If, however, parts of the lung have previously become fixed to the chest-wall by a former attack of pleurisy, or if adhesions rapidly develop, liquid may accumulate between the lung and the pericardium, or between two lobes of the lung itself, without there being any at the base. Even when the whole of the pleura is affected, the seat of a serous or purulent effusion may be more or less irregularly circumscribed.

In one case recorded by Dr Fagge, there was a broad adhesion to the lateral region of the chest and another to the diaphragm, so that the serum filled the upper part of the pleural cavity while crepitant lung-tissue still existed in the lower part. In another case there was liquid at the base behind, and also above the root of the lung, with an intervening zone where the lung was perfectly adherent.

A necessary consequence of the presence of liquid in the pleural sac is that the lung becomes *compressed*, reduced in size, and at last emptied of its blood as well as of its air by effusion. The elasticity of the pulmonary tissue must lead to its receding as soon as the pressure in the pleural cavity exceeds that of the atmosphere, until it has become collapsed to the same extent as when air is admitted into the serous cavity after death. But, further, Lichtheim proved by experiment (cf. p. 1056) that the elasticity of the lung does not become exhausted until the alveoli are completely emptied of all their gaseous contents. The reason why a lung is not rendered altogether airless when the pleural sac is laid open in the dead body seems to be partly that the walls of the smaller tubes presently fall together, and partly that the tenacious mucus always present offers resistance to the further escape of air, which the elasticity of the pulmonary tissue is unable to overcome. During life an additional force is in operation to empty the alveoli of air, namely, absorption by the blood-current circulating in their walls. A lung undergoes compression by pleural fluid until, when the cavity is full, it is absolutely free from air; but beyond this point it may become further compressed until the blood is also driven out of its vessels. A lung which is bloodless as well as airless may be said to be *carnified*, while a lung which is merely airless may be spoken of as *collapsed*. A carnified lung has a very peculiar appearance; it has a slaty-grey tint, described as "mouse-coloured." Its cut surface is smooth, very firm, and dry, showing the flattened orifices of air-tubes and vessels closely packed together. Sometimes, if bronchitis has existed as a complication, the tubes contain pus; and if pneumonia or œdema should happen to be present, its characters are modified. The position occupied by a lung entirely emptied of blood and air by pleural effusion is, as a rule, determined by its root; it becomes flattened backwards towards the spine, and if covered by a mass of false membranes its very presence may be overlooked. When, however, the lung has previously been firmly fixed by adhesions, it cannot thus be driven inwards, and it may lie in the summit of the pleural space, or be irregularly pushed to one side, or even downwards.

When pleural effusion is insufficient to drive out the air from the whole of the lung, the effect is remarkable. Instead of a gradual and uniform shrinking of its substance, so that all parts would contain less air than before, without any becoming completely airless, the compression is complete, but partial. As Moxon long ago pointed out, the presence of even a few ounces of liquid in any part of the pleural cavity causes a total collapse of the pulmonary tissue which ought to occupy that space. Dr Fagge repeatedly verified his observation; and Cohnheim ('Vorlesungen,' vol. ii, p. 190) arrived at the same conclusion. A small triangular area of completely collapsed lung may be seen at the lowest corner of the organ, or a thin strip of it along its posterior margin. Nay, a mere enlargement of the heart, without there being any pleural effusion, may cause complete collapse of the inner surface of the left lung; and distension of the abdomen, thrusting up the diaphragm, may cause a similar affection of the lower surface either on one side or on both. The explanation of these remarkable facts depends on the mechanics of collapse of the pulmonary tissue, which were discussed in the chapter on bronchitis (*supra*, p. 1057). The general principle is that, whenever a part of the lung is no longer acted upon by the inspiratory forces, it becomes airless, notwithstanding that the tubes which serve it remain patent.

The production of local collapse of the lung-substance as the result of the effusion of moderate quantities of liquid into the pleural cavity has important clinical bearings. It accounts for a fact well known in the wards, that changes of posture of the patients often fail to alter the position of the liquid within the chest, so far as one can tell by percussion. In fact it is quite the exception to find evidence of gravitation; and even when one can alter the level of dulness by making the patient sit up, the alteration does not amount to more than a finger's breadth or two. The contrast is very great between these results and the free gravitation in cases of hydro-pneumothorax. The truth is that if a part of the lung is rendered airless by pleuritic effusion, the fluid may be, as it were, *held up* in a fixed position in opposition to the force of gravity.

When empyema fills the pleural cavity, the ribs may be obviously wider apart, and the spaces between them may bulge; but this is seldom or never observed in the case of serous effusion. On measurement one generally finds, if there is much liquid, that the affected side is enlarged, and sometimes the difference between the two halves of the chest is considerable. In determining this Dr Gee's cyrtometer is very useful. The play of the ribs in respiration is greatly impaired, much more so than during the plastic stage of pleurisy. Moreover, in consequence of the sternum being carried forwards, the movement even of the unaffected side may be impeded.

Displacement of the organs.—Long before the lung has undergone complete compression, other adjacent structures feel the pressure of the effused fluid. The mediastinum is pushed over to the opposite side, the elasticity of the unaffected lung no doubt assisting to displace it. Thus if the pleurisy be on the right side, the impulse of the heart is felt and seen during life to be situated further to the left than usual, until it may be at some distance beyond the left nipple. If the effusion be on the left side, the impulse may be felt in the epigastrium, or between the sternum and the right nipple, or even beyond the nipple, while in its normal position no trace of pulsation can be detected. Some observers have supposed that in such cases the heart swings over like a pendulum, and that its long axis is now directed downwards and to the right, so that the part which beats against the chest wall is still the apex of the left ventricle. But nothing seen in the deadhouse supports this opinion. Probably the displacement of the heart is attended with little change in the inclination of its axis, and the impulse is given by the right ventricle.

Displacement of the heart is much more obvious and extensive when the effusion is in the left pleura than when in the right.

The cardiac sounds have occasionally been found altered with a large effusion into the pleura. Hope heard a systolic murmur over the aorta, which disappeared when the pleuritic fluid underwent absorption. Walshe met with a case in which each sound of the heart was more or less masked by a blowing murmur, for several successive days, while effusion into the left pleural cavity was at its height.

It is of some importance to know what amount of liquid is required in order to produce a perceptible cardiac displacement. Fräntzel says that effusion scarcely ever reaches up to the third rib without more or less affecting the position of the apex-beat, and that even smaller amounts of liquid often suffice. He also observes that in children the heart is more easily thrown out of place than in older persons. Another point mentioned by him is that when there have been previous adhesions between the pericardium and the left lung, pleurisy on the left side may cause the heart to be carried backwards away from the chest wall, so that no impulse whatever can be felt or seen.

Even when the effusion is moderate, the normal apex-beat may be absent without there being any discoverable impulse elsewhere, the reason probably being that the sternum conceals it. In all cases of this kind the stethoscope must be used with the object of determining the apex by the loudness of the cardiac sounds.

The diaphragm is pushed downwards whenever the amount of pleuritic effusion is at all considerable. The displacement of the liver or of the spleen may be recognised by percussion, or one or the other may be felt projecting below the ribs.*

In many cases of empyema and in some of serous pleurisy the effusion is circumscribed by adhesions, and hence the physical signs to be next enumerated are modified, the symptoms are less severe, and the treatment more easy.

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* According to Fräntzel, when the distension of the pleura is extreme, it is sometimes possible to detect an elongated, tense, fluctuating swelling, which protrudes below the costal

Physical signs of effusion.—In the clinical recognition of pleuritic effusion we depend on physical examination of the patient. The signs already mentioned must be carefully sought for: enlargement of the affected side, impairment of its movements, and displacement of the thoracic and abdominal organs. There remain the results of percussion and of auscultation; and the former are by far the more valuable, as was long ago maintained by Piorry, in opposition to Laennec.

Dulness on *percussion* is, in fact, the most important sign of pleuritic effusion. The percussion-sound becomes altered long before there is any evidence of pressure upon adjacent viscera. A circumscribed collection of serum and pus may of course cause dulness of any part of the chest, but when fluid lies free in the pleural space, the dulness is to be made out first at the base behind. One must not, however, suppose that small quantities of fluid ought always to be discovered by this means. Wintrich declared that eight or ten ounces could scarcely be detected with certainty, and a considerably larger amount may sometimes escape recognition.

Much depends upon the habitual posture of the patient. If he is sitting upright in bed, the phrenic, rather than the posterior, surface of the lung becomes compressed, and the percussion-sound may at first be scarcely altered. If he is lying down, the fluid is more or less spread out over the back. When the percussion-sound down to the base of the lung is perfectly resonant, one may feel sure that effusion, if present at all, is in such small quantity as to be clinically unimportant.

The dulness caused by fluid in the pleura differs from that produced by consolidation of the lung in being more absolute, and in the greater sense of resistance which it conveys to the finger in percussion.

If the effusion is large, the whole of the back and side of the chest are devoid of resonance. It is remarkable in such cases that over the front of the chest—below the clavicle, and downwards nearly to the nipple—the percussion note is "subtympanitic," *i. e.* though it retains its musical quality it is shorter and less sonorous than usual (cf. pp. 1026-7). Skoda first pointed out this fact, which remains associated with his name. Its explanation is still doubtful.*

Auscultation is of less assistance than percussion in the detection of pleuritic effusion. As a rule the breath-sounds on the affected side of the chest are feeble, indistinct, or altogether wanting, so that no impression is made on the ear when the patient makes the greatest efforts in breathing. But, occasionally, tubular breathing is audible over part of the compressed lung, and in some exceptional cases—usually in children—this can be heard over the whole of the affected side.

The same curious uncertainty applies to *auscultation of the voice*. As a rule, it is conveyed to the ear more feebly than on the healthy side of the chest, and often vocal resonance is completely absent. But occasionally, still more rarely, there is bronchophony.

There is, however, a modification of vocal resonance which is very characteristic of pleural effusion. It was discovered by Laennec, who called it *ægophony*, from its resemblance to the bleating of a goat. He also compared it to the artificial voice of "Punch" in the street (p. 1038).

* Some writers are content to ascribe it to "relaxation of the pulmonary tissue." Walshe taught that it depends upon the presence of air in the minute tubes of the carnified lung, so that a condition essential to its production is that these tubes should not have undergone compression as well as the lung-substance. The explanation suggested by Bristowe was the diminution of the vibrating area formed by the thoracic walls (p. 1026).

Both these comparisons are excellent, and the sign is readily caught even by an untrained ear. It may be described as high-pitched, tremulous vocal resonance, with a nasal twang. With regard to the frequency of this sign, widely opposed statements have been made in consequence of different definitions of the term. Perfect ægophony is rare, and seldom lasts more than a few days. But if one is to speak of the voice as ægophonic whenever it reaches the ear with more or less of a twang, there are few instances of pleuritic effusion in which this change in its character is altogether absent. In one particular region ægophony is observed far more frequently than elsewhere, namely, about the inferior angle of the scapula, and round towards the axilla. But sometimes it is heard in front, near the nipple, or even close to the clavicle.

Its production is usually believed to depend upon the presence of a rather thin layer of liquid between the lung and the parietes. It may disappear as the effusion increases; but in some cases, not explained by the existence of adhesions, ægophony persists in spite of abundant accumulation of fluid; and, according to experienced auscultators, it may occasionally be present when there is no effusion at all. Dr Frederick Taylor believes that ægophony always depends on compression of the bronchial tubes (cf. *supra*, p. 1038, and the diagrams in his paper in the 'Med.-Chir. Trans.,' vol. lxxviii, pp. 147, 149).

With pleural effusion the *tactile fremitus* felt when the patient speaks is diminished or absent, a sign which is of the greatest value in distinguishing this condition from solidification of the lung.

General symptoms.—The symptoms of pleurisy vary widely in severity. They are sometimes so marked that their import cannot be overlooked; and they are sometimes almost, if not quite, absent.

Foremost among them is *pain* in the side, the *point de côté* of French writers. This is often violent, and of a sharp tearing, cutting, or stabbing character. It is increased both by movement and by pressure, but much more by a deep breath, by a laugh, a cough, or a hiccup. The patient, therefore, breathes in a shallow, jerky manner. His cough, if he coughs at all, is short and half suppressed, and he abstains as much as possible from laughing or sneezing. He lies during the early part of his illness on his back or on the unaffected side, and he shrinks from percussion.

The severe pain contrasts with its absence in bronchitis. It is comparable to that of the inflamed peritoneum, and, like it, is excited or greatly increased by pressure. Hence the pain of breathing and the relief obtained by restraining the movements of the chest by bandages. Its seat is often limited to the lower part of the chest—about the nipple or between the fifth and eighth ribs—because, as Cruveilhier observed, there is much more movement of the visceral upon the costal pleura here than higher up. But the pain is sometimes felt in the shoulder, in the armpit, or beneath the clavicle. In some cases it is referred to the terminal distribution of the lower intercostal nerves; to the hypochondrium, so as to lead to a mistaken diagnosis of hepatitis; to the loins, so that the case has been called one of lumbago; or to the neighbourhood of the umbilicus, so that peritonitis has been suspected.*

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* I once had a patient whose sole complaint was of pain in the crista ilii; I feel sure that if it had not happened that a short while before my attention had been specially directed to this question, I should have failed to discover that he had pleurisy, although on applying my stethoscope I at once heard a rub.—C. H. F.

In some cases pain seems to be absent, or only slight, and thus a large quantity of effusion may accumulate in the pleural cavity without its presence being thought of. This is most apt to occur in children, in aged people, and in lunatics. It often happens that pain subsides or disappears towards the end of the first week, or even after two or three days.

Next to pain, *dyspnœa* is the most striking symptom of pleurisy. The breathing is short and jerking, but it is also increased in frequency, especially when the patient exerts himself, as in lifting anything or in going upstairs. Sometimes the scaleni and the other muscles of forced inspiration are brought into action; and the nostrils dilate each time air is drawn into the chest. As effusion accumulates, the patient begins to find that he can lie over towards the affected side more comfortably than in any other position, because the weight of the fluid is then removed from the mediastinum and the sound lung can move more freely. Orthopnœa is often present, for the diaphragm works more freely with an upright posture; and sometimes lividity of the cheeks and lips may be noticed.

The dyspnœa of pleurisy, like its pain, is generally more marked in robust plethoric patients than in those who are anæmic and wasted. As Andral long ago observed, pleurisy may scarcely interfere with the patient's comfort. He had a patient who went on with his work as a carter, in spite of an enormous effusion into his pleura; and Watson speaks of a butcher, who in the same condition was convinced that he was well, and fit to leave the hospital.

Cough is seldom absent in pleurisy; and there is no doubt that it may occur independently of pneumonia or bronchitis. Sometimes it can be excited by percussion or pressure over the painful intercostal spaces, or by changes of posture; and during the operation of paracentesis it may be produced by movements of the trocar. The cough of uncomplicated pleurisy is short and dry, *i. e.* unattended with expectoration.

Pyrexia is generally moderate in pleurisy. Slight chills returning day after day are observed in many cases, especially when the patient remains out of bed during the day; but an initial rigor is very rare. The temperature commonly ranges at about 101° or 102°, but in the most severe forms of the disease it may reach 104°, or even higher in children. In persons suffering from cancer, or from chronic renal disease, there may be no pyrexia at all.

The *pulse* is accelerated, and as effusion goes on, its volume and tension diminish, in consequence of the obstruction in the lungs. This affords a valuable indication of the degree of danger to the patient's life: and during paracentesis the physician may feel the pulse becoming fuller and slower under his finger, showing the immediate relief given by the removal of the fluid.

Side affected.—It has been thought, and perhaps with justice, that pleuritic effusion, or at least empyema, is more common on the left side than the right.* Dr Eddison, of Leeds, among 40 purulent cases found 20 right to 20 left; Dr Richards, of Birmingham, 3 right to 7 left: Dr Griffith, of Leeds, 19 right to 26 left; Mr Godlee, 22 right to 28 left. Of 44 cases of empyema under the writer's care, 16 were right and 28 left. Adding these numbers together, we find that of 189 cases of empyema, 80 were right and

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* "That pleurisies are *only* on the left side is a popular tenet not less absurd than dangerous" (Sir Thos. Browne, 'Pseudodoxia epidemica,' bk. iii, chap. 3).

109 left. Double serous or plastic pleurisy is not uncommon, and cases of double empyema have been recorded in children.

Course and event.—The course of plastic or serous pleurisy varies widely in different cases. Although attended with no alarming symptoms, a large amount of effusion is never free from danger; for death sometimes occurs quite suddenly and unexpectedly.

In 1874 this happened to a patient in Guy's Hospital with double pleurisy, who a short time before had been seen quietly asleep. For some days previously this patient had suffered from dyspnœa, and it seems not unlikely that the immediate cause of death was exhaustion of the respiratory centre. But in another case, which ended fatally after an hour's extreme distress of breathing on the evening after admission, it was observed that the pulse ceased before the respiration. Here the effusion was on the right side; but there was œdema of the left lung, which no doubt helped to kill the patient.—C. H. F.

For the occurrence of fatal syncope, when the left pleura is the one affected, a special explanation was suggested by Bartels, of Kiel, in the 'Deutsches Archiv' for 1868. It depends upon the anatomical fact (which has been verified after death on several occasions by him and by Fräntzel) that when the heart is pushed far over to the right the mouth of the inferior vena cava becomes bent almost at a right angle, just above the quadrilateral aperture in the diaphragm, the wall of the auricle forming a fold which covers a large part of the aperture. This interference with the flow of blood to the heart may be fatal if the diaphragm suddenly ascends in a fit of coughing, or if a sudden muscular effort is made.*

But pleurisy is not often attended with such risks. The inflammation need not go beyond the exudation of lymph, and after a time it may subside, leaving adhesions which fix the lung to the chest wall for the rest of life. Whether dry pleurisy always ends thus in adhesion of the affected part, or whether it may not sometimes pass off without permanent traces, is a difficult question. What is well known to every pathologist is the fact that an adherent pleura is often found when there has been no history of any affection of the chest. Dr Gee gives a tracing, made with the cyrtometer, which shows that in a child the chest may be markedly contracted on one side, as the result of a universal closure of the pleural space, without there having been any symptoms to suggest the presence of such a morbid condition. There is no clear evidence that partial adhesions left by a dry pleurisy at all affect the health.

The *duration* of an attack of dry pleurisy is sometimes exceedingly brief, often not more than two or three days.

I was once asked to visit a student who had been seized the same day with violent pain in the side, and who told me that he knew he had pleurisy, because he had had the disease before. I heard a very well-marked rub on auscultation, and told him that I should come to see him on the following day. But when I came he assured me that he was well again; and on listening I could detect nothing abnormal.—C. H. F.

When primary pleurisy gives rise to effusion and the amount of liquid remains small, we may feel sure that absorption will take place in time, and that the compressed pulmonary tissue will expand and resume its functions. And even if the quantity should be large, we may always hope, if it be serous, that the patient's ultimate recovery will be complete.

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* Trousseau had previously attributed the occurrence of sudden death in cases of pleuritic effusion to "twisting of the aorta and large vessels," as a result of displacement of the heart; but he does not seem to have had the inferior vena cava in view.

Percussion usually gives the earliest indication of the subsidence pleural effusion. The dulness becomes less extensive and less absolute. only in front, but also over the upper part of the lung behind. A lit later the displaced organs return to their proper places, and the side fa back to its previous dimensions. A feeble vesicular murmur may then heard where none had been discoverable before. But with regard to th there is a source of fallacy which must be borne in mind. At a cert stage of pleurisy, even while the affected lung still remains complet flattened and airless (as subsequently appears from an autopsy), the spiratory sound from the opposite lung is very apt to be carried across t spine as if air were entering both sides of the chest. It seems that t pleura when overfilled may transmit sounds almost as well as a solid lun and this agrees with the occasional presence of bronchophony and vo fremitus in pleuritic effusion (cf. p. 1090).

Another physical sign which often attends the absorption period pleural effusion is what is termed a “redux rub.” This exactly resembl the friction-sound of the earliest stage of the disease: it often remai audible for several days or even weeks together, and may be accompani by a return of pain in the side, without fresh inflammation.

Even when the attack has so far subsided that the patient feels we and perhaps resumes his occupation, it often happens that the side st remains more or less dull on percussion, and the breath-sounds ov the affected part are much feebler than natural.

Empyema.—One event of pleurisy is empyema, either as a sequel of serous effusion, or as a purulent exudation from the first. The determini cause of this untoward event is the presence of pyogenic organisms, strept cocci, staphylococci, pneumococci, or tubercle bacilli. It is more comm in children than in adults. It is rare in idiopathic or traumatic pleuris rare after rheumatism or Bright's disease, common after pneumonia, rare the course of phthisis, and constant when due to pyæmia.

Circumscribed empyema is often met with, and in adults is probab as frequent as that unlimited by adhesions. It most often occupies the ba and back part of the pleural space; sometimes the axilla, and occasional the apex. In a well-marked case of right apical empyema in a patient Dr Douglas, of Newbury (1892), which occurred after acute pneumonia, simulated tuberculous disease so closely as to deceive the writer.

Double empyema can be only partial, and is a rare condition, and from a desperate one, for both sides of the chest may be evacuated. It of course, circumscribed by adhesions.

Course of empyema.—If the pleuritic effusion is purulent, it probal never undergoes complete absorption: if unrelieved by operation, perfora the pleura, and thus makes its way out of the body. This never happe with a serous effusion.

Sometimes the empyema escapes *through the bronchial tubes.* If tl should occur while the patient is asleep, or if he should be so exceeding feeble as not to be able to expectorate, he may be instantly suffocat But surprisingly large quantities of pus are sometimes ejected, with mu less distress than might have been anticipated. If the opening lea directly into a large tube, air passes into the pleural cavity to take t place of the liquid, and a “pyopneumothorax” is established, which w be discussed further on (p. 1113). But in many cases of empyema d charging through the lung no pneumothorax follows. This appeared remarkable to some of the older physicians, that they supposed that pus was capable of undergoing absorption from the pleura, and of being afterwards excreted from the bronchial mucous membrane. The true explanation was given by Traube in 1872 ('Ges. Abhandlungen,' vol. iii, p. 44). If the pleura is ulcerated through, the alveolar texture of the compressed lung may allow pus to be forced through it by violent coughing, while it affords no passage to air in the opposite direction. There is little or no movement of that side of the chest during inspiration, and if there were the air would not be able to penetrate the pus which fills the air-passages adjacent to the orifice.

Such cases often end in recovery, as Hippocrates knew, when he wrote: "Those in whom a pleurisy ends in suppuration, may be cured if they bring up the matter within forty days from the rupture into the pleura" ('Aph.,' v, 15). The treatment is considered below (p. 1105).

In other cases, but less often, an empyema makes its way outwards *through the parietal pleura.* An intercostal space is perforated; and a soft elastic swelling, of greater or less size, forms beneath the skin, which ultimately becomes reddened, ulcerates through, and allows an enormous quantity of pus to escape. At the present day one seldom has an opportunity of observing this result of pleurisy, because scarcely any practitioner now fails to diagnose a large empyema, and to treat it. The point at which perforation is most apt to occur was thought by the late Mr Marshall to be in the fifth space, below the nipple, where there is a weak spot in the chest wall, covered only by the internal intercostal and external oblique muscles. But the orifice may be elsewhere, and used to be most often seen as high as the second space. Occasionally the diaphragm may be perforated. In 1865 a man died in Guy's Hospital of an empyema, which had been tapped eleven days previously. A hole a quarter of an inch in diameter was found in the fleshy substance of the diaphragm, and below it was a large circumscribed abscess, which had not yet opened into the peritoneum.

Again, the pus may make its way backwards and point *in the loin.* A boy nine years old was once in Guy's Hospital for pleurisy, and was discharged convalescent. Afterwards he came back with a pulsating swelling in the left lumbar region, which proved to be an abscess and was opened. Two months later the boy died of tubercular meningitis, and an autopsy showed that the left lung was still contracted, and that a sinuous channel, six inches long, extended down from the pleural space behind the diaphragm to the external opening. In one recorded case the pus from an empyema burrowed until it actually reached the popliteal space.

Except when the pus escapes through the pulmonary tissue, the spontaneous discharge of an empyema is almost always followed by a protracted illness, and may end in the death of the patient. Entrance of air into the serous space renders the contained fluids putrid; and this leads to irritative fever, and to more or less rapid emaciation and exhaustion. If not, the pleural fistula may go on discharging for five, ten, fifteen years, or even longer, until lardaceous changes develop themselves in the abdominal viscera, and cause death by renal dropsy. In one patient, however, under the writer's care, an old lady of about seventy-two, empyema had lasted fourteen years, and shortly before her death there was no albuminuria nor sign of enlargement of the liver or spleen.

The only chance of recovery in cases of this kind seems to be for the whole cavity to be gradually obliterated by granulation tissue, and by the contraction of the dense fibrous material which develops from it, and sometimes reaches the thickness of an inch. At the same time all the structures which surround the pleural cavity become dragged inwards, so as to reduce it within the smallest limits. The ribs fall in and may almost touch each other; they remain motionless during inspiration, or (as was once observed by Dr Gee) their anterior parts may actually recede and move backwards each time that the healthy half of the chest expands and draws the sternum forwards. The dorsal spine becomes curved, so as to present a concavity towards the affected side. The shoulder sinks, the diaphragm is dragged upwards with the abdominal viscera; the mediastinal structures are pulled over, and the heart is displaced and brought widely into contact with the ribs.

Diagnosis. As a rule, the recognition of pleurisy and pleuritic effusion is easy, being based directly upon the characteristic physical signs. It is doubtful how to regard cases in which there is no other symptom than a *pain in the side.* Hospital practice teaches us to distrust the statements of patients when they tell us that they have been under treatment for pleurisy. Pleurodynia may be neuralgia or intercotsal "rheumatism," *i.e.* myalgia; or an eruption of shingles which has escaped notice, from not having been looked for. Costal periostitis and abscess of the chest wall are other affections, the possible presence of which must not be forgotten. The only case in which it is allowable to diagnose pleurisy without the evidence of physical signs is when violent pain in the lower part of the chest is increased by each breath and accompanied by pyrexia. We may then reasonably suppose that there is inflammation of the phrenic pleura.

Even when we think we hear a *pleuritic rub,* there is need of caution. Gairdner has recorded an instance in which a shuffling sound with a tactile sensation, as of something rubbing up and down against the walls of the chest, proved to be due to emphysema of the lung. And Guttmann cites a case of Jurgensen's, in which a similar effect was produced by tubercles of the pulmonary pleura. It is said that the crepitus of a broken rib has been mistaken for a pleuritic rub.

Apart from these rare events, what has been called a soft pleuritic rub frequently turns out to be a mucous râle. Another mistake is that of attributing to pericarditis a friction-sound which is really pleuritic.

Pleuritic effusion has sometimes been diagnosed when the disease (on the right side) has been a *hydatid* in the liver, or a hepatic *abscess,* or hypophrenic abscess, or when (on the left side) it has been an abscess between the liver and the diaphragm connected with the spleen. Again, it is remarkable that most examples of very large chronic *pericardial effusion* which have occurred at Guy's Hospital have been set down to pleurisy. This error was made by the writer in 1889, there being moderate effusion in the left pleura combined with the presence of forty-two ounces of serum in the pericardium in a case of Bright's disease: the accumulation was very gradual, and the symptoms remarkably slight. One ought to be on one's guard, carefully mapping out the area of percussion-dulness and marking exactly how far it extends in front, at the side, and behind: the figure produced would probably decide the point. No doubt the disease might still be a circumscribed empyema: but paracentesis would probably do no harm.

When there is a very large effusion of pus into the left side of the chest, pulsation synchronous with the heart can sometimes be felt in the intercostal spaces near the nipple or above it and towards the clavicle, so that the presence of an *aneurysm* may be suspected. A case of this "pulsating empyema" was recorded two centuries and a half ago by Baillon; its real nature was cleared up by the bursting of the swelling with discharge of pus. In our own time Walshe and Macdonnell, of Montreal ('Dublin Journ. Med. Sci.,' 1844), have studied "pulsating empyema;" Comby in France ('Arch. Gén.,' 1883), Kepler in Germany ('Arch. f. kl. Med.,' 1887), and Osler in America ('Trans. Assoc. Amer. Physicians,' 1888, p. 330) have collected about forty cases. Its pathology is not clear, but seems to depend on extreme tension, with weakening of the intercostal muscles. Pulsating empyema occurs almost always on the left side.

The physical signs may point to the presence of a large pleuritic effusion occupying the lower and back part of the chest on one side, and yet they may turn out to be due to *solid lung*. Traube has related a case in which he imagined that there was, besides hepatisation of the left lung, a large pleuritic effusion, but the autopsy showed that the serous cavity had been closed by former adhesions; the great diminution of tactile vibration in this instance was attributed to plugging of the smaller bronchial tubes by lymph. Most English physicians have followed Laennec in believing that the detection of ægophony is conclusive evidence that there is at least some fluid effusion into the pleura; but Fräntzel was convinced that this rule was not invariable, as had, indeed, long ago been asserted by Skoda; and more recently Osler and Frederick Taylor have recorded ægophony in pneumonia without effusion.

A frequent mistake is to take for pleuritic effusion considerable *thickening of the pleura* with chronic adhesions. Here there is dulness on percussion, the breath-sounds are often weak or inaudible, and vocal resonance and fremitus are more or less completely abolished. In such cases, if seen for the first time, we must often be doubtful, and may legitimately have recourse to the hypodermic syringe to decide the question.

The only other condition in which this short and ready method of diagnosis is, as a rule, necessary are, in the writer's judgment, those in which we feel doubt after repeated examination whether a child is suffering from pleuritic effusion or from confluent lobular pneumonia. Here bronchial breathing and dulness may be present in both cases and vocal resonance and fremitus often give no help.

A mass of *malignant growth* has before now been mistaken for pleuritic effusion. Every physician of experience must have seen this mistake made by others, if he has not made it himself. The points to be especially noticed are whether the area of dulness corresponds in locality with that caused by effusion, and whether tactile vibration is or is not still to be felt in certain positions. It must be borne in mind that a new growth situated in the mediastinum or in the lung is often accompanied by effusion into the pleura, so that a confident differential diagnosis may after all be less accurate than a more doubtful opinion.

Even when the presence of liquid in the pleural cavity is positively and correctly diagnosed, it is not always inflammatory in origin. Passive dropsy of the serous cavity, or *hydrothorax*, may give rise to similar physical signs, except that, being probably never unilateral, it is not likely to displace the heart. This condition is most common in cardiac disease or in extreme

pleura. In another case, in 1869, pus was found outside the parietal layer of the pleura, infiltrating the intercostal muscles; there was also an abscess as large as a walnut in the mediastinum. In a third case, in 1873, the mediastinal tissues were three quarters of an inch thick from infiltration with puriform lymph. All three were examples of double pleurisy with pericarditis. Some of these cases of inflammation of the mediastinum are accompanied with great hypertrophy of fibrous tissue, and may seriously press on the air-passages or the great vessels.

The secondary pleurisy of phthisis seems seldom to extend to the pericardium. But the more intense and violent forms of pleurisy are exceedingly apt to do so. Moreover, there are cases in which both pleuræ, the pericardium, and the peritoneum become simultaneously inflamed; at least it cannot be determined that one of them was affected first. As a rule such cases present acute symptoms and run a rapid course, but this is not by any means invariable.

In 1876 a woman, aged twenty-three, was admitted into Guy's Hospital with what was supposed to be enteric fever. After a fortnight fluid effusion was detected in the left pleura, and forty-two ounces were drawn off by the aspirator. Her febrile symptoms continued; she became emaciated and died. Towards the last it was naturally thought that she was suffering from tubercular disease. However, on making the autopsy, we could discover no tubercles nor any primary visceral lesion. Beside some pleuritic effusion on the left side there was lymph over the whole of the right pleura; the pericardium was adherent by a recent plastic exudation; the liver and the spleen were fixed to the diaphragm; and the lower part of the abdominal cavity contained some purulent fluid. It may be that the disease was of rheumatic origin, for the patient had a painful affection of her joints about two months before; but there was no evidence of endocarditis, present or past. The case is of interest, not only pathologically, but also on account of its obscurity during life. For the thoracic serous cavities and the upper part of the peritoneal space have been repeatedly found all closed by old adhesions, when there had been no history of any chest affection; and it would now seem that such a result may arise from an illness which clinically might be taken for fever.—C. H. F.

Ætiology.—(*a*) *Of "idiopathic" pleurisy.*—Foremost among the causes of pleurisy is *cold*. This has been so commonly set down as giving rise to all forms of internal inflammation, and often with so little reason, that one cannot be surprised that some able physicians are, like Dr W. H. Ransom, of Nottingham, reluctant to recognise its operation at all. But the clinical evidence of the direct dependence of pleurisy upon cold cannot be explained away. It is particularly strong when cold affecting one side only has caused unilateral pleurisy.

Injuries to the chest may give rise to pleurisy. Not only does this occur when the ribs have been broken, but sometimes when there is no evidence of damage to the walls of the chest.

(*b*) *Of secondary pleurisy.*—Of general acute diseases the most apt to be attended by pleurisy as a complication is acute rheumatism. Rheumatic inflammation of the pleura occurs only during the fever, not independently of it. It is, at least, very difficult to prove its rheumatic origin in other cases, and we have no right to assume it.* Pleurisy may complicate scarlet fever or smallpox, but seldom or never occurs during the early stage of enteric fever, and is not frequent later.

Pleurisy is not infrequently a complication or sequel of influenza when no pneumonia is present. Like the other "febrile" pleurisies, it is usually serous and its course favourable.

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* By "rheumatic pleurisy" many writers only mean pleurisy due to catching cold—a misleading use of the term.

Dry pleurisy occurs in every case of acute pneumonia, and we must remember that what seems to be an uncomplicated and simple attack of pleurisy may really be dependent upon pneumonia, of which there is sometimes little or no clinical evidence; for when pleural effusion has once taken place it may be impossible to discover by physical signs the presence of hepatisation of the corresponding part of the lung. It often happens that the characters of the sputa afford the only clue to the real nature of the case, or a correct diagnosis may depend entirely upon one's having seen the patient before the effusion occurred. Again, it is not improbable that a very limited patch of pneumonia, involving the surface of the lung, may sometimes be the starting-point of a diffused and severe pleurisy.

Moreover, pleurisy may be set up by the pneumococcus without any affection of the lung, and the result will be a combination of the signs of pleurisy with the symptoms of pneumonia (see a paper by Dr Washbourn in the 'Med.-Chir. Trans.' for 1894).

Among chronic affections none is so commonly accompanied by pleurisy as Bright's disease; so that the urine should always be carefully and repeatedly examined, not only for albumen, but for the colour, abundance, and specific gravity.

Lastly, there are certain cases in which chronic or subacute pleurisy is associated with pericarditis and peritonitis without tubercle or Bright's disease being present, as if the whole of the parts of the primitive cœlom were affected together (cf. *supra*, p. 1084).

Secondary purulent pleurisy is often due to the direct extension of inflammation from some adjacent structure; familiar as this is in the deadhouse, it is, as a rule, quite recent, so that it has little clinical significance. Among the less obvious starting-points for a severe or even fatal pleurisy may be mentioned abscesses in the armpit, operations upon the breast, suppuration of the cervical connective tissue after tracheotomy, caries of the ribs, mediastinal abscess, cancer of the œsophagus, and caries of the dorsal vertebræ. Sometimes its origin may be below the diaphragm, the connecting lymph-channels discovered by von Recklinghausen conveying the inflammatory process from one serous cavity to another. Thus pleurisy, which was the immediate cause of death, has been found to be due to extension from a peritonitis of puerperal origin, or following ovariotomy, or arising from disease of the colon. Again, a very acute inflammation of the pleura, with fœtor of the pus, has started from the upper end of a psoas abscess, or from a localised abscess behind the stomach due to a perforating gastric ulcer. Mediastinal growths without inflammation frequently produce pleuritic effusion; so also do malignant tumours of the walls of the chest or of the mamma.

In pyæmia (especially when resulting from thrombosis of the cerebral sinuses, itself consequent upon disease of the temporal bone) pleurisy is sometimes the most conspicuous feature of the case, and may be mistaken for the primary disease.

The relation of pleurisy to phthisis is of the greatest importance, on account of its bearing on prognosis. One frequently sees patients who, having passed favourably through an attack of pleurisy, sooner or later afterwards develop signs of tuberculous disease of the lungs. In many cases the interpretation of the facts is that there was already disease of the lungs, of which the pleurisy was the first sign, as we shall in a future chapter see is the true relation of phthisis following hæmoptysis. Tuberculous pleurisy (which is almost always serous, not purulent) may be due to a few pulmonary tubercles on the surface of the lung setting up irritation and effusion. But in many cases all the clinical evidence points to the conclusion that the pulmonary disease has been of later development; and we can scarcely doubt that in some of these cases the tubercle bacillus found its point of least resistance, not in the lungs or in the lymph-glands, the spleen or the peritoneum, but in the pleura: that in fact the pleura is the first scene of the process which afterwards goes on in the lungs. Lately some physicians have perhaps exaggerated the frequency of this relation. In France particularly a remarkable number of cases have been observed among young recruits of apparently idiopathic and benign pleurisy, which ran a favourable course, but was subsequently to an apparently good convalescence followed by symptoms of phthisis. These cases we may still believe are the exception, not the rule; for in civil practice in this country it is certain that many cases of pleurisy in young patients get perfectly well and are never tuberculous at all. But we must admit that the proportion in which pleurisy is the first symptom of tuberculosis is much larger than was suspected twenty years ago; and we look upon a history of pleuritic effusion with suspicion in the case of any one under thirty who is proposing to insure his life.*

It is a curious question whether pleuritic effusion, while it continues to compress one of the lungs, favours a fresh development and further growth of tubercles in that organ, or whether it may not rather be adverse to such an occurrence, even though it may increase the susceptibility of the other lung. Dr Fowler has found tubercles in the pleura between the lobes of the lung in some cases of what was apparently idiopathic pleurisy.

The following six cases bear on this question. In one there were no tubercles except in the opposite lung; in another the tubercles were much less numerous on the side of the pleurisy; in two others it was observed that on that side they were all of old date and inactive. On the other hand there was one case in which they were more abundant in the compressed lung than in the opposite one; and once a lung entirely airless was full of tubercles (some grey and other caseous) in its lower lobe, where their presence, the apex being healthy, is an occurrence so exceptional that one could hardly doubt that the pleurisy had determined their formation.—C. H. F.

The cases of pleurisy due to the microbes of pneumonia, of suppuration, and of tubercle, and secondary pleurisy generally, are now ascertained to be more frequent, and primary or "idiopathic" pleurisy from cold or other unknown cause to be less frequent, than was formerly believed.

On the whole, it is important to remember that (excluding traumatic cases and direct exposure to cold) the majority of cases of pleuritic effusion in young adults, if not occurring in the course of rheumatic fever, are tuberculous, and that the majority of cases in elderly persons are renal or secondary to malignant disease.

Prognosis.—This depends chiefly upon the origin of the pleurisy, secondly on its extent, and thirdly on the serous or purulent character of the effusion.

Acute idiopathic pleurisy from exposure to cold in a healthy subject is scarcely ever fatal. With judicious treatment it is speedily relieved, and cured without leaving sequelæ behind. Even when one side of the chest has been filled with serum, it is remarkable how well such cases do after aspiration.

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* See on this subject a paper by Dr Barrs, of Leeds ('Brit. Med. Journ.,' 1890, i, 1858); and the writings of Landouzy, Germain Sée, and other French physicians.

An effusion which half fills the pleura, without pain or fever, and is only discovered by physical examination in search of a cause for the patient's dyspnœa,—this should make one suspect tubercle or Bright's disease. But when these can both be negatived, the prognosis still is good.

The pleurisy which forms part of acute pneumonia adds to the pain, but not to the danger of the primary disease; and the same is true when pleurisy is secondary to lymphoma, or other mediastinal growths. In the latter case it is usually latent, and often as much dropsical as inflammatory.

Tuberculous pleurisy does not usually add to the dangers of phthisis, and sometimes effusion with carnification appears to retard its progress by limiting the amount of blood as well as of air in the diseased lung, for tuberculous inflammation does not flourish in anæmic and airless pulmonary tissues. On the other hand, pleurisy secondary to Bright's disease, whether dry or combined with hydrothorax, is a very serious complication, only less so than the pericarditis which often supervenes under the same circumstances.

To leave a patient with one side of the chest full of fluid is never safe. He may die suddenly from syncope, or œdema of the other lung may suffocate him before help can be brought.

When pleuritic effusion is purulent, the prognosis is, so far, graver. Formerly the event was often fatal, by pyæmia, by hectic fever, by secondary phthisis, or by lardaceous disease; and in the best cases much deformity was the usual result.

But the bolder treatment on which modern surgery safely ventures has wonderfully improved the prognosis of empyema. In the case of children, making a single free opening, and inserting a drainage-tube is often followed by re-expansion of the lung and complete recovery, without the least sign of the disease remaining except the scar of the operation. With adults the result is not so often perfect, but here also complete cures are not infrequent, and complete failure is the exception.

Much attention has been lately directed, especially in France, to the bearing of microscopical examination on the prognosis of pleuritic effusion. When secondary to pneumonia, and when the pneumococcus alone is present, it is believed that after emptying the chest recovery ensues without further interference. This is not always true, for in two cases under the writer's care in which the fluid was a pure cultivation of Fränkel's diplococcus, it returned after paracentesis, and we had to cure it by means of drainage. Most cases of pleuritic effusion secondary to pneumonia are purulent, and when streptococci are found, incision should be performed at once with good hope of recovery. In the "broncho-pneumonia" of children, if pleuritic effusion occurs, it is usually purulent, and the prognosis very good. These are the only cases in which the writer has seen recovery after aspiration without incision.

When the bacillus of tubercle is present, the effusion is usually serous, and if not so large as to threaten the patient's life it is probably safest to leave it alone. If, along with the bacillus, staphylococci or streptococci are found, incision and drainage is indicated.

In the rare cases of septic pleurisy secondary to gangrene of the lung or some other necrotic process, various saprophytes may be found in the pus or sero-pus. Such cases are of very bad prognosis.

Treatment.—A patient suffering from pleurisy should be kept strictly in bed during the early stage. For the relief of pain, the application of a blister is perhaps more generally serviceable than anything else, and it may be used with advantage at any period. But many physicians hold that for prompt and grateful relief of the acute pain, as well as of the dyspnœa, no remedy is so efficacious as the application of half a dozen leeches to the side. Among milder measures, such as poulticing and the application of belladonna, none are so effectual as careful strapping of the affected side, so as to restrain its movement in respiration.

Some patients find ice-bags a more pleasant application than hot fomentations or poultices, but in the writer's experience children dislike it, and most adults find more relief from hot-water bottles.

Of drugs, the most useful when effusion has taken place are probably purgatives and diuretics (such as digitalis, with acetate, iodide, or citrate of potass). In cases of chronic serous effusion, rubbing the affected side with mercurial liniment has often proved of great value. Friction which had been heard day after day for weeks has ceased almost at once, or fluid effusion has cleared away after a somewhat longer period. The diet should be light and spare. The plan of allowing the patient very little to drink was often practised by Sir William Gull and by Dr Moxon, and was advocated by Niemeyer. Rapid absorption of fluids from the chest has been noticed when the patient has been attacked by cholera; and from an attack of summer diarrhœa. As a rule, it is better to act on the kidneys than on the skin or the bowels.

Paracentesis thoracis.—Whenever the physical signs indicate that there is considerable liquid effusion into the pleural cavity, the question of removing it by tapping arises. This procedure, it is interesting to know, dates back to Hippocrates. But although Trousseau advocated it as far back as 1843, and Hughes as strongly in 1844,* few physicians, until fifty years later, understood its importance or its safety.

There is still difference of opinion as to how soon the operation should be undertaken. Trousseau in Paris, and the late Dr J. W. Begbie in Edinburgh, used to tap early and frequently; but if as soon as the signs of fluid in the pleura were recognised, paracentesis were performed, "dry tapping" would be much more common and the results less satisfactory.

If percussion shows that the quantity of fluid is moderate, it is as a rule advisable to wait. The presence of pyrexia is, at an early stage of pleurisy, another reason for delay. The patient must be watched very closely, to see whether the effusion increases or diminishes as time goes on. Unless it begins to diminish in the course of a fortnight, no further postponement of tapping is desirable. Even though the quantity of liquid should remain unaltered, the probability that the lung will quickly expand and regain its functions after paracentesis will become less and less, in proportion to the length of time during which it has been compressed; for the layers of lymph that bind it down gradually become fibrous and contract. Moreover, the withdrawal of a part of the fluid by operation seems often to facilitate the absorption of the rest. It is supposed that the subpleural lymph-channels are mechanically pressed upon by the liquid, and thus the flow through them is interfered with.

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* A paper by the late Dr Hughes and Mr Cock recording twenty cases of pleuritic effusion treated by paracentesis appeared in the 'Guy's Reports' for 1844.

We may safely delay paracentesis with a patient in a hospital ward, watched day and night, and with immediate assistance at hand, when it would be imprudent to leave fluid in the chest of one living at a distance from medical aid. In cases of Bright's disease when one lung is half carnified by fluid, paracentesis should be performed at once, or sudden œdema of the other may prove fatal before help can come.

In any case, if percussion shows that one side of the chest is full of fluid, the operation cannot safely be delayed even until the following day: and this in spite of the absence of obvious distress of breathing, or the presence of pyrexia.

Whenever we have reason to believe empyema to be present by the signs and probabilities mentioned above (p. 1098), we should aspirate at once. If there be pus in the pleural cavity, its removal is often followed by the cessation of pyrexia.

Of this I saw a most striking instance in 1881 in a man who had pleurisy as a sequel of enteric fever. His temperature rose every afternoon to between 102° and 103°, falling in the night and morning. I had made one unsuccessful attempt to draw off the effused fluid, the reason of my failure being that I used a very fine aspirator needle, because the area of percussion dulness was not in the usual position behind, but at the side of the chest, just outside the situation of the pericardium, so that I felt some hesitation in acting upon my diagnosis. However, six days later, when I visited him, I found him in a most critical condition, with great anxiety of face and with extreme distress of breathing. As the only chance of saving his life, I had a somewhat larger trocar plunged into the chest at exactly the same spot as before. Aspiration was then performed, but at first no fluid appeared. However, I took the instrument and pushed it inwards, feeling, as I did this, that the end of it encountered and seemed to pass through a resisting membrane. A quantity of rather viscid blood-stained liquid at once escaped, and of this four and a half ounces were withdrawn. The patient was instantly relieved, and from that time went on to recovery without a bad symptom. His temperature, which was rising at the time of the operation, and had reached 100·8°, fell at once, and three hours later it was normal.—C. H. F.

When it is decided to tap the chest, it is a common practice to make a preliminary puncture with a hypodermic syringe. This ought not to be necessary (except sometimes in the case of children) to determine the presence of fluid: but it is often desirable to use staining and cultivation to determine the nature of the inflammation. When no result follows the puncture and no frothy blood fills the syringe, it is well to fill it with boiled water, and inject a few drops first, so as to increase the pressure at that point before drawing up the piston.

Paracentesis of the chest has been much facilitated by the introduction of Dieulafoy's aspirator.*

Even with the help of the aspirator, there is sometimes difficulty in getting out the liquid. Fragments of fibrin are drawn against the inner orifice of the tube, or its channel may be occluded by viscid pus, and sometimes one can restore the flow by passing a sterilised probe through the cannula. If this fails, it may be necessary to make a second puncture at a different spot.

In tapping the chest in children it is often advisable to give chloroform, not so much for the sake of the pain (which is, however, not so trifling as those think who have not felt it) as for the possibility of having to puncture repeatedly before obtaining the fluid; and this is impossible with a child screaming and struggling on his mother's lap.

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* This procedure was, it appears, devised and carried out by the late Dr H. J. Bowditch, of Boston, some years before Dieulafoy introduced it to the profession in Europe.

The trocar and cannula, the patient's skin, and the operator's hands should all be rendered aseptic before the operation is performed.

Mr Godlee fixes the best spot for puncturing the chest at the fifth or sixth space in the mid-axillary line; but many prefer the seventh space just outside the angle of the scapula. Bowditch recommended that the instrument should be introduced between the ninth and the eleventh ribs. Whichever space is chosen, we should keep close to the upper edge of a rib in order not to wound the intercostal artery. In 1855 this accident happened during an operation performed at Guy's Hospital; the patient (who had phthisis) became faint at the time, and died the same evening; a pound of clotted blood was found in the base of the chest.

It is not desirable, in performing paracentesis, to attempt to empty the pleural cavity. Not more than from one to three pints should be withdrawn at once, and the operation should be stopped when the patient begins to cough* or when the serum is blood-stained.

Occasionally after thoracocentesis, the patient expectorates large quantities of frothy liquid containing much albumen. Within an hour after the operation his breathing becomes distressed, he begins to cough, and he may die cyanosed in a quarter of an hour. Twenty-one instances of this complication were collected by Terrillon in a monograph published in 1873; most of them, however, ended in recovery, and some were comparatively slight. It had been supposed that the expectorated serum came from the pleura, either from the lung being accidentally wounded or from a previous perforation, which opened out as the pressure was removed. But, as Terrillon had no difficulty in showing, both explanations are quite untenable, and the only reasonable hypothesis is that of pulmonary œdema (or, rather, serous effusion into the air-vesicles and bronchioles) following sudden hyperæmia of the lung when released from the pressure of the fluid.

An objection against tapping the chest except in cases of necessity, one which had the support of Stokes of Dublin, was that it may lead to the conversion of a serous effusion into pus. This objection was not without justice in the days before the danger of sepsis was guarded against.

In certain cases the fluid quickly accumulates again after paracentesis, so that the patient's condition becomes as bad as before, and the operation has to be repeated. When this happens two or three times in succession, perhaps at intervals of only a few days, Fräntzel advised to desist from further interference: but this is not the writer's experience. In the case of a girl, a patient of Mr Lacey, of Woolwich, who was tapped fourteen times for hydrothorax in the course of mitral disease, sometimes at intervals of only a very few days, good recovery ensued at last.

Treatment of empyema.—If the fluid obtained by paracentesis is purulent, the further treatment must be modified, for the old plan of repeated tapping led to disastrous results, and we cannot trust to nature even though we must admit that spontaneous recovery of empyema is not impossible.†

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* That cough is not always due to irritation of the surface of the lung by the cannula is shown by its being sometimes produced when the quantity of fluid is still great. Moreover, one may feel the orifice of the instrument rest against the pulmonary pleura without any cough resulting. If one draws off pleuritic effusion very slowly these severe fits of coughing are seldom seen.

† Cheesy masses, and even deposits of calcareous matter, are now and then found after death lying between pleural adhesions; and there can be little doubt that such residues have had their origin in an empyema. In other cases a collection of liquid pus, enclosed in a dense capsule, has been discovered in the pleural cavity, after death from some other cause.—C. H. F.

The present practice is to make a free incision as soon as the pus is seen, and after examination to put in a drainage-tube, so that the pus may escape into an antiseptic dressing as fast as it is secreted—treating the empyema, in fact, as a large abscess. It may be wise, if the patient is a child or if the pus contains only pneumococcus or pneumo- and staphylococcus, to allow the chance of recovery after once or even after twice tapping; but in most cases of empyema the shortest and safest plan is to incise the chest.

In children we have sometimes seen excellent results attained by making an aperture just large enough to admit one end of a long elastic tube, of which the other end is carried beneath the surface of carbolised liquid in a jar placed beneath the bed. The elastic skin of a child grasps the tube firmly, and does not ulcerate. The negative pressure of the column of liquid, acting hydrostatically, seems gradually to raise the compressed lung. Within a short time the flow of pus may cease, and a permanent cure be obtained. With older patients similar success is rare.

A practical difficulty is that the narrow space between the ribs—which is often a serious obstruction in inserting the trocar at first—makes it difficult to put in a large enough tube and thus secure effectual drainage. To meet this it is often necessary to resect one or more ribs, and by preserving the periosteum they will repair well so as sometimes to enclose the cannula in a ring of new bone. The tube must be gradually shortened, the sinus prevented from becoming narrow and tortuous, and the opening kept free. Under favourable conditions it was marvellous to those of us who were accustomed to the ill results of the older plan of treatment, to see how well the chest recovers, and how free from deformity the patient remains.

It is often necessary to irrigate the cavity, after evacuating the pus, with boiled water, so as to wash out masses of fibrin. It was formerly the practice to inject iodine after tapping a serous hydrothorax, and to wash out the pleura with antiseptics in cases of empyema: but phenol poisoning has occurred before now, and only the weakest solutions should be used, if any. The operation of washing out the chest is not altogether free from danger. In 1876 Dr Cayley read before the Clinical Society a case which had occurred to him, and in which, while a solution of iodine was being injected, the patient suddenly became pale, unconscious, and convulsed: the temperature rose to 107°, and death followed in sixteen hours. He cited three cases recorded by French physicians, in each of which like symptoms appeared, though one of them ended favourably. Mr Godlee has also recorded a case in a child.

In 1874, at Guy's Hospital, a girl aged sixteen died in precisely the same way. She had had a drainage-tube inserted into the right chest for an empyema five weeks before, and was going on well. One day she was sitting up, and her chest was being washed out with carbolic acid, when she suddenly ceased to breathe, and, although artificial respiration was set in action, remained unconscious, with muscular twitchings, until death.

Nothing has been found after death to account for these terrible accidents. They seem not to be due to Embolism. It is noteworthy that in every one of the cases the chest had been washed out repeatedly before without any ill effect: the only difference being that in two instances a somewhat larger quantity of fluid was injected than usual.

The results of treatment of empyema by free incision and drainage, with modern methods and antiseptic precautions, are most encouraging. Even in adults one may again and again see complete recovery of the lung with no resulting deformity. In children the practice is still more successful. Thus Dr Goodhart reports that of 50 cases under Dr Frederick Taylor's care or his own 42 completely recovered, a sinus remained in 3, and only 5 died—from suppurative pericarditis, from septic pneumonia, or from broncho-pneumonia.

It has often been debated what is the best treatment when an empyema has already broken into a bronchial tube (p. 1094). It used to be taught that the passage of pus through the lung was injurious and would lead to phthisis or gangrene, so that another outlet ought to be at once provided. But the writer's experience is that these cases often do very well if left alone, and he would advise paracentesis only if there are signs either in the quantity or quality of the expectoration, or in hectic or other symptoms, that due freedom of exit is not provided. In such cases incision, drainage, and washing out the pleura have proved satisfactory means to recovery.

When empyema is (as usual) circumscribed and affects both sides, it is possible, as in a case recently under the writer's care, to incise and drain both sides, and the result may be satisfactory, at least in the case of a child.

In a certain number of cases the cure of an empyema remains incomplete. The cavity may have shrunk to very narrow dimensions, the chest may have regained a fair amount of resonance over a large part of its surface, air may enter the lungs pretty freely, but there is a fistulous opening from which small quantities of pus continually drain away. In such cases the best practice is to excise portions of several ribs, so as to allow the side of the chest to fall in and meet the lung. This operation seems to have been first performed by Peitavy. It was improved by Estlander, of Helsingfors, in 1877, and is often known by his name.

PNEUMOTHORAX.—Morgagni mentions the fact that air sometimes accumulates in the cavity of the pleura; but the word pneumothorax was first used in 1803 by Itard, and Laennec gave the first complete description of the condition.

Ætiology.—The older writers supposed that gases might form in the pleural space as the result of chemical decomposition of a serous or purulent effusion, or by direct secretion from the parietal pleura. But this belief was erroneous; air is never found in the pleural space except when admitted from without.

In the great majority of cases pneumothorax is a consequence of perforation of the visceral layer of the pleura, whereby air escapes from the lung. Often this arises from direct violence: broken ribs are exceedingly apt to wound the lung, and in persons who are run over or severely crushed the lung may be torn even when there is no fracture. Exceptional instances of pneumothorax are recorded in which a lung, previously healthy, is ruptured in a paroxysm of whooping-cough, or during a straining effort. Again, the bullæ of emphysema have often the thinnest possible walls, and one might suppose that pneumothorax would occasionally arise from their rupture. But this, if it ever happens, is extremely rare.

Occasionally the entrance of air into the pleural space is the result of local ulceration or sloughing of the pulmonary pleura, or of acute pneumonia running on to gangrene. More often it follows a slough from an infective embolus in a case of pyæmia. Cases are on record of pneumothorax due to sacculated dilatations of the bronchial tubes opening into the pleura; but they are extremely rare.

The only frequent cause of pneumothorax is the rupture of a superficial vomica in phthisis. Walshe estimated that nine out of ten cases arise in this way, and Fräntzel put it at fourteen out of fifteen. It would occur oftener than it does, but for the adhesive pleurisy which accompanies chronic phthisis and generally seals up the pleural cavity. Even when there is no evidence of phthisis either before or afterwards, it is possible that the spontaneous development of pneumothorax, without any previous violent muscular effort, is due to the rupture of a small vomica, by which neither physical signs nor symptoms have been produced.

The following case was related by Vogel, of Dorpat, in the second volume of the 'Deutsches Archiv.' A woman, aged twenty-nine, became suddenly the subject of pneumothorax one morning at nine o'clock. Some months previously she had a slight loose cough and more recently a little pricking pain in the region of the liver; when the attack began she was engaged in turning over her child's bed, and just before she had been lifting its bath. Vogel himself was inclined to think that she had latent tubercular disease, but in four weeks she recovered completely.

Another way in which pneumothorax may arise is from perforation of the visceral pleura by an empyema discharging itself through the air-passages. In medical practice this cause comes next to phthisis in frequency.

In 1869 a man aged forty-two was brought into Guy's Hospital very ill, and died half an hour after. He was found to have acute pericarditis, mediastinal inflammation, and early pleurisy on the left side. But the principal seat of disease was the right pleural cavity. This contained fœtid gas, and four and a half pints of dirty purulent fluid. In the upper lobe of the lung there were two openings, and through these air had doubtless entered. But the pulmonary pleura was gangrenous over an area of two square inches, and the substance of the lung beneath it to a depth of half an inch. That the pleurisy was of exceptional severity was evident from the fact that there was suppuration outside its parietal layer, among the intercostal muscles.

There still remain cases in which the air is not derived from the lung at all, but either from outside the chest, or from some part of the alimentary canal. As a consequence of perforation of the thoracic walls (apart from the effect of penetrating wounds of the chest), pneumothorax may be set up by physicians when an empyema has broken through spontaneously or has been let out by operation. When the pus points of its own accord, the channel by which it reaches the surface is commonly oblique and narrow, so that air fails to find its way along it. The late Dr Moxon drew attention to the possible occurrence of double pneumothorax as a cause of death after tracheotomy, subcutaneous emphysema extending down from the wound so as to fill the mediastinal connective tissue with air, which then bursts into both pleural cavities. He met with one such case in a woman aged thirty-three, who died in less than twenty-four hours after the operation. Emphysema had spread over the neck, chest, and arms as far as the fingers. Both lungs were found collapsed and almost airless.

In some few cases the starting-point of pneumothorax is the œsophagus; a cancer of this tube may eat its way into the pleural cavity, or ulceration due to a foreign body may have a like result. Sometimes a gastric ulcer, after setting up a circumscribed hypophrenic abscess, has been followed by perforation of the diaphragm; and a hydatid cyst of the liver has been known to open communications in two opposite directions, with the colon below and with the pleural space above.

Of [illegible] cases of pneumothorax collected by Biach from the hospitals of Vienna during thirty-eight years, 715 were due to phthisis, 65 to [illegible] gangrene, [illegible] emphysema, and 32 were traumatic. No other cause accounted for more than [illegible] cases.*

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* Quoted in Eichhorst's Handbuch, Bd. [illegible], S. 582.

Physical conditions of pneumothorax.—Few hospital physicians can have failed to meet with cases difficult to explain, in which communication of the pleura with the outer air, through the parietes of the chest or through the lung, has not been followed by pneumothorax. In most cases pleural adhesions, at or in the near neighbourhood of the perforation, afford a probable solution of the difficulty, and their presence has been confirmed after the patient's death. But certain cases seem incapable of this explanation. In a lecture reported in the 'British Medical Journal' for June 4th, 1887, Dr Goodhart—after referring to the fact that pleuritic effusion may be "held up," so to speak, over the lung instead of gravitating to the bottom of the chest (cf. p. 1088)—proceeded to comment on the rarity of pneumothorax after fracture of the ribs, particularly in young patients, and on the free re-expansion of the lung, even after a large opening has been made into the chest by paracentesis. He thought there must be some forces at work to allow of the lung becoming inflated again by inspiration different from those of physics. Mr Godlee replied in a subsequent number of the same journal from the surgical point of view; and the subject was further dealt with by Dr Samuel West in his interesting Bradshaw Lecture before the College of Physicians in the same year (*ibid.*, August, 1887). He showed experimentally that there is considerable power of cohesion between opposed surfaces of serous membrane, and attributed to this cause the fact that pneumothorax does not occur so readily as one might have supposed.

There can be no doubt in the mind of any physiologist who has seen the unfailing and immediate collapse of the lungs which follows a free incision into the pleura in the case of an animal, that when the pressure on the inside and outside of the lung is equal, it at once shrinks to the bulk which the elasticity of its tissue permits. This is proved experimentally by Donders' "schema," and by a similar arrangement of the dead thorax with manometers to gauge the pressure. The terrible result of tapping a healthy pleura when the other lung is incapable of expansion confirms the conclusion derived from more frequent and harmless experiments.

Apart from any mechanical obstacle to separation of the two layers of pleura, either from adhesions or from their natural cohesion, it is obvious that when a pleuritic effusion is tapped and runs out, the intra-thoracic pressure must be greater than the barometric pressure of the air at the time. When it becomes greater with expiration, the pus or serum runs with a jerk; when it lessens with inspiration, the flow lessens also; when the pressure on the chest becomes negative, the flow ceases; or, if there is not enough fluid to close the trocar, air is sucked in. But when the orifice is closed after a little air has been admitted, no more will enter; and that already in the pleura will be quickly dissolved by the remaining fluid, or will be absorbed by the lymphatics of the pleura—first the oxygen, and then the nitrogen and carbonic dioxide. As the air is thus absorbed, the pressure in the pleura diminishes, and the lung again expands. Even if a large opening is made the lung is not emptied of air; it is only reduced to the condition observed after the thorax is opened, when death has occurred from some disease which has not affected the thorax. There is no carnified (*i. e.* completely airless) lung to be re-expanded (p. 1087), unless pleural effusion has compressed it for a long time. As soon as the pressure on its surface becomes a little less than that in the trachea, the compressed lung will begin to expand again, if not mechanically prevented by thick adhesions.

Anatomy.—The recognition of pneumothorax in the dead body is not always easy. In making an autopsy, at the moment when the knife first enters the thorax, the air can sometimes be heard to rush out; or, if a puncture is made with a trocar, it may escape in a jet, so as to blow out a lighted match. But it only happens when the pressure in the pleura is greater than that of the atmosphere, and this is the exception. In all probability the existence of air in the pleural space is often overlooked in *post-mortem* examinations, especially in the bodies of phthisical patients, in whom, from the presence of extensive adhesions, the collapse of the lung has been only partial. The best way of making sure whether there is pneumothorax or not is to puncture the chest under water, which may be done either by dissecting off the tissues from the ribs, so as to form a pouch that can be filled with water, or by pouring water into the abdomen, and then perforating the diaphragm with a trocar. When pleuritic effusion is present, it is sufficient to shake the body first: if there is any air, the liquid will be found frothy when the chest is opened.

When the pneumothorax arises from perforation of the visceral pleura, the aperture by which the air entered is sometimes plainly visible: it may be as large as a threepenny piece. More often it is covered by recently formed lymph, and the only way of detecting it is to inflate the lung with bellows through the trachea. Or it may have become completely closed by adhesions during the interval between the occurrence of the pneumothorax and the death of the patient, so that there is no possibility of discovering it (see Dr Hughes' case, p. 1116). It is most commonly situated upon the lateral surface of the lung, in the upper lobe near its lower border, or in the lower lobe near its upper border.

The chemical nature of air withdrawn from the pleural space was investigated by Dr John Davy in 1823,* and analyses have since been made by other chemists; it has always been found to consist mainly of nitrogen, and the amount of carbonic acid in it has generally been greater than that of the oxygen; sulphuretted hydrogen has been present when the other contents of the cavity were putrid. Walshe attributed the difference of this pleural air from that of the atmosphere to its having traversed the lung before reaching the pleura. But it can hardly be said to have passed through pulmonary tissue, and, moreover, its composition is far more altered than that of expired air. It must, therefore, have undergone change while in the pleural space, either from the solvent action of the liquid effusion, or from absorption by the pleural membrane—*i. e.* by the lymph which fills its stomata and lymphatics, and by the subpleural veins.

The secondary pleurisy.—When death occurs within a few hours after the development of pneumothorax, the cavity of the pleura is, of course, found empty, there having been no time for the occurrence of effusion. But in other cases (at least such as are seen by physicians) an empyema is the result in a few days, and it may begin within twenty-four hours. A striking exception is afforded by Vogel's case, already quoted (p. 1108). He repeatedly examined his patient during the month after she was attacked, and could never detect the slightest indication of pleurisy.

There is no doubt that the power of the air to set up suppuration depends upon its containing septic microbes. As we have seen, pneumothorax is, in most cases, due to the rupture of a phthisical vomica into the pleural space. The contents of the vomica must generally escape with the air, and they are probably the cause of the pleurisy which follows. When the original affection is a sloughing block in the lung, or when the pleura is perforated by a malignant œsophageal growth, or by a hypophrenic abscess communicating with the stomach, the consequent inflammation is peculiarly severe and rapid in its course.

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* 'Phil. Trans.' for that year; and in his collected 'Researches,' vol. ii, p. 249.

Of this we had an instance (June, 1890) in a patient with extensive sero-purulent effusion on the right side, caused by a sloughing abscess perforating the diaphragm The source of suppuration here was a cancer of the stomach.

In surgical practice, when a healthy lung is wounded by fractured ribs. pleurisy is often absent.

It is clearly impossible for subcutaneous emphysema to be produced by fracture of the ribs without there being also pneumothorax, unless the pleural space at the seat of injury happens to have been closed by former adhesions. But in cases of this kind we have often failed to detect any signs of the presence of air in the pleura when a day or two had passed before the patient was examined. So that air must often be absorbed very rapidly from the pleural space; and this conclusion is quite in accordance with the results of experiments on animals. Cohnheim says that in rabbits it is not possible by injection of air into the pleura to cause compression of the lung so as to study the effects of pneumothorax, because the air is so quickly absorbed.

Locality.—Walshe found of eighty-seven cases of perforation of a tuberculous lung collected from various sources, fifty-five on the left and thirty-two on the right side. Douglas Powell, in fifteen cases, found ten left and five right. But among twenty-six cases of pneumothorax taken from the records of Guy's Hospital without selection, the number on each side of the chest was exactly equal. Of the 83 cases reported by Dr Samuel West, there were 41 on the right and 42 on the left side: and of 65 cases (all in the course of 1000 of phthisis) Dr Fowler found 29 on the right, 34 on the left, and 2 on both sides.

Physical signs.—Pneumothorax is sometimes easy of diagnosis, but sometimes may be overlooked up to and even after the patient's death. Its certain recognition depends on physical examination of the chest, for the pateint's symptoms, though often characteristic, are sometimes strangely absent.

In general, pneumothorax is to be suspected whenever, over a large part of the chest, but on one side only, tympanitic resonance on percussion is associated with marked deficiency of the pulmonary murmur.

This enfeeblement or absence of breath-sounds is a very important and frequent indication; but in many cases there is marked *amphoric breathing*. This is not due to the passage of air backwards and forwards into the pleural cavity; but to the resonance of a large cavity full of air, giving its character to the breath-sounds.

The *voice*, like the breath, may either be less audible than on the healthy side, or it may acquire the character of amphoric bronchophony or pectoriloquy. Vocal fremitus appears to be always absent.

The *percussion-note* is over-resonant, with a tympanitic quality. When air escapes into a healthy pleural sac the sound is musically clear and resounding. But if the air should accumulate so as to cause extreme distension, it may at length become, according to Walshe and some other authorities, "muffled, toneless, almost dull," like that of a drum tightened to the highest possible point; and with all escape of air from its cavity prevented. This Skodaic resonance or "tympanitic dulness" is like that of a tightly distended coil of intestine (p. 1027). More frequently the reason why the percussion-sound in pneumothorax is imperfectly resonant is that the pleura itself is thickened; the chest walls fail to vibrate, and are incapable of transmitting the percussion-note to the air within. In such cases one may obtain osteal, tracheal, or subtympanitic modifications of the percussion-sound (cf. *supra*, p. 1026).

Metallic sounds.—There still remain certain physical signs, which when present are striking and characteristic, although their absence is no argument against the existence of pneumothorax.

Laennec described *metallic tinkling*[*] as a sound like that "produced in a metal cup, or in one made of glass or porcelain, by gently striking it with a pin, or by dropping into it a grain of sand." He described it as being heard when the patient either breathed, or spoke, or coughed. It is probably most often caused by the bursting of bubbles of fluid in a large space which is filled with air and has a smooth surface. In other words, metallic tinkling is a moist sound, or râle, modified by the vibrations of the walls of a large air-containing cavity. Another way in which Laennec believed the sound may be produced was the dropping of liquid from the upper into the lower part of the pleural space when it contains air as well as pus (*gutta cadens*). There is no difficulty in understanding how the sign may be produced by coughing, but metallic tinkling does not seem to arise as a mere result of speaking. What is then heard is rather an *echo* of the voice, which acquires a metallic quality from the conditions under which it is produced. So also the heart-sounds, and even the sound produced by percussion of the chest, may be reverberated with a similar musical quality. The thin, clear "tick-tack" of the heart in pneumothorax is like the sound of a watch or a child's "musical cart," and is a most characteristic sign.

A particular kind of musical echo was described by Trousseau as *le bruit d'airain*. Of all the highly consonating signs it only is completely under the control of the observer. Metallic tinkling is exceedingly capricious, accompanying certain respiratory movements and being absent with others, according as bubbles happen or do not happen to burst. Even a metallic echo of the patient's voice may probably fail to be heard unless he speaks distinctly and with a certain pitch and loudness. But the *bruit d'airain* can not only be determined as to the time, but the sound which is to produce it can be varied, until one obtains a satisfactory result. The method of eliciting it was originally given in the 'Gazette des Hôpitaux' for 1859. It consists in applying one's ear to the back of the patient's chest, while a third person strikes the front of the chest, either with the hammer upon the plessimeter, or with one coin upon another. The metallic echo which results is sometimes wonderfully distinct, and there are probably few cases of pneumothorax in which it is absent. One may sometimes, however, fail to obtain a "bell-sound" by percussion during life, and yet have no difficulty in eliciting it from the dead body of the same patient. Traube attributes this fact to the tension of the air in the pleural space being lowered, as the result of cooling of the tissues after death.

The succussion-splash.—Another sign of pneumothorax, which is of great historical interest, is "Hippocratic succussion." To obtain it, one may shake the patient's body while one has one's head pressed against his chest; but sometimes it can be heard at a little distance off, and the patient himself is often conscious of it every time he makes an abrupt movement, as in stepping downstairs, or sitting up in bed. It is nothing else than the splashing of air and liquid in the pleural cavity. It is therefore, properly, a sign, not of pneumothorax, but of pyopneumothorax. The following is the *locus classicus*.

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[*] *Fr.* Tintement métallique.—*Germ.* **Metallklang—Geräusch d. fallenden Tropfens.**

"Another malady. When the time grows long [after an inflammation of the chest has appeared], then the fever becomes higher and the cough increases, and the patient's side pains him, and he can no longer bear to lie on the sound side but only on the diseased one, and his feet swell and the hollow of his eyes. Then, when fifteen days have elapsed since the rupture [*i. e.* the bursting of an abscess into the pleura as the result of peripneumony, for that was the Hippocratic pathology of empyema], give the patient a warm bath [or, possibly, bathe the affected side with hot water] and set him upon a good steady stool. Then, while a friend holds his hands, do you shake him by the shoulder and listen, so as to tell on which side of the chest there is a splash (ψοφέη). [The word ψόφος denotes a noise, ***strepitus***, as opposed to an articulate sound: it is applied to doors banging, armour clanging, and streams splashing.]"—Hipp., 'De Morbis,' lib. ii, cap. xvi.

In such cases the signs of pleuritic effusion are to be observed, as well as those of pneumothorax; it may also be noted that alterations of the level of dulness when the patient changes his posture are generally very conspicuous, whereas in uncomplicated pleurisy they can seldom be made out satisfactorily (cf. p. 1088).

Dislocation of viscera.—In most instances pneumothorax is attended with lateral displacement of the heart. Douglas Powell showed (in vol. lix of the 'Med.-Chir. Trans.') that the mere elasticity of the opposite lung drags the mediastinum over whenever air has free entrance into one pleural space, without there being of necessity any excess of pressure above that of the atmosphere. He remarks, however, that, in some cases of phthisis, consolidation of the lung on the side opposite to the pneumothorax prevents the mediastinum from being thus displaced; and probably a like effect is also produced by consolidation and adhesion of any considerable part of the lung on the side of the pleural affection, or, again, by the rigidity and thickening of the pleura, which so often occur in cases of empyema before perforation takes place. Thus one must not expect to find the heart beating in an abnormal position in those chronic cases in which it is sometimes difficult to determine whether pneumothorax is or is not present.

Even in such cases it is possible for the pressure of air in the pleural cavity to be considerably increased. This is probably due to false membrane lying over the aperture and acting as a valve, so as to allow air to enter the cavity during inspiration, but hinder its escape during expiration. Cohnheim, indeed, declares that for air confined in the pleural space to retain for any length of time a high pressure after closure of the opening by which it entered is impossible, on account of the rapidity with which it undergoes absorption. But it is certain that among 17 cases collected by Powell there were 12 in which, after death, the pressure was found to be above the atmospheric pressure, the difference amounting in these cases to that of a column of from five and a half to seven inches of water. When the adjacent organs are capable of yielding to it, one cannot be surprised that the elastic force exerted by air in the pleural cavity should displace them even more than they are displaced by liquid effusion. Thus Dr Gee speaks of the diaphragm as being pushed down so that the upper surface of the liver lies below the level of the anterior costal margin, and percussion yields a tympanitic sound in the right hypochondrium. The intercostal spaces, too, may be flattened or bulging; and the affected side of the chest may be obviously enlarged as well as motionless.

Diagnosis.—The limitation of the physical signs to one side of the chest obviously suffices to exclude the possibility of their being due to emphysema, which from the time of Laennec has been given as the disease most needing distinction from pneumothorax; in practice the two are not in the least likely to be counfounded.

If, when distension of the pleura with air is extreme, the percussion-sound becomes muffled and toneless, this never reaches such a point that the case could be supposed to be one of liquid effusion.

In vol. xi of the 'St Bartholomew's Hospital Reports,' Mr Butlin recorded an example of rupture of the diaphragm, with escape of the distended stomach and colon into the left pleural cavity: it was the result of a severe crush between the buffers of two railway coal-waggons, and was diagnosed during life as traumatic pneumothorax.

But in general the only cases which are attended with doubt are those in which air is confined to a limited portion of the pleural space. Thus at the upper part of the chest it might very likely be impossible to diagnose a localised pneumothorax from an exceedingly large vomica. It is doubtful, however, whether limited pneumothorax ever occurs in that position, and perhaps the cases that have been admitted as open to question have all been examples of vomicæ attended with unusual signs, such as metallic tinkling, or Hippocratic succussion-splash. At the base of the chest, a cavity within the lung of sufficient size to be mistaken for pneumothorax is a thing almost, if not quite unknown.

It is possible that during the contraction of an empyema on the left side, the diaphragm, with the stomach, may be drawn upwards so far that percussion may yield a tympanitic sound over a considerable area, where complete dulness might have been expected. A similar condition may also arise when the lung is affected with cirrhosis. Probably one might avoid an error of diagnosis by re-examining the patient after having made him swallow a large quantity of fluid.

A subdiaphragmatic abscess, which arose from a perforating ulcer of the stomach, and consequently contained air, has been mistaken for pneumothorax.

The mistake which is most easily made is not that pneumothorax is mistaken for any other condition, or anything else mistaken for it, but that its presence is overlooked. This is due to the fact that the clinical symptoms of pneumothorax, though often striking in character, are sometimes absent, and so the physical signs are not sought for.

Symptoms.—The amount of dyspnœa produced by the escape of air into the pleural sac depends upon two conditions: first, upon whether the patient's vital functions are or are not being actively carried on at the time; and, secondly, upon whether he has or has not been accustomed to make full use of the lung on that side in breathing. A healthy person always experiences great distress when attacked with pneumothorax: and among phthisical patients the distress is greater in those who are able to take food and to bear exertion, and also if the least affected lung is the seat of pneumothorax. It accordingly reaches its maximum when a man who has one lung extensively diseased, but whose health is not yet seriously affected, is suddenly attacked with pneumothorax on the opposite side. A directly fatal result is then inevitable. Accordingly pneumothorax has to be remembered among the possible causes of sudden death in persons who are walking about and earning their living. One morning, in the year 1874, there was brought into Guy's Hospital the body of a man who had fallen dead while on his way to his work; he was found to have pneumothorax on the right side, and chronic phthisis of the left lung.

On the other hand, if air escapes into the pleura of a person who is wasted, and whose functions are already at a low ebb, and especially if the lung on that side has before been rendered almost useless by advanced tubercular disease, the supervention of the pneumothorax may give rise to no symptoms whatever. This fact was stated in the 'Medical Gazette' for 1844, by Dr Hughes, who was one of the best auscultators of that day.

In persons who are in the last stage of phthisis, it is possible for pneumothorax to produce a shock that may be directly fatal, without any warning symptoms. The patient is perhaps found dead in bed, and nothing has occurred to attract the attention of the nurse.

Between the two extremes just described there are all degrees of severity in the symptoms of pneumothorax. The most typical but the least frequently seen are cases in which the patient is suddenly seized with an agonising pain in the side, and has a sensation of something having given way, or possibly of a stream of air or of water trickling down within his chest. His dyspnœa is extreme; the respirations may reach forty or even sixty in the minute, while the beats of the heart, although accelerated, are not so to any proportionate extent. The pulse is small, the radial arteries being imperfectly filled as a consequence of the deficient flow through the obstructed lungs. The hands, the feet, the cheeks, the lips, and the visible mucous membranes become cyanosed; the extremities and the tongue itself feel cold; a cold sweat breaks out over the body; the temperature, even in the rectum, falls considerably. The voice is weak, or reduced to a whisper, and the patient cannot cough. He is usually obliged to sit up in bed, sometimes inclining towards the affected side, sometimes towards the healthy one.

Prognosis.—In some cases of pneumothorax the symptoms continue unabated until the death of the patient, which may take place after a few hours, or in a day or two. But in other cases they subside as the shock of the accident passes off; the breathing may remain rapid and yet the patient may experience little or no distress. In some very exceptional instances the air gradually undergoes absorption, and complete recovery takes place. Vogel's case, referred to above (p. 1008), was probably one of these.

The following case, reported by the late Dr Hughes in the 'Guy's Hospital Reports' for 1852 (2nd series, vol. viii, p. 20), is a proof that the perforation in the lung may be occluded.

Miss P—, æt. 26, had a slight attack of hæmoptysis the preceding year, and a brother had died of consumption. She suffered from cough, pain in the right side, and dyspnœa on exertion. There was dulness under the right clavicle. After an attack of pleurisy on the right side in December, 1850, Dr Hughes found tympanitic resonance on the right side except over the apex of the lung and behind, with amphoric breath-sounds,

metallic resonance of voice and cough, and succussion-splash on movement. There h been no sudden attack of dyspnœa. She somewhat improved under treatment, but April, 1851, Dr. Hughes found her much worse with signs of extensive effusion. T right side was tapped in May with much relief, and all the signs of pneumothorax return These were again replaced by those of hydrothorax until she was again tapped in Ju She was much better for a time, and able to visit the Exhibition in Hyde Park September, but disease of the pelvic bones showed itself, the symptoms of phthisis increas and she died in May, 1852.

At the autopsy, the right pleura was found universally adherent, and it contained abo 30 oz. of sero-purulent fluid without any air. The lung showed tubercles and cavitati with calcareous deposit, but it was not solidified. Careful experiments showed that wh the lung was placed under water no bubbles could be seen when it was inflated with a During the last ten months of the patient's life there had been no sign of pneumothora but unequivocal signs of air entering an expanded lung. It really appears that the cu of pneumothorax is in this case little short of actual demonstration.

What usually happens is that after a few days pleurisy sets in. Eve then it is not impossible for the disease to subside. In two cases Walsh saw all signs of air and fluid in the pleura disappear in the course of tw months; in all probability the exudation was sero-fibrinous. As a rul when an empyema follows pneumothorax the patient's best chance of r covery is for a free external opening to be made so as to allow the cavity become obliterated by granulation. The same treatment is necessa when the entrance of air into the pleura is secondary to pleurisy.

In cases in which there is already advanced phthisis the safest plan not to interfere.

There is good reason to believe that if the immediate effects of pneum thorax are got over, the destructive process in the perforated lung is check or, in some cases, entirely arrested. No fresh infection can take place wh the access of blood is cut off, no hæmoptysis can occur, and the ulcerati and septic processes, with abundant secretion of pus, must be to a gre extent diminished. And this probability is borne out by experience. 1872 Czernicki ('Gaz. hebd.') drew attention to the fact that in so phthisical patients the supervention of pneumothorax leads to an impro ment in the general symptoms, and to cessation of expectoration, effe which can only be ascribed to anæmia of the diseased lung after its c lapse. The same condition of the carnified lung explains cases in whi severe hæmoptysis has been stopped by the occurrence of pneumothorax.

Traube relates a case of pneumothorax occurring in a woman who h been attacked by it some years before he first saw her, and in whom sev years later scarcely any physical signs were discoverable. She looked we and could even walk uphill without discomfort. The history pointed phthisis, for she had previously had a febrile illness, with night-swea cough, and hæmoptysis.

As a rule, death occurs within two or three weeks after perforation the pleura. Traube insisted on the rapidity with which emaciation a vances in many cases. Œdema of the limbs, and even of the face, som times develops itself; and the urine is sometimes albuminous.

On the other hand, it is surprising how long a pyopneumothorax m be tolerated, and how little discomfort it causes. The patient is sometim able to take horse exercise, and thus may hear the fluid splashing within h chest while he is riding.

Treatment.—In cases of pneumothorax a great deal can be done diminish the patient's sufferings, and perhaps to avert a fatal terminatio Cupping, dry or wet, often gives remarkable relief, and venesection probably still more efficacious. A small dose of morphia should be injected subcutaneously, or, as Walshe recommended, a little chloroform may be given by inhalation from time to time.

If great enlargement of the side and depression of the diaphragm make it probable that the pressure of air within the thorax is greater than the atmospheric pressure, paracentesis should be performed with a small trocar. But displacement of the heart alone is not evidence of increased pressure: it may be due to the elasticity of the mediastinal tissues. Fräntzel appears to have tapped for pneumothorax rather frequently, and he quotes a dissertation of Bärensprung, in which are recorded a number of cases treated in this way with success. Dr Hughes only advised paracentesis in urgent cases, and this has since been our practice at Guy's Hospital. It is also advised by Mr Godlee in traumatic cases. When there is hydropneumothorax present in a case of phthisis, the case is better left alone, unless excessive dyspnœa should compel surgical interference.

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